# Young Children Develop in an Environment of Relationships

**WORKING PAPER 1** 

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The National Scientific Council on the Developing Child, housed at the Center on the Developing Child at Harvard University, is a multidisciplinary collaboration designed to bring the science of early childhood and early brain development to bear on public decisionmaking. Established in 2003, the Council is committed to synthesizing and communicating science to help inform policies that promote successful learning, adapative behavior, and sound physical and mental health for all young children. For more information, go to http://www.developingchild.net.

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# The Issue

HEALTHY DEVELOPMENT DEPENDS ON THE QUALITY AND RELIABILITY OF A YOUNG CHILD'S relationships with the important people in his or her life, both within and outside the family. Even the development of a child's brain architecture depends on the establishment of these relationships. 1, 2, 3, 4, 5, 6

Growth-promoting relationships are based on the child's continuous give-and-take ("serve and return" interaction) with a human partner who provides what nothing else in the world can offer - experiences that are individualized to the child's unique personality style; that build on his or her own interests, capabilities, and initiative; that shape the child's self-awareness; and that stimulate the growth of his or her heart and mind.

Young children experience their world as an environment of relationships, and these relationships affect virtually all aspects of their development - intellectual, social, emotional, physical, behavioral, and moral. The quality and stability of a child's human relationships in the early years lay the foundation for a wide range of later developmental outcomes that really matter - self-confidence and sound mental health, motivation to learn, achievement in school and later in life, the ability to control aggressive impulses and resolve conflicts in nonviolent ways, knowing the difference between right and wrong, having the capacity to develop and sustain casual friendships and intimate relationships, and ultimately to be a successful parent oneself.

Stated simply, relationships are the "active ingredients" of the environment's influence on healthy human development. They incorporate the qualities that best promote competence and well-being - individualized responsiveness, mutual action-and-interaction, and an emotional connection to another human being, be it a parent, peer, grandpar-

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ent, aunt, uncle, neighbor, teacher, coach, or any other person who has an important impact on the child's early development. Relationships engage children in the human community in ways that help them define who they are, what they can become, and how and why they are important to other people. 7, 8, 9, 10, 11, 12, 13

In the words of the distinguished developmental psychologist Urie Bronfenbrenner: ... in order to develop normally, a child requires progressively more complex joint activity with one or more adults who have an irrational emotional relationship with the child. Somebody's got to be crazy about that kid. That's number one. First, last, and always.

# What Science Tells Us

NURTURING AND STABLE RELATIONSHIPS WITH caring adults are essential to healthy human development beginning from birth. Early, secure attachments contribute to the growth of a broad range of competencies, including a love of learning, a comfortable sense of oneself, positive social skills, multiple successful relationships at later ages, and a sophisticated understanding of emotions, commitment, morality, and other aspects of human relationships. Stated simply, establishing successful relationships with adults and other children provides a foundation of capacities that children will use for a lifetime. 14, 15, 16, 17

The "serve and return" interaction between parent and baby – in which young children naturally reach out for interaction through babbling, facial expressions, and gestures and adults respond with the same kind of vocalizing and gesturing back at them – builds and strengthens brain architecture and creates a relationship in which the baby's experiences are affirmed and new abilities are nurtured. Children who have healthy relationships with their primary caregivers are more likely to develop insights into other people's feelings, needs, and thoughts, which form

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a foundation for cooperative interactions with others and an emerging conscience. Sensitive and responsive parent-child relationships also are associated with stronger cognitive skills in young children and enhanced social competence and work skills later in school, which illustrates the connection between social/emotional development and intellectual growth. The broader quality of the home environment (including toys, activities, and interactions within the family setting) also is strongly related to early cognitive and language development, performance on IQ testing, and later achievement in school. <sup>12, 18, 19, 20, 21, 22, 23, 24, 25, 26</sup>

Young children also learn a great deal from each other. They learn how to share, to engage in reciprocal interactions (e.g., taking turns, giving and receiving), to take the needs and desires of others into account, and to manage their own impulses. Just being around other children, however, is not enough. The development of friendships is essential, as children learn and play more competently in the rapport created with friends rather than when they are dealing with the social challenges of interacting with casual acquaintances or unfamiliar peers.<sup>27, 28</sup>

The warmth and support of the caregiver in a

child care setting also influence the development of important capabilities in children, including greater social competence, fewer behavior problems, and enhanced thinking and reasoning skills at school age. Young children benefit in these ways because of the secure relationships they develop in such settings, and because of the ways in which the caregivers provide cognitively stimulating activities and support for developing positive relationships with other children. Unfortunately, the generally poor quality of care provided in many child care arrangements in the United States does not support these benefits because of high caregiver turnover, poorly designed programs, or inadequate preparation of staff. Current research also suggests the additional risk that a greater amount of time in outof-home care during infancy may be associated with greater disobedience and aggression by the time children enter school. 12, 22, 23, 29, 30, 31, 32, 33

Relationships are important to school adjustment. Children who develop warm, positive relationships with their kindergarten teachers are more excited about learning, more positive about coming to school, more self-confident, and achieve more in the classroom. Relationships with peers also are important. Children who experience greater peer acceptance and friendship tend to feel more positively about the school experience and perform better in the classroom. <sup>33, 34, 35, 36, 37, 38</sup>

Children have different ways of interacting with their peers. Some are gregarious, others are too shy to get involved (although they want to), some need time to "warm up," and others are not as interested in being sociable. All of these variations fall within a normal range, and it is essential to differentiate among the many potential reasons (both biological and environmental) that a young child may have limited or difficult interactions with others. Playing cooperatively, making friends, and sustaining friendships over time are not always easy. Any child with severely limited peer involvement is at considerable risk for significant adverse developmental consequences. 39, 40, 41, 42, 43

Secure and stable relationships with caring adults assure that young children are adequately nourished; protected from dangerous illnesses, exposure to toxins, and hazards that can lead to preventable injuries; provided preventive health check-ups; protected from excessive stress; and afforded predictable daily routines that convey a sense of security. These influences contribute significantly to healthy brain development and depend upon the care and support provided by individuals in the community as well as in the family.12,44

Young children are highly vulnerable emotionally to the adverse influences of parental mental health problems and family violence. One of the most extensively documented of these vulnerabilities is the negative impact of a mother's clinical depression on her young children's emotional development, social sensitivity, and concept of themselves, effects that have been demonstrated in both developmental research and studies of brain functioning. Young children who grow up in seriously troubled families, especially those who are vulnerable temperamentally, are prone to the development of behavioral disorders and conduct problems. 45, 46, 47, 48, 49

Animal studies have shown that the quality of the mother-infant relationship can influence gene expression in areas of the brain that regulate social and emotional function and can even lead to changes in brain structure. The nature of the relationship also can have long-term influences (into adulthood) on how the body copes with stress, both physically and emotionally. 15, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60

Science indicates that the quality of early parent-child relationships can be strengthened, but successful interventions are more difficult to achieve when relationships are significantly troubled or disturbed. Preventive interventions also can produce a variety of positive outcomes, depending on the extent to which the knowledge and skills of the staff and the quality of the implementation are matched to the magnitude of the challenges being addressed. 12,61,62,63,64,65

# **Popular Misrepresentations of Science**

AS THE PUBLIC'S APPETITE FOR SCIENTIFIC INFORmation about the development of young children is whetted by exciting new findings, the risk of exaggerated or misleading messages grows. Within this context, it is essential that scientific fact be differentiated from popularly accepted fiction.

Contrary to common assumptions, scientific evidence shows that the influence of relationships on development continues throughout the lifespan. These relationships are not more important at a particular stage of a child's life compared to another, but the nature of those impacts does vary by age and developmental status.4

In contrast to frequently cited concerns, science indicates that young children can benefit significantly from secure relationships with multiple caregivers (within or outside the family), while their attachments to their parents remain primary and central.13 There is no credible scientific evidence to support the claim that close

relationships with other nurturing and reliable adults who they trust, especially early in life, interfere with the strength of the young child's primary relationship with his or her parents.

Although young children certainly can establish healthy relationships with more than one or two adults, prolonged separations from familiar caregivers and repeated "detaching" and "re-attaching" to people who matter are emotionally distressing and can lead to enduring **problems.** There is no scientific evidence to support the belief that frequently rotating relationships with large numbers of adult caregivers provide valuable learning opportunities in the early years of life. Although the importance of sustained, reliable relationships within the family is well understood, the need for stable and predictable relationships in child care settings is acknowledged less frequently, and the disruptive impacts of the abrupt changes related to high caregiver turnover are too often disregarded.66,67

# The Science-Policy Gap

THE IMPORTANCE OF MOTHER-CHILD RELATIONships is old news. The importance of other family relationships (with fathers, siblings, and grandparents) is semi-old news. The impact of these relationships on the development of the brain is new news. And the important influence of relationships outside of the family - with child care providers, peers, teachers, neighbors, and other adults and children in the community - is even newer, because these individuals are often valued more for what they do than for the meaning of their role in the life experience of very young children. Greater understanding of what science tells us about the importance of a range of relationships for early childhood development leads us to think about many areas of policy and practice in a new light.

"Quality" in early child care and education, for example, is often defined in terms of adult-child ratios, group size, physical facilities, and, more recently, cognitively oriented curriculum. But "quality" is perceived differently when we view child care as a prominent feature of the environment of relationships in which young children develop. The importance of ensuring that relationships in child care are nurtur-

Parental leave policies in the United States currently provide parents of young children with few options.

ing, stimulating, and reliable leads to an emphasis on the skills and personal attributes of the caregivers, and on improving the wages and benefits that affect staff turnover. 12, 68, 69, 70

Parental leave policies in the United States currently provide parents of young children with few options. A maximum of only three months of unpaid leave is assured for parents of newborns, and these policies cover only about half of American workers. Of those who are eligible for leave, only those who can get by without earned income can afford to take it, and fewer than half of workers even have this option without risking loss of their jobs. These policies seem highly

problematic when viewed in relation to extensive scientific evidence of the vital importance of establishing a strong and healthy mother-infant bond beginning in the early months of life. They elicit even greater alarm when viewed in the context of concerns about the potential adverse effects on very young babies of early and extended experiences in out-of-home child care arrangements of highly variable quality.<sup>71,72,73</sup>

For mothers receiving welfare support under Temporary Assistance to Needy Families (TANF), federal rules require that states impose work requirements of 30 or more hours per week. Although modifications are permissible, about half of the states do not exempt mothers of children less than 12 months of age, and some states permit mandated maternal employment beginning a few weeks after a baby's birth. When viewed as an adult-oriented employment policy, TANF can be a subject for reasonable debate. But when examined from a child-oriented perspective, it reflects a wide gap between what we know about the importance of early family relationships and what we are doing to promote the health and well-being of our nation's most vulnerable young children.<sup>74,75</sup>

# Implications for Policy and Programs

THE SCIENCE OF EARLY CHILDHOOD DEVELOPment is sufficiently mature to support a number of well-documented, evidence-based implications for those who develop and implement policies that affect the health and well-being of young children. Five compelling messages are particularly worthy of thoughtful consideration:

When considered within the context of a child's environment of relationships, the concept of school readiness is not exclusively a matter of fostering literacy and number skills. It must also include the capacity to form and sustain positive relationships with teachers, children, and other adults, and develop the social and emotional skills for cooperating with others.66,76,77

When viewed as an important part of a child's environment of relationships, early childhood education must strive to involve young children in reciprocal learning interactions with teachers and peers rather than isolated "pre-academic" work, and it should capitalize on children's natural interests and intrinsic drive to learn, rather than follow an adult-determined agenda. Stated simply, young children learn best in an interactive, relational mode rather than through an education model that focuses on rote instruction.<sup>78</sup>

Extending the length and coverage of leave currently provided under the Family and Medical Leave Act would provide the critical necessities of time and economic security that are required for parents to develop the nurturing relationships with their children that are essential to healthy **development.** Much can be learned from other industrialized nations that promote greater parental choice and child well-being by providing subsidized parental leave for those who wish to stay at home with their babies, and affordable, decent quality, early care and education for the children of those who choose or are compelled to return to work.73,78

In the absence of consistent evidence that maternal employment intrinsically helps or hurts most children, science has little to add to the ongoing political debate about whether paid work should be a mandated requirement for mothers on public assistance. Nevertheless, emerging data that

suggest that maternal employment in the first six months of an infant's life may be associated with later developmental problems, and concerns about the potential adverse impacts of extended out-of-home child care experiences on young children's social development and

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behavior, require thoughtful public discussion. Each raises serious concerns about the potential harm of mandated maternal employment and the limited availability of affordable, high quality child care, particularly for the already vulnerable babies of low-income women on public assistance. It is time for society to weigh the evidence carefully and fashion a more thoughtful policy for parents in the workforce, particularly for those who earn low wages. 12,79,80

Traditional child welfare approaches to maltreatment focus largely on physical injury, the relative risk of recurrent harm, and questions of child custody, in conjunction with a criminal justice **orientation.** In contrast, when viewed through a child development lens, the abuse or neglect of young children should be evaluated and treated as a matter of child health and development within the context of a family relationship crisis, which requires sophisticated expertise in both early childhood and adult mental health. The regularized referral of suspected cases of child abuse or neglect from the child welfare system to the early intervention system would assure appropriate developmental and behavioral assessment and treatment as needed. Child abuse prevention strategies that emphasize both the developmental needs of children and the importance of community-based supports for families provide another clear example of how we can close the gap between science and practice for our most vulnerable young children.<sup>12</sup>

# References

- Berscheid, E., & Reis, H.T. (1998). Attraction and close relationships. In D.T. Gilbert, S.T. Fiske, & G. Lindzey (Eds.), *Handbook of social psychology, Vol. 1* (2nd Ed.). New York: McGraw-Hill.
- Collins, W.A., & Laursen, B. (1999). Relationships as developmental contexts. *The Minnesota Symposia on Child Psychology, Vol. 30*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Dunn, J. (1993). Young children's close relationships: Beyond attachment. Newbury Park, CA: Sage Publications.
- 4. Reis, H.T., Collins, W.A. & Berscheid, E. (2000). Relationships in human behavior and development. *Psychological Bulletin*, 126(6), 844-872.
- Dawson, D., & Fischer, K.W. (Eds.) (1994). Human behavior and the developing brain. New York: Guilford Press.
- Panksep, J. (1998). Affective neuroscience. New York: Oxford.
- 7. Bornstein, Marc (Ed.) (2002). *Handbook of parenting* (2nd ed.). Mahwah, NJ: Lawrence Erlbaum Assoc.
- 8. Cassidy, J. & Shaver, P.R. (Eds.) (1999). Handbook of attachment: Theory, research, and clinical applications (pp. 89-111). New York: Guilford.
- Cochran, M., Larner, M., Riley, D., Gunnarsson, L., & Henderson, C.R., Jr. (1990). Extending families: The social networks of parents and their children. New York: Cambridge University Press.
- 10. Fogel, A. (1993). Developing through relationships: Origins of communication, self, and culture. Chicago: University of Chicago Press.
- Rogoff, B. (1990). Apprenticeship in thinking: Cognitive development in social context. New York: Oxford University Press.
- Shonkoff, J.P., & Phillips, D. (Eds.) (2000). From neurons to neighborhoods: The science of early childhood development. Committee on Integrating the Science of Early Childhood Development. Washington, DC: National Academy Press.
- Belsky, J., & Cassidy, J. (1994). Attachment: Theory and evidence. In M. Rutter & D. Hay (Eds.), *Development* through life. (pp. 373-402). Oxford, UK: Blackwell Scientific.
- Thompson, R.A. (1999). Early attachment and later development. In J. Cassidy & P.R. Shaver (Eds.), Handbook of attachment: Theory, research, and clinical applications (pp. 265-286). New York: Guilford Press.
- 16. Thompson, R.A. (2000). The legacy of early attachments. *Child Development*, 71(1),145-152.
- 17. Waters, E., Kondo-Ikemura, K., Posada, G., & Richters, J.E. (1991). Learning to love: Mechanisms and milestones. In M. Gunnar & L. Sroufe (Eds.), Self processes and development. *Minnesota Symposia on Child Psychology, Vol. 23.* (pp. 217-255). Hillsdale NJ: Erlbaum.
- 18. Bradley, R.H., Caldwell, B.M. Rock, S.L., & Ramey, C.T. (1989). Home environment and cognitive development in the first three years of life: A collaborative study

- involving six sites and three ethnic groups in North America. *Developmental Psychology*, 25(18), 217-235.
- Bradley, R.H., Caldwell, B.M., & Rock, S.L. (1988). Home environment and school performance: A ten-year followup and examination of three models of environmental action. *Child Development*, 59(2), 852-867.
- Estrada, P., Arsenio, W.F., Hess, R.D., & Holloway, S.D. (1987). Affective quality of the mother-child relationship: Longitudinal consequences for children's schoolrelevant cognitive functioning. *Developmental Psychology*, 23(2), 210-215.
- Gottfried, A.W., & Gottfried, A.E. (1984). Home environment and early cognitive development. New York: Academic Press.
- 22. Peisner-Feinberg, E.S., Burchinal, M.R., Clifford, R.M., Culkin, M.I., Howes, C., Kagan, S.I., Yazejian, . . . Zelazo, J. (2000). The children of the Cost, Quality, and Outcomes Study go to school: Technical report. Chapel Hill, NC: Frank Porter Graham Child Development Center, University of North Carolina at Chapel Hill.
- Pianta, R.C., Nimetz, S.L., & Bennett, E. (1997). Motherchild relationships, teacher-child relationships, and school outcomes in preschool and kindergarten. *Early Childhood Research Quarterly*, 12(3), 263-280.
- 24. Kochanska, G., & Thompson, R.A. (1997). The emergence and development of conscience in toddlerhood and early childhood. In J.E. Grusec & L. Kuczynski (Eds.), Parenting and children's internalization of values (pp. 53-77). New York: Wiley.
- 25. Thompson, R.A., Meyer, S., & McGinley, M. (2006). Understanding values in relationship: The development of conscience. In M. Killen & J. Smetana (Eds.), Handbook of moral development. Mahwah, NJ: Lawrence Erlbaum Associates.
- Kochanska, G. (2002). Mutually responsive orientation between mothers and their young children: A context for the early development of conscience. *Current Directions* in *Psychological Science*, 11(6), 191-195.
- 27. Rubin, K.H., Bukowski, W., & Parker, J.G. (1998). Peer interactions, relationships, and groups. In W. Damon (Ed.) & N. Eisenberg (Vol. Ed.), Handbook of child psychology, Vol. 3: Social, emotional, and personality development (5th ed., pp. 619-700). New York: Wiley.
- Rose-Krasnor, L. (1997). The nature of social competence: A theoretical review. Social Development, 6, 111-135.
- 29. Lamb, M.R. (1998). Nonparental child care: Context, quality, correlates. In W. Damon (Ed.), & I.E. Seigel & K.A. Renninger (Vol. Eds.), *Handbook of child psychology,* Vol. 4: Child psychology in practice. (5th ed., pp.73-134). New York: Wiley.
- NICHD Early Child Care Research Network (2000). The relation of child care to cognitive and language development. *Child Development*, 71(4), 958-978.
- NICHD Early Child Care Research Network (2002). Early child care and children's development prior to school entry: Results from the NICHD Study of Early Child Care. American Educational Research Journal, 39(1), 133-164.
- 32. NICHD Early Child Care Research Network (2003). Does amount of time spent in child care predict socioemotional adjustment during the transition to kindergarten? *Child Development*, 74(4), 976-1005.

- 33. Pianta, R.C. (1999). Enhancing relationships between children and teachers. Washington, DC: American Psychological Association.
- 34. Birch, S., & Ladd, G. (1997). The teacher-child relationship and children's early school adjustment. Journal of School Psychology, 35, 61-79.
- 35. Ladd, G.W., Birch, S.H., & Buhs, E.S. (1999). Children's social and scholastic lives in kindergarten: Related spheres of influence? Child Development, 70(6), 1373-1400.
- 36. Ladd, G.W., Kochenderfer, B.J., & Coleman, C.C. (1996). Friendship quality as a predictor of young children's early school adjustment. Child Development, 67(6), 1103-1118.
- 37. Ladd, G.W., Kochenderfer, B.J., & Coleman, C.C. (1997). Classroom peer acceptance, friendship, and victimization: Distinct relational systems that contribute uniquely to children's school adjustment? Child Development, 68, 1181-1197.
- 38. Pianta, R.C., & Steinberg, M. (1992). Teacher-child relationships and the process of adjusting to school. In R.C. Pianta (Ed.), Beyond the parent: The role of other adults in children's lives. New Directions for Child Development, 57, 61-80.
- 39. Dunn, J. (2004). Children's friendships: The beginnings of intimacy. Oxford, UK: Blackwell Publishers.
- 40. Fox, N.A., Henderson, H.A., Rubin, K.H., Calkins, S.D., & Schmidt, L.A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. Child Development, 72 (1), 1-21.
- 41. Kagan, J., Reznick, J.S., & Snidman, N. (1987). The physiology and psychology of behavioral inhibition in children. Child Development, 58(6), 1459-1473.
- 42. Rothbart, M.K., Ahadi, S.A., & Hershey, K.L. (1994). Temperament and social behavior in childhood. Merrill-Palmer Quarterly, 40(1), 21-39.
- 43. Rubin, K.H., Coplan, R.J., Nelson, L.J., Cheah, C.S.L., & Lagace-Seguin, D.G. (1999). Peer relationships in childhood. In M.H. Bornstein & M.E. Lamb (Eds.), Developmental psychology: An advanced textbook (4th ed., pp. 451-501). Mahwah, NJ: Lawrence Erlbaum Associates.
- 44. Gunnar, M.R., Brodersen, L., Nachmias, M., Buss, K., & Rigatuso, R., (1996). Stress reactivity and attachment security. Developmental Psychology, 29, 10-36.
- 45. Dawson, G., & Ashman, D.B. (2000). On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk of affective disorder. In C.A. Nelson (Ed.), The effects of early adversity on neurobehavioral development. Minnesota Symposia on Child Psychology, Vol. 31 (pp. 245-279). Mahwah, NJ: Erlbaum.
- 46. Dawson, G., Frey, K., Panagiotides, H., Yamada, E., Hessl, D., & Osterling, J.(1999). Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. Child Development, 70(5), 1058-1066.
- 47. Shaw, D.S., Gilliom, M., Ingoldsby, E.M., & Nagin, D.S.(2003). Trajectories leading to school-age conduct problems. Developmental Psychology, 39(2), 189-200.
- 48. Shaw, D.S., Owens, E.B., Giovannelli, J., & Winslow, E.B. (2001). Infant and toddler pathways leading to early externalizing disorders. Journal of the American Academy of Child & Adolescent Psychiatry, 40, 36-43.
- 49. National Scientific Council on the Developing Child. (2008). Mental health problems in early childhood can

- impair learning and behavior for life. Working Paper No. 6. Retrieved from http://www.developingchild.net
- 50. National Scientific Council on the Developing Child. (2004). Children's emotional development is built into the architecture of their brains. Working Paper No. 2. Retrieved from http://www.developingchild.net
- 51. Champagne, F.A., Francis, D., Mar, A., & Meaney, M.J. (2003). Variations in maternal care in the rat as a mediating influence for the effects of environment on development. Physiology and Behavior, 79, 359-371.
- 52. Meaney, M.J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. Annual Review of Neuroscience, 24, 1161-192.
- 53. Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Pearson, D., Plotsky, P.M., & Meaney, M.J. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. Science, 277, 1659-1662.
- 54. Leiderman, P. (1981). Human mother-infant social bonding: Is there a sensitive phase? In K. Immelmann, G. Barlow, L. Petrinovich, & M. Main (Eds.), Behavioral development (pp. 454-468). Cambridge: Cambridge University Press.
- 55. Caldji, C., Tannenbaum, B., Sharma, S., Francis, D., Plotsky, P.M., & Meaney, M.J.(1998). Maternal care during infancy regulates the development of neural systems mediating the expression of fearfulness in the rat. Proceedings of the National Academy of Sciences of the United States of America, 95 (9), 5335-5340.
- 56. Coplan, J.D., Andrews, M.W., Rosenbaum, L.A., Owens, M.J., Friedman, S., & Gorman, J.M. (1996). Persistent elevations of cerebrospinal fluid concentrations of corticotropin-releasing factor in adult nonhuman primates exposed to early-life stressors: Implications for the pathophysiology of mood and anxiety disorders. Proceedings of the National Academy of Sciences of the United States of America, 93, 1619-1623.
- 57. Liu, D., Diorio, J., Day, J.C., Francis, D.D., & Meaney, M.J. (2000). Maternal care, hippocampal synaptogenesis and cognitive development in rats. Nature Neuroscience, 3(8), 799-806.
- 58. Sanchez, M.M., Ladd, C.O., & Plotsky, P.M. (2001). Early adverse experience as a developmental risk factor for later psychopathlogy: Evidence from rodent and primate models. Development and Psychopathology, 13, 419-449.
- 59. Sanchez, M.M., Hearn, E.F., Do, D., Rilling, J.K., & Herndon, J.G. (1998). Differential rearing affects corpus callosum size and cognitive function of rhesus monkeys. Brain Research, 812(1-2), 38-49.
- 60. Shonkoff, J. & Meisels, S. (Eds.) (2000). Handbook of early childhood intervention. (2nd Ed.) New York: Cambridge University Press.
- 61. Brooks-Gunn, J., Berlin, L.J., & Fuligni, A.S. (2000). Early childhood intervention programs: What about the family? In J.P. Shonkoff & S.J. Meisels (Eds.), Handbook of early childhood intervention (2nd ed., pp. 549-587). New York: Cambridge University Press.
- 62. Campbell, F.A., Ramey, C., Pungello, E., Sparling, J.,& Miller-Johnson, S.(2002) Early childhood education: Young adult outcomes from the Abecedarian Project. Applied Developmental Science, 6(1), 42-57.
- 63. Currie, J. (2000). Early childhood intervention programs: What do we know? Working Paper from the Children's Roundtable: The Brookings Institution, Washington, DC: http://www.Brookings.org/es/children/docs/currie2000000401.htm.

- 64. Gomby, D.S., Culross, P.L., & Behrman, R.E. (1999). Home visiting: Recent program evaluations – Analysis and recommendations. *The Future of Children, 9*, 4-26.
- Thompson, R.A. (1995). Preventing child maltreatment through social support: A critical analysis. Thousand Oaks, CA: Sage.
- 66. Howes, C. (1999). Attachment relationships in the context of multiple caregivers. In J. Cassidy & P. R. Shaver (Eds.), Handbook of attachment: Theory, research, and clinical applications (pp. 671-687). New York: Guilford Press.
- 67. Howes, C.H.,& Ritchie, S.(2002).A matter of trust.New York:Teachers College Press.
- Phillips, D., Mekos, D., Carr, S., McCartney, K., & Abbott-Shim, M. (2000). Within and beyond the classroom door: Assessing quality in child care centers. *Early Childhood Research Quarterly*, 15(4), 475-496.
- NICHD Early Child Care Research Network (1996).
   Characteristics of infant child care: Factors contributing to positive caregiving. Early Childhood Research Quarterly, 11(3), 269-306.
- NICHD Early Child Care Research Network (2000).
   Characteristics and quality of child care for toddlers and preschoolers. Applied Developmental Science, 4(3), 116-125.
- Kamerman, S., & Kahn, A. (1995). Starting right: How America neglects its young children and what we can do about it. New York: Oxford University Press.
- 72. Waldfogel, J.(1999). The impact of the Family and Medical Leave Act. *Journal of Policy Analysis and Management*, 18 (2), 281-302.
- Waldfogel, J. (2001). International policies toward parental leave and child care. The Future of Children, 11(1), 99-111.
- 74. Duncan, G., & Chase-Lansdale, L. For better and for worse: Welfare reform and the well-being of children and families. New York: Russell Sage.
- 75. Huston, A.C. (2002). Reforms and child development. *The Future of Children*, *12*(1), 59-77.
- Pianta, R.C., Steinberg, M.,& Rollins, K.(1997). The first two years of school: Teacher-child relationships and deflection in children's school adjustment. *Development* and Psycholopathology, 9, 63-79.
- Stipek, D.(2004). The early childhood classroom observation measure. Unpublished manuscript, Stanford University.
- Waldfogel, J., Higuchi, Y., & Abe, M. (1999). Family leave policies and women's retention after childbirth: Evidence from the United States, Britain, and Japan. *Journal of Population Economics*, 12, 523-545.
- Han, W., Waldfogel, J., & Brooks-Gunn, J. (2001). The effects of early maternal employment on children's later cognitive and behavioral outcomes, *Journal of Marriage* and the Family, 63(2), 336-354.
- Brooks-Gunn, J., Han, W., & Waldfogel, J. (2002).
   Maternal employment and child cognitive outcomes in the first three years of life. *Child Development*, 73(4), 1052-1072.

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# NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD

Center on the Developing Child HARVARD UNIVERSITY

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# Children's Emotional Development Is Built into the Architecture of Their Brains

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The National Scientific Council on the Developing Child, housed at the Center on the Developing Child at Harvard University, is a multidisciplinary collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children. For more information, go to www.developingchild.net.

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# The Issue

A GROWING BODY OF SCIENTIFIC EVIDENCE TELLS US THAT EMOTIONAL DEVELOPMENT BEGINS EARLY in life, that it is a critical aspect of the development of overall brain architecture, and that it has enormous consequences over the course of a lifetime. These findings have far-reaching implications for policymakers and parents, and, therefore, demand our attention.

From birth, children rapidly develop their abilities to experience and express different emotions, as well as their capacity to cope with and manage a variety of feelings. <sup>1,2,3</sup> The development of these capabilities occurs at the same time as a wide range of highly visible skills in mobility (motor control), thinking (cognition), and communication (language).<sup>4</sup>

Yet, emotional development often receives relatively less recognition as a core emerging capacity in the early childhood years. The foundations of social competence that are developed in the first five years are linked to emotional well-being and affect a child's later ability to functionally adapt in school and to form successful relationships throughout life. 5,6,7,8

As a person develops into adulthood, these same social skills are essential for the formation of lasting friendships and intimate relationships, effective parenting, the ability to hold a job and work well with others, and for becoming a contributing member of a community.<sup>9,10</sup>

Disregarding this critical aspect of the

developing child can lead parents and policymakers to underestimate its importance and to ignore the foundation that emotions establish for later

# As young children develop, their early emotional experiences literally become embedded in the architecture of their brains.

growth and development. Thus, it is essential that young children's feelings get the same level of attention as their thinking. Indeed, learning to manage emotions is more difficult for some children than learning to count or read and may, in some cases, be an early warning sign of future psychological problems. The failure to address difficulties in this equally important domain can result in missed opportunities for interventions. Had they been initiated early, these interventions could have yielded tremendous benefits for large numbers of children and for society.

# What Science Tells Us

THE CORE FEATURES OF EMOTIONAL DEVELOPment include the ability to identify and understand one's own feelings, to accurately read and comprehend emotional states in others, to manage strong emotions and their expression in a constructive manner, to regulate one's own behavior, to develop empathy for others, and to establish and sustain relationships.<sup>2,11,12</sup>

Emotional development is actually built into the architecture of young children's brains in response to their individual personal experiences and the influences of the environments in which they live. Indeed, emotion is a biologically based aspect of human functioning that is "wired" into multiple regions of the central nervous system that have a long history in the evolution of our species. 13,14,15,16,17

These growing interconnections among brain circuits support the emergence of increasingly mature emotional behavior, particularly in the preschool years. Stated simply, as young children develop, their early emotional experiences literally become embedded in the architecture of their brains. Here is what we know:

The emotional experiences of newborns and young infants occur most commonly during periods of interaction with a caregiver (such as feeding, comforting, and holding).<sup>8,11,18,19</sup> Infants display distress and cry when they are hungry,

cold, wet, or in other ways uncomfortable, and they experience positive emotions when they are fed, soothed, and held. During this early period, children are incapable of modulating the expression of overwhelming feelings, and they have limited ability to control their emotions in the service of focusing or sustaining attention.13 Associations between positive emotions and the availability of sensitive and responsive caregiving are strengthened during infancy in both behavior and brain architecture.20

The emotional states of toddlers and preschoolers are much more complex.21 They depend on their emerging capacities to interpret their own

# The emotional health of young children is closely tied to the social and emotional characteristics of the environments in which they live.

personal experiences and understand what others are doing and thinking, as well as to interpret the nuances of how others respond to them.<sup>2,11,22,23</sup> As they (and their brains) build on foundations that are established earlier, they mature and acquire a better understanding of a range of emotions. They also become more capable of managing their feelings, which is one of the most challenging tasks of early childhood.<sup>3,24,25,26,27</sup>

By the end of the preschool years, children who have acquired a strong emotional foundation have the capacity to anticipate, talk about, and use their awareness of their own and others' feelings to better manage everyday social interactions.<sup>2,11</sup> Their emotional repertoires have expanded dramatically and now include such feelings as pride, shame, guilt, and embarrassment — all of which influence how individuals function as contributing members of a society.<sup>21,28</sup> Throughout the early childhood years, children develop increasing capacities to use language to communicate how they feel and to gain help without "melting down," as well as to inhibit the expression of emotions that are inappropriate for a particular setting.3,29

When feelings are not well managed, thinking can be impaired. Recent scientific advances have

shown how the interrelated development of emotion and cognition relies on the emergence, maturation, and interconnection of complex neural circuits in multiple areas of the brain, including the prefrontal cortex, limbic cortex, basal forebrain, amygdala, hypothalamus, and brainstem.30 The circuits that are involved in the regulation of emotion are highly interactive with those that are associated with "executive functions" (such as planning, judgment, and decision-making), which are intimately involved in the development of problem-solving skills during the preschool years.31 In terms of basic brain functioning, emotions support executive functions when they are well regulated but interfere with attention and decisionmaking when they are poorly controlled. 19,32,33,34,35

We now know that differences in early childhood temperament - ranging from being extremely outgoing and adventurous to being painfully shy and easily upset by anything new or unusual are grounded in one's biological makeup.36,37 These variations lead to alternative behavioral pathways for young children as they develop individual strategies to control their emotions during the preschool years and beyond. They also present diverse challenges for parents and other adults who must respond differently to different kinds of children.<sup>38</sup> When it comes to finding the "best" approach for raising young children, scientists tell us that one size does not fit all.39

Young children are capable of surprisingly deep and intense feelings of sadness (including depression), grief, anxiety, and anger (which can result in unmanageable aggression), in addition to the heights of joy and happiness for which they are better known. 40,41,42,43 For some children, the preschool years mark the beginning of enduring emotional difficulties and mental-health problems that may become more severe than earlier generations of parents and clinicians ever suspected.

The emotional health of young children - or the absence of it - is closely tied to the social and emotional characteristics of the environments in which they live, which include not only their parents but also the broader context of their families and communities. 44,45,46,47,48 Young children who grow up in homes that are troubled by parental mental-health

problems, substance abuse, or family violence face significant threats to their own emotional development. The experience of chronic, extreme, and/or uncontrollable maltreatment has been documented as producing measurable changes in the immature brain. 49,50

Children's early abilities to deal with their emotions are important not only for the foundation these capacities provide for the future, but also for the children's current social functioning with their parents, teachers, and peers. Indeed, differences in how young children understand and regulate their own emotions are closely associated with peer and teacher perceptions of their social competence, as well as with how well-liked they are in a child-care setting or preschool classroom. 51,52,53

# **Correcting Popular Misrepresentations of Science**

AS THE PUBLIC'S APPETITE FOR SCIENTIFIC INFORMAtion about the development of young children is whetted by exciting new findings, the risk of exaggerated or misleading messages grows. Within this context, it is essential that scientific fact be differentiated from popularly accepted fiction.

There is no credible scientific evidence that young children who have been exposed to violence will invariably grow up to be violent adults themselves. Although these children clearly are at greater risk for adverse impacts on brain development and later problems with aggression, they are not doomed to poor outcomes, and they can be helped substantially if provided with early and appropriate treatment, combined with reliable and nurturing relationships with supportive caregivers.<sup>54</sup>

Science does not support the claim that infants and toddlers are too young to have serious mental-health problems. Young children who have experienced significant maltreatment

# Science does not support the claim that infants and toddlers are too young to have serious mental-health problems.

exhibit an early childhood equivalent of posttraumatic stress disorder, which presents a predictable array of clinical symptoms that are amenable to successful therapeutic intervention.55 (See Working Paper 6, "Mental Health Problems in Early Childhood Can Impair Learning and Behavior for Life.")

# The Science-Policy Gap

THE FACT THAT YOUNG CHILDREN HAVE FEELINGS is old news. The extent to which infants can experience deep emotional pain as a result of early traumas and losses is less understood. The realization that young children can have serious mental-health problems, including anxiety disorders and signs of depression accompanied by the same kind of brain changes seen on electroencephalograms in clinically depressed adults, is startling news to most people. 40,44,56,57

The fact that significant and prolonged emotional distress can affect the emerging architecture of a young child's brain should be a sobering wake-up call for society as a whole. Despite the availability of rich and extensive knowledge on the emotional and social development

of young children, including its underlying neurobiology, current early-childhood policies focus largely on cognition, language, and early literacy. Policies addressing children's emotional and behavioral needs have been the exception, not the rule. This gap between what we "know" about healthy emotional development and the management of behavioral difficulties, and what we "do" through public policies and programs, is illustrated by the following examples:

Uneven availability of support for parents and providers of early care and education to deal with common, age-appropriate behavioral challenges, such as discipline and limit setting.<sup>58</sup>

Limited caregiver and teacher training to evaluate and deal with children who present significant emotional and/or behavioral problems in early care and education programs. This is particularly alarming in the face of recent evidence of dramatic increases in prescriptions for behavior-modifying medications to treat preschoolers.<sup>59,60</sup>

Minimal expertise in early childhood development or "infant mental health" within

child-welfare agencies that assess and treat children who have been the victims of serious maltreatment, despite extensive evidence that very young children can experience debilitating anxiety and trauma from parental abuse or neglect or from witnessing violence in their family or neighborhood, as well as data illustrating that early interventions can moderate the effects of these traumas.<sup>61</sup>

# **Implications for Policy and Programs**

THE SCIENCE OF EARLY CHILDHOOD DEVELopment is sufficiently mature at the present time to support a number of welldocumented, evidence-based implications for those who develop and implement policies that affect the health and well-being of young

All early childhood programs, including Head Start, must balance their focus on cognition and literacy skills with significant attention to emotional and social development.

children. Five compelling messages are particularly worthy of thoughtful consideration:

All early childhood programs, including Head Start, must balance their focus on cognition and literacy skills with significant attention to emotional and social development. Children clearly need the social and emotional capabilities that enable them to sit still in a classroom, pay attention, and get along with their classmates just as much as they need the cognitive skills required to master the reading and math concepts taught in kindergarten.<sup>62</sup>

The rich and growing science of early emotional and social development must be incorporated into services to support parents who are struggling to manage routine behavioral difficulties in their young children, as well as those who are trying to figure out whether, when, and how to deal with more serious social or emotional problems.<sup>63</sup>

Providers of early care and education must have sufficient knowledge and skills to help children who present common behavior problems early on, particularly those who exhibit significant aggression or difficulties with attention and "hyperactivity." The achievement of this goal requires a two-pronged approach. First, greater attention must be focused on the social and emotional development of children in both pre-professional training programs and continuing professional education. Second, all early childhood programs must have access to specialized mental-health services that have professionals available to meet the needs of young children whose problems cannot be addressed adequately by front-line staff.<sup>19</sup>

Expertise in early identification, assessment, and clinical treatment must be incorporated into existing intervention programs to address the complex and currently unmet needs of young children with serious mental-health problems such as depression, anxiety, and significant antisocial behaviors. Central to this challenge is the need to accurately differentiate transient emotional difficulties that reflect a "phase" that the child will outgrow from diagnosable disorders that require clinical treatment.<sup>19</sup>

All child-welfare agencies that have responsibility for investigating suspected abuse or neglect must include a sophisticated assessment of the child's developmental status, including cognitive, linguistic, emotional, and social competence. This could be accomplished through closer collaboration between child-protective services and early intervention programs

for children with developmental delays or disabilities, as mandated by the Keeping Children and Families Safe Act of 2003 (Public Law 108-36).64

THESE IMPLICATIONS FOR POLICY AND PRACTICE are striking in their simplicity, the extent to which they reflect common sense, and their solid grounding in the science of early childhood and brain development. Closing the science-policy gap as it affects the future of our children, and therefore our society, should be an important priority for all who are engaged in public life.

# References

- 1. Saarni, C., Mumme, D.L., & Campos, J.J. (1998). Emotional development: Action, communication, and understanding. In W. Damon (Ed.), & N. Eisenberg, Handbook of Child Psychology, Vol. 3, (5th Ed.), Social, emotional and personality development (pp. 237-309). New York: Wiley.
- Thompson, R.A., & Lagattuta, K. (2006). Feeling and understanding: Early emotional development. In K. McCartney & D. Phillips (Eds.), The Blackwell Handbook of Early Childhood Development (pp. 317-337). Oxford, UK: Blackwell.
- 3. Thompson, R.A. (1994). Emotion regulation: A theme in search of definition. In N.A. Fox (Ed.), The development of emotion regulation and dysregulation: Biological and behavioral aspects. Monographs of the Society for Research in Child Development, 59(2-3), 25-52 (Serial no. 240).
- 4. Thompson, R.A. (2001). Development in the first years of life. The Future of Children, 11(1), 20-33.
- 5. Collins, W.A., & Laursen, B. (1999). Relationships as developmental contexts. The Minnesota Symposia on Child Psychology, Vol. 30. Mahwah, NJ: Erlbaum.
- Dunn, J. (1993). Young Children's Close Relationships: Beyond attachment. Newbury Park, CA: Sage.
- 7. Cassidy, J. & P.R. Shaver (Eds.) (1999). Handbook of Attachment: Theory, research, and clinical applications (pp. 89-111). New York: Guilford.
- 8. Thompson, R.A. (1998). Early sociopersonality development. In W. Damon (Ed.), & N. Eisenberg (Vol. Ed.), Handbook of Child Psychology, Vol. 3, (5th Ed.), Social, emotional, and personality development (pp. 25-104). New York: Wiley.
- Berscheid, E., & Reis, H.T. (1998). Attraction and close relationships. In D.T. Gilbert, S.T. Fiske, & G. Lindzey (Eds.), Handbook of Social Psychology, Vol. 1, (2nd Ed.). New York: McGraw-Hill.
- 10. Reis, H.T., Collins, W.A., & Berscheid, E. (2000). Relationships in human behavior and development. Psychological Bulletin, 126, 844-872.
- 11. Denham, S. (1998). Emotional Development in Young Children. New York: Guilford.
- 12. Harris, P.L. (1989). Children and Emotion: The development of psychological understanding. Oxford, UK: Blackwell.
- 13. LeDoux, J. (2000). Emotion circuits in the brain. Annual Review of Neuroscience, 23, 155-184.
- 14. Panksepp, J. (1998). Affective Neuroscience. London: Oxford University Press.
- 15. Panksepp, J. (2000). Developing mechanisms of selfregulation. Development and Psychopathology, 12(3), 427-442.

- 16. Dawson, G.,& Fischer, K.W. (Eds.) (1994). Human Behavior and the Developing Brain. New York: Guilford.
- 17. Gunnar, M.R., & Davis, E.P. (2003). Stress and emotion in early childhood. In R.M. Lerner & M.A. Easterbrooks (Eds.), Handbook of Psychology, Vol. 6. Developmental Psychology (pp. 113-134). New York: Wiley.
- 18. Fogel, A. (1993). Developing Through Relationships: Origins of communication, self, and culture. Chicago: University of Chicago Press.
- 19. Shonkoff, J.P., & Phillips, D. (Eds.) (2000). From Neurons to Neighborhoods: The science of early childhood development. Committee on Integrating the Science of Early Childhood Development. Washington, DC: National Academy Press.
- 20. Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. In N.A. Fox (Ed.), The development of emotion regulation and dysregulation: Biological and behavioral aspects. Monographs of the Society for Research in Child Development, 59(2-3), 228-249 (Serial
- 21. Lewis, M. (2000). Self-conscious emotions: Embarrassment, pride, shame, and guilt. In M. Lewis & J.M. Haviland-Jones (Eds.), Handbook of Emotions (pp. 563-573). New York: Guilford.
- 22. Banerjee, M. (1997). Peeling the onion: A multilayered view of children's emotional development. In S. Hala (Ed.), The Development of Social Cognition (pp. 241-272). Hove, UK: Psychology Press.
- 23. Wellman, H.M., Harris, P.L., Banerjee, M., & Sinclair, A. (1995). Early understanding of emotion: Evidence from natural language. Cognition and Emotion, 9, 117-149.
- 24. Eisenberg, N. & Morris, A.S. (2002). Children's emotionrelated regulation. In R. Kail (Ed.), Advances in Child Development and Behavior, Vol. 30 (pp. 190-229). San
- 25. Buss, K.A., & Goldsmith, H.H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. Child Development, 69, 359-374.
- 26. Eisenberg, N., Fabes, R., Guthrie, I., & Reiser, M. (2000). Dispositional emotionality and regulation: Their role in predicting quality of social functioning. Journal of Personality and Social Psychology, 78, 136-157.
- 27. Kopp, C.B. (1989). Regulation of distress and negative emotions: A developmental view. Developmental Psychology, 25(3), 343-355.
- 28. Barrett, K. (1998). The origins of guilt in early childhood. In J. Bybee (Ed.), Guilt and Children (pp. 75-90). San Diego: Academic.

- Lagattuta, K.H., & Wellman, H.M. (2002). Differences in early parent-child conversations about negative versus positive emotions: Implications for the development of emotion understanding. *Developmental Psychology*, 38, 564-580.
- Davidson, R.J., Lewis, M., Alloy, L.B., Amaral, D.G., Bush, G., Cohen, J., et al. (2002). Neural and behavioral substrates of mood and mood regulation. *Biological Psychiatry*, 52(6), 478-502.
- 31. Posner, M., & Rothbart, M. (2000). Developing mechanisms of self-regulation. *Development and Psychopathology*, 12(3), 427-442.
- 32. Damasio A.R. (1999). *The Feeling of What Happened*. New York: Harcourt Brace.
- Davis, M. (1992). The role of the amygdala in fear and anxiety. Annual Review of Neuroscience, 15, 353-375.
- 34. LeDoux, J.E. (1996). *The Emotional Brain*. New York: Simon & Schuster.
- Bush, G., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends* in Cognitive Sciences, 4(6), 215-222.
- Rothbart, M.K., & Bates, J.E. (1998). Temperament. In W. Damon (Ed.), & N. Eisenberg (Vol. Ed.), Handbook of Child Psychology Vol. 3, (5th Ed.), Social, emotional and personality development (pp. 105-176). New York: Wiley.
- 37. Rothbart, M.K., Derryberry, D., & Posner, M.I. (1994). A psychobiological approach to the development of temperament. In J.E. Bates & T.D. Wachs (Eds.), Temperament: Individual differences at the interface of biology and behavior (pp. 83-116). Washington, DC: American Psychological Association.
- Kochanska, G. (1997). Multiple pathways to conscience for children with different temperaments: From toddlerhood to age 5. *Developmental Psychopathology*, 33, 228-240.
- Teti, D.M., & Candelaria, M.A. (2002). Parenting competence. In M.H. Bornstein (Ed.), *Handbook of Parenting*, *Vol. 4*. Social conditions and applied parenting (2nd Ed.) (pp. 149-180). Mahwah, NJ: Erlbaum.
- Shaw, D.S., Owens, E.B., Giovannelli, J., Winslow, E.B. (2001). Infant and toddler pathways leading to early externalizing disorders. *Journal of the American Academy* of Child & Adolescent Psychiatry, 40, 36-43.
- 41. Ashman, S.B., & Dawson, G. (2002). Maternal depression, infant psychobiological development, and risk for depression. In S.H. Goodman & I.H. Gotlib (Eds.), *Children of Depressed Parents* (pp. 37-58). Washington, DC: American Psychological Association.
- 42. Rubin, K.H., Burgess, K.B., Dwyer, K.M., & Hastings, P.D. (2003). Predicting preschoolers' externalizing behaviors from toddler temperament, conflict, and maternal negativity. *Developmental Psychology*, 39, 164-176.
- Vasey, M.W., & Dadds, M.R. (2001). The Developmental Psychopathology of Anxiety. London: Oxford University Press.
- 44. Dawson, G., & Ashman, D.B. (2000). On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk of affective disorder. In C.A. Nelson (Ed.), The effects of early adversity on neurobehavioral development. Minnesota Symposia on Child Psychology, Vol. 31 (pp. 245-279). Mahwah, NJ: Erlbaum.
- 45. Cummings, E.M., & Davies, P. (1994). *Children and Marital Conflict*. New York: Guilford.

- Reid, J.B., Patterson, G.R., & Snyder, J. (2002). Antisocial Behavior in Children and Adolescents: A developmental analysis and model for intervention. Washington, DC: American Psychological Association.
- Thompson, R.A., & Calkins, S. (1996). The doubleedged sword: Emotional regulation for children at risk. Development and Psychopathology, 8(1), 163-182.
- Davies, P.T., & Forman, E.M. (2002). Children's patterns of preserving emotional security in the interparental subsystem. *Child Development*, 73, 1880-1903.
- Glaser, D. (2000). Child abuse and neglect and the brain

   A review. *Journal of Child Psychology and Psychiatry*, 41, 97-118.
- De Bellis, M.D., Keshavan, M.S., Clark, D.B., Casey, B.J, Giedd, J.B., Boring, A.M., et al. (1999). Developmental traumatology, Part 2: Brain development. *Biological Psychiatry*, 45, 1271-1284.
- Denham, S.A., Blair, K.A., DeMulder, E., Levitas, J., Sawyer, K., Auerbach-Major, S., & Queenan, P. (2003). Preschool emotional competence: Pathway to social competence. *Child Development*, 74, 238-256.
- Halberstadt, A.G., Denham, S.A., & Dunsmore, J.C. (2001). Affective social competence. *Social Development*, 10, 79-119.
- 53. Rubin, K.H., Coplan, R.J., Nelson, L.J., Cheah, C.S.L., & Lagace-Seguin, D.G. (1999). Peer relationships in childhood. In M.H. Bornstein & M.E. Lamb (Eds.), *Developmental Psychology: An advanced textbook* (4th Ed.) (pp. 451-501). Mahwah, NJ: Erlbaum.
- 54. Graham-Berman, S.A., & Hughes, H.M. (2003). Intervention for children exposed to interparental violence (IPV): Assessments of needs and research priorities. Clinical Child & Family Psychology Review, 6, 189-204.
- Scheeringa, M.S., & Zeanah, C.H. (1995). Symptom expression and trauma variables in children under 48 months of age. *Infant Mental Health Journal*, 16(4), 259-270.
- Dawson, G., Frey, K., Panagiotides, H., Yamada, E. Hessl, D., & Osterling, J. (1999). Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. Child Development, 70, 1058-1066.
- Shaw, D.S., Gilliom, M., Ingoldsby, E.M., & Nagin, D.S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology*, 39, 189-200.
- 58. Knitzer, J. (2001). Building Services and Systems to Support the Healthy Emotional Development of Young Children: An action guide for policymakers. New York: National Center for Children in Poverty, Columbia University Mailman School of Public Health.
- 59. Knitzer, J. (2000). Early childhood mental health services: A policy and systems development perspective. In J.P. Shonkoff & S.J. Meisels (Eds.), *Handbook of Early Childhood Intervention* (2nd Ed.) (pp. 416-438). New York: Cambridge University Press.
- 60. Zito, J., Safer, D., dosReis, S., Gardner, J., Boles, M., & Lynch, F. (2000). Trends in the prescribing of psychotropic medications to preschoolers. *Journal of the American Medical Association*, 283, 1025-1030.
- 61. Melton, G.B., & Thompson, R.A. (2002). The conceptual foundation: Why child protection should be neighborhood-based and child-centered. In G.B. Melton, R.A. Thompson, & M.A. Small (Eds.), Toward a Child-centered, Neighborhood-based Child Protection System: A report of the Consortium on Children, Families, and the Law (pp. 3-27). Westport, CT: Praeger.

- 62. Thompson, R.A., & Raikes, H.A. (2007). Early socioemotional development and the roots of school readiness. In J. Knitzer, R. Kaufmann, & D. Perry (Eds.), Early Childhood Mental Health (pp.13-35). Baltimore, MD: Paul H. Brookes Publishing Co.
- 63. Brooks-Gunn, J., Berlin, L.J., & Fuligni, A.S. (2000). Early childhood intervention programs: What about the family? In J.P. Shonkoff & S.J. Meisels (Eds.), Handbook of Early Childhood Intervention (2nd Ed.) (pp. 549-577). New York: Cambridge University Press.
- 64. Thompson, R.A. & Flood, M.F. (2002). Toward a childoriented child protection system. In G.B. Melton, R.A. Thompson, & M.A. Small (Eds.), Toward a Childcentered, Neighborhood-based Child Protection System: A report of the Consortium on Children, Families, and the Law (pp. 155-194). Westport, CT: Praeger.

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# Excessive Stress Disrupts the Architecture of the Developing Brain

**WORKING PAPER 3** 

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# The Issue

THE ABILITY TO COPE WITH NOVEL AND/OR POTENTIALLY THREATENING SITUATIONS, SUCH AS AN unfamiliar environment or physical danger, is essential to survival. This capacity is built into specific brain circuits whose development is influenced by multiple experiences beginning early in life. Environmental stimuli that activate these circuits are often referred to as stressors, and stress reactions are the body's chemical and neural responses that promote adaptation.

Stressful events can be harmful, tolerable, or beneficial, depending on how much of a bodily stress response they provoke and how long the response lasts. These, in turn, depend on whether the stressful experience is controllable, how often and for how long the body's stress system has been activated in the past, and whether the affected child has safe and dependable relationships to turn to for support. Thus, the extent to which stressful events have lasting adverse effects is determined more by the individual's response to the stress, based in part on past experiences and the availability of a supportive adult, than by the nature of the stressor itself. This matters because a child's ability to cope with stress in the early years has consequences for physical and mental health throughout life. Furthermore, categorizing the nature and severity of early stressful experiences helps us make better judgments about the need for interventions that reduce the risk for later negative impacts.

Toxic stress refers to strong, frequent or prolonged activation of the body's stress management system. Stressful events that are chronic, uncontrollable, and/or experienced without the child having access to support from caring adults tend to provoke these types of toxic stress responses. Studies indicate that such stress responses can have an adverse impact on brain architecture. In the extreme, such as in cases of severe, chronic abuse, toxic stress may result in the development of a smaller brain. Less extreme exposure to toxic stress can change the stress system so that it responds at lower thresholds to events that might not be stressful to others, thereby increasing the risk of stress-related physical and mental illness.

**Tolerable stress** refers to stress responses that could affect brain architecture but generally occur for briefer periods that allow time for the brain to recover and thereby reverse potentially harmful effects. In addition to their relative brevity, one of the critical ingredients that make stressful events tolerable rather than toxic is the presence of supportive adults who create safe environments that help children learn to cope with and recover from major adverse experiences, such as the death or serious illness of a loved one, a frightening accident, or parental separation or divorce. In some circumstances, tolerable stress can even have positive effects.

# A child's ability to cope with stress in the early years has consequences for physical and mental health throughout life.

Nevertheless, it also can become toxic stress in the absence of supportive relationships.

Positive stress refers to moderate, shortlived stress responses, such as brief increases in heart rate or mild changes in the body's stress hormone levels. This kind of stress is a normal part of life, and learning to adjust to it is an essential feature of healthy development. Adverse events that provoke positive stress responses tend to be those that a child can learn to control and manage well with the support of caring adults, and which occur against the backdrop of generally safe, warm, and positive relationships. The challenge of meeting new people, dealing with frustration, entering a new child care setting, getting an immunization, and overcoming a fear of animals all can be positive stressors if a child has the support needed to develop a sense of mastery. This is an important part of the normal developmental process.

# What Science Tells Us

SCIENTIFIC KNOWLEDGE IN THIS AREA COMES from research on animals as well as humans. These extensive bodies of work have generated common principles of developmental biology that support valid generalizations across species and reasonable hypotheses about humans based on consistent findings from animal studies. The ability to control exposure to negative life experiences in animals makes it additionally possible to conduct studies of the impacts of more graded forms of stress on the brain than could be done in human research.

The capacity to deal with stress is controlled by a set of highly inter-related brain circuits and hormonal systems that are specifically designed

# The neural circuits for dealing with stress are particularly malleable (or "plastic") during the fetal and early childhood periods.

to deal adaptively with environmental challenges. When an individual feels threatened, stress hormones are produced that convert the physical or emotional stress into chemical signals that are sent throughout the body as well as to the brain.

The neural circuits for dealing with stress are particularly malleable (or "plastic") during the fetal and early childhood periods. Early experiences shape how readily they are activated and how well they can be contained and turned off. Toxic stress during this early period can affect developing brain circuits and hormonal systems in a way that leads to poorly controlled stress-responsive systems that will be overly reactive or slow to shut down when faced with threats throughout the lifespan. <sup>1,2</sup>

Well-functioning brain systems that respond to stress are essential to preserve life. However, like the immune system, which defends the body against threatening infections but can cause autoimmune disease when it turns against the body's own cells, a poorly controlled response to stress can be damaging to health and well-being if activated too often or for too long.<sup>3</sup>

Frequent or sustained activation of brain systems that respond to stress can lead to heightened vulnerability to a range of behavioral and physiological disorders over a lifetime. These undesirable outcomes can include a number of stress-related disorders affecting both mental (e.g., depression, anxiety disorders, alcoholism, drug abuse) and physical (e.g., cardiovascular disease, diabetes, stroke) health.<sup>3</sup>

Stress responses include activation of a variety of hormone and neurochemical systems throughout the body. Two hormonal systems have received extensive attention in this regard: (1) the sympathetic-adrenomedullary (SAM) system, which produces adrenaline in the central part of the adrenal gland, and (2) the hypothalamic-pituitary-adrenocortical (HPA) system, which produces cortisol in the outer shell of the adrenal gland.<sup>4</sup> Both chemicals are produced under normal circumstances and help prepare the body for coping with stressors.

Adrenaline production occurs in response to many forms of acute stress. It mobilizes energy stores and alters blood flow, thereby allowing the body to effectively deal with a range of stresses. Its release is essential to survival.<sup>4</sup>

Cortisol also is produced in response to many forms of stress, and likewise helps the body cope effectively with adverse situations. It also mobilizes energy stores, as well as suppresses immune responses, when it is released acutely. Longerterm effects of cortisol include regulation of gene expression in neural circuits involved in modulating stress responsiveness, emotion, and memory.<sup>4</sup>

Sustained or frequent activation of the hormonal systems that respond to stress can have serious developmental consequences, some of which may last well past the time of stress exposure. For example, when children experience toxic stress, their cortisol levels remain elevated for prolonged periods of time. Both

animal and human studies show that long-term elevations in cortisol levels can alter the function of a number of neural systems, and even change the architecture of regions in the brain that are essential for learning and memory.<sup>5,6</sup>

MUCH OF WHAT WE KNOW ABOUT THE SPECIFIC effects of stress on the developing architecture of the brain comes from research on rodents, non-human primates, and other animal species. These studies indicate that:

Increases in the level of cortisol in the brain actually can turn specific genes "on" or "off" at specific times and locations.7 Examples include regulation of the glucocorticoid receptor gene, which affects the long-term responsiveness of the brain to stress-induced cortisol release, and the myelin basic protein gene, which is involved in regulating the development of the "insulation" that increases the efficiency of nerve signal transmission.8,9

High, sustained levels of cortisol or corticotropin-releasing hormone (CRH), which is the brain chemical that regulates the HPA system, result in damage to a part of the brain called the hippocampus. This can lead to impairments in learning, memory, and the ability to regulate certain stress responses in both young and adult animals.10

Significant maternal stress during pregnancy and poor maternal care during infancy both affect the developing stress system in young animals and alters genes that are involved in brain development. Pregnant females who experience exceptionally high levels of stress have offspring that are more fearful and more reactive to stress themselves. Young animals that experience inattentive maternal care have similar problems and show impaired production of brain growth factors important for brain development and repair.11,12 Both groups of animals also have impaired memory and learning abilities, and they experience more aging-related memory and cognitive deficits in adulthood.3,13

Positive experiences after infancy in young animals, such as being exposed to an environment rich in opportunities for exploration and social play, have been shown to compensate to some degree for the negative behavioral consequences of prenatal stress and postnatal neglect. This compensation actually involves adaptive changes in both the architecture and the chemistry of the developing brain (such as reversal of the effects of mild adversity on stress hormone output), although deprivation-induced changes in some of the regulatory components of the stress system (e.g., reduced glucocorticoids receptors in the hippocampus) are more resistant to change.14

Individual responses to early stressful experiences can vary dramatically. This variability is thought to be related to differences among animals in the expression of so-called "vulnerability genes," which make it more likely that early stressors will lead to subsequent problems in stress hormone

The relationships children have with their caregivers play critical roles in regulating stress hormone production during the early years of life.

regulation and behavioral difficulties. In such cases, positive early caregiving can decrease the likelihood of these adverse outcomes, demonstrating that beneficial environmental influences can moderate the impact of genetic vulnerability.<sup>15</sup>

BUILDING ON THE EXTENSIVE KNOWLEDGE gained from animal research, studies of children are beginning to document a compelling story about the relation between early stress experiences and human development. The following findings appear to be particularly salient:

The relationships children have with their caregivers play critical roles in regulating stress hormone production during the early years of life. Those who experience the benefits of secure relationships have a more controlled stress hormone reaction when they are upset or frightened. This means that they are able to explore the world, meet challenges, and be frightened at times without sustaining the adverse neurological impacts of chronically elevated levels of hormones such as cortisol that increase reactivity of selected brain systems to stress and threat. In contrast, children whose relationships are

insecure or disorganized demonstrate higher stress hormone levels when they are even mildly frightened. This results in an increased incidence of elevated cortisol levels which may alter the development of brain circuits in ways that make some children less capable of coping effectively with stress as they grow up.<sup>2</sup>

Research has shown that the presence of a sensitive and responsive caregiver can prevent elevations in cortisol among toddlers, even in children who tend to be temperamentally fearful or anxious. <sup>16</sup> Thus, sensitive and responsive caregiving from a parent or a child care provider

# Young children who experience debilitating anxiety and trauma as a result of personal abuse or neglect are amenable to early treatment.

can serve as a powerful buffer against stress hormone exposure, even in children who might otherwise be highly vulnerable to stress-system activation.

The quality of the early care and education that many young children receive in programs

outside their homes also plays an important role in whether (and to what extent) their brains are exposed to elevated stress hormones early in life.

For example, toddlers and young preschoolers show increases in cortisol as the child care day progresses, while older preschoolers and school-aged children can manage long hours in care without activating their stress system.<sup>17</sup> Young children in poorer quality child care show larger elevations, however, than those in better quality care.<sup>18</sup>

Children who grow up in families facing economic hardship commonly exhibit elevated cortisol levels. These elevations are often exacerbated when mothers experience symptoms of depression. <sup>19,20,21</sup> Recent research also has demonstrated that a mother's depression during her child's early years increases the child's cortisol reactions to adverse family conditions later in childhood. <sup>22,23,24</sup>

Young children who are neglected or maltreated have abnormal patterns of cortisol production that can last even after the child has been moved to a safe and loving home. <sup>25,26</sup> This is especially true for children who show symptoms of post-traumatic stress, even if their behavior is not sufficient to warrant a definitive diagnosis of post-traumatic stress disorder. <sup>27,28,29</sup>

# Popular Misrepresentations of Science

AS THE PUBLIC'S APPETITE FOR SCIENTIFIC INformation about the development of young children is stimulated by exciting new findings, the risk of exposure to misleading or, frankly, irresponsible messages grows. Within this context, it is essential that we distinguish scientific fact from popularly accepted fiction.

Science does not support the claim that infants and young children are too young to be affected by significant stresses that negatively affect their family and caregiving environments. In fact, animal studies have shown that adverse early infant experiences (e.g., neglectful maternal care), as well as serious disruptions of the pre-natal environment (e.g., drug and alcohol exposure), can

lead to short-term neurobehavioral and neurohormonal changes in offspring that may have long-term adverse effects on memory, learning, and behavior throughout life. Human studies suggest that similar effects may be seen in infants and children.<sup>30</sup>

Notwithstanding the preceding statement, there is no credible scientific evidence that supports the conclusion that young children who have been exposed to significant early stresses will always develop stress-related disorders. In both animal and human studies, interventions that provide more appropriate and supportive care help to stimulate positive growth and prevent poor outcomes. <sup>14,25,31</sup>

# The Science-Policy Gap

THE FACT THAT MANY YOUNG CHILDREN ARE exposed to significant stresses is old news. How different aspects of a child's environment can be a source of continuous stress, and the degree to which children's past developmental experiences influence their biological responsiveness to later stressful conditions are not appreciated by most adults. The realization that stresses experienced by parents and other caregivers can affect a child's developing brain architecture and chemistry in a way that makes some children more susceptible to stress-related disorders later in life is startling news to most people.

A rich and growing scientific knowledge base illuminates the multiple adverse effects of early life stresses, including their long-term impacts on how individuals cope with stress throughout the life cycle. Yet little attention has been paid to the development and implementation of strategies to reduce stressors that affect everyday life for families with young children. This gap between what we know about the potentially harmful developmental impacts of stresses experienced by both caregivers and children, and what we do to promote healthy coping and adaptation through informal supports, voluntary workplace practices, and formal public policies and programs, is illustrated by the following examples:

Limited availability of family leave after the birth or adoption of a baby, and little financial support for parents who wish to stay at home with their newborns but do not have the economic resources to make ends meet in the absence of paid employment. In some circumstances, this creates situations where the supportive relationships necessary to help very young children manage stress are intermittent or seriously compromised.32,33,34,35

Limited supports for working parents at all income levels who are struggling to balance the demands and responsibilities of work and raising children. These balancing challenges are particularly difficult for low-income, working families whose economic security depends on multiple low-wage jobs, often during non-

standard working hours, and for families whose children have chronic health problems or special developmental needs that require multiple medical appointments and skilled child care. In such circumstances, some young children are subjected to excessive stress that can have lasting effects on their health and well-being.31

Limited efforts to reduce high job turnover in child care programs, which affects the quality of relationships between adults and the children under their care. This is a particularly serious problem for those children whose family's socioeconomic circumstances limit their access to better-quality programs that have well trained, adequately compensated, and more stable staff. 35,36,37,38

Limited availability of expert help for parents and providers of early care and education who are struggling to manage behavioral difficulties in young children. This is particularly problematic in the face of recent data on expulsion of children from preschool programs, which indicate the extent to which staff members are unable and/or unwilling to deal with challenging behavioral problems.39 The growing "offlabel" use of prescription drugs, particularly stimulant and anti-depressant medications, for increasingly younger children with emotional or behavioral difficulties is another sign of the extent to which parents are putting greater pressure on professionals to provide more help in managing behavior problems during the preschool years.40

Limited access to clinical expertise in mental health for very young children and their families. This is particularly problematic in child welfare agencies that are mandated to assess children who are coping with toxic stress that can have lasting adverse effects on their well-being. Most important, young children who experience debilitating anxiety and trauma as a result of personal abuse or neglect, or who witness violence in their family or neighborhood, are amenable to early treatment. 41,42

# **Implications for Policy and Programs**

THE SCIENCE OF EARLY CHILDHOOD DEVELOPment, including knowledge about the impact of stress on the developing brain, is sufficiently mature to support a number of evidencebased implications for those who develop and implement policies that affect the health and well-being of young children. To this end, both public and private actions can prevent the kinds of adverse circumstances that are capable of derailing healthy development, as well as increase the likelihood that effective interventions

# Both public and private actions can prevent the kinds of adverse circumstances that are capable of derailing healthy development.

will reduce potential damage to a young child's developing brain architecture and thereby promote greater resilience. Five points are particularly worthy of thoughtful consideration.

The rich and growing scientific understanding of how individuals cope with stress should be used to strengthen a range of informal supports and formal services to bolster parents who are struggling to manage the challenges of raising their children. These could be provided through varying combinations of extended family support, community-based volunteer efforts, flexible work-place policies, and publicly funded programs.<sup>43</sup>

The availability of affordable expert assistance should be expanded for parents and providers of early care and education to provide them with sufficient knowledge and skills to help young children who have symptoms related to abnormal stress responses. This is particularly important for children who exhibit excessive fears, aggressive behavior, or difficulties with attention and "hyperactivity." 35,41

Expertise in the identification, assessment, and clinical treatment of young children with serious, stress-related, mental health problems (as well as access to mental health services for mothers with

depression) should be incorporated into existing intervention programs to address these complex and widely unmet needs. Research indicates that young children can experience a range of mental health impairments that used to be viewed solely as adult problems, such a depression, anxiety disorders, and anti-social behaviors.<sup>35</sup>

Investigations of suspected child abuse or neglect should include a sophisticated assessment of the child's developmental status, including cognitive, linguistic, emotional, and social competence. This could be accomplished through closer collaboration between child welfare services and early intervention programs for children with developmental delays or disabilities, 44 as mandated by the Keeping Children and Families Safe Act of 2003 and the recent reauthorization of the Individuals with Disabilities Education Act (IDEA).

Children of mothers who are receiving welfare payments or related services under the Temporary Assistance to Needy Families (TANF) program represent another identified group whose experience with stress is likely to exceed that of the **general population.** In this context, it is difficult to justify the extent to which public discussion about welfare reform focuses primarily on maternal employment and other adult behaviors, while the special needs of the young children in these families are afforded relatively little attention. Our knowledge of the importance of supportive relationships as buffers against the adverse effects of stress on the architecture of the developing brain indicates the need for serious reconsideration of mandated employment for mothers of very young children, particularly when access to high quality child care is not assured. Research also underscores the importance of timely assessments and intervention services (when indicated) for children living in stressful environments who show early signs of developmental difficulties. 45,46

# References

- 1. Zhang, T., Parent, T., Weaver, I., & Meaney, M. J. (2004). Maternal programming of individual differences in defensive responses in the rat. Annals of the New York Academy of Science, 1032, 85-103.
- 2. Loman, M., & Gunnar, M.R. (in press). Early experience and the development of stress reactivity and regulation in children. Neuroscience & Biobehavioral Reviews.
- 3. McEwen, B. S. (2008). Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. European Journal of Pharmacology, 583, 174-185.
- 4. Sapolsky, R.M., Romero, L.M., & Munck, A. (2000). How do glucorticoids influence stress responses? Integrating permissive, suppressive, stimulatory and preparative actions. Endocrine Reviews, 21(1), 55-89.
- 5. Lupien, S.J., de Leon, M.J., Santi, S.D., Convit, A., Tarshish, C., Nair, N.P.V., Thakur, M., McEwen, B., Hauger, R.L, & Meaney, M.J. (1998). Cortisol levels during human aging predict hippocampal atrophy and memory deficits. Nature Neuroscience, 1(1), 69-73.
- 6. McEwen, B. S., & Sapolsky, R. M. (1995). Stress and cognitive function. Current Opinion in Neurobiology, 5(2), 205-216.
- 7. De Kloet, E.R., Rots, N.Y., & Cools, A.R. (1996). Braincorticosteroid hormone dialogue: Slow and persistent. Cellular and Molecular Neurobiology, 16(3), 345-356.
- 8. Gunnar, M., & Vazquez, D. M. (2006). Stress neurobiology and developmental psychopathology. In D. Cicchetti & D. Cohen (Eds.), Developmental Psychopathology, 2nd Edition, Volume 2: Developmental Neuroscience. New York: Wiley.
- 9. Weaver, I.C., Diorio, J., Seckl, J.R., Szyf, M., & Meaney, M.J. (2004) Early environmental regulation of hippocampal glucocorticoid receptor gene expression: Characterization of intracellular mediators and potential genomic target sites. Annals of the New York Academy of Sciences, 1024, 182-212.
- 10. Brunson, Grigoriadis D.E., Lorang M.T., & Baram T.Z. (2002) Corticotropin-releasing hormone (CRH) downregulates the function of its receptor (CRF1) and induces CRF1 expression in hippocampal and cortical regions of the immature rat brain. Experimental Neurology, 176(1), 75-86.
- 11. Roceri, M., Cirulli, F., Pessina, C., Peretto, P., Racagni, G., & Riva, M. A. (2004). Postnatal repeated maternal deprivation produces age-dependent changes in brain-derived neurotrophic factor expression in selected rat brain regions. Biological Psychiatry, 55, 708-714.
- 12. Roceri, M., Hendriks, W., Racagni, G., B.A., E., & Riva, M. A. (2002). Early maternal deprivation reduces the expression of BDNF and NMDA receptor subunits in rat hippocampus. Molecular Psychiatry, 7, 609-616.
- 13. Weinstock, M. (2001). Alterations induced by gestational stress in brain morphology and behaviour of the offspring. Progress in Neurobiology, 62(4), 427-451.
- 14. Francis, D., Diorio, J., Plotsky, P.M, & Meaney, M.J. (2002). Environmental enrichment reverses the effects of maternal separation on stress reactivity. Journal of Neuroscience, 22(18), 7840-7843.
- 15. Barr, C.S., Newman, T.K., Lindell, S., Shannon, C., Champoux, M., Lesch, K.P., Suomi, S., Goldman, D., Higley, J.D. (2004). Interaction between serotonin gene

- variation and rearing history in alcohol preference and consumption in female primates. Archives of General Psychiatry, 61(11), 1146-1152.
- 16. Nachmias, M., Gunnar, M. R., Mangelsdorf, S., Parritz, R., & Buss, K. A. (1996). Behavioral inhibition and stress reactivity: Moderating role of attachment security. Child Development, 67(2), 508-522.
- 17. Vermeer, H. J., & van IJzendoorn, M. H. (2006). Children's elevated cortisol levels at daycare: A review and meta-analysis. Early Childhood Research Quarterly, 21, 390-401.
- 18. Gunnar, M.R., Kryzer, E., VanRyzin, M.J., & Phillips, D. (in press). The rise in cortisol in family day care: Associations with aspects of care quality, child behavior, and child sex. Child Development.
- 19. Essex, M.J., Klein, M.H., Cho, E., & Kalin, N.H. (2002). Maternal stress beginning in infancy may sensitize children to later stress exposure: Effects on cortisol and behavior. Biological Psychiatry, 52(8), 776-784.
- Lupien, S., King, S., Meaney, M.J., McEwen, B.S. (2000). Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. Biological Psychiatry, 48(10), 976-980.
- 21. Lupien, S., King, S., Meaney, M.J., & McEwen, B.S. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. Development and Psychopathology, 13, 653-676.
- 22. Dawson, G. & Ashman, S.B. (2000). On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk for affective disorder. In C.A. Nelson (Ed.), The Effects of Adversity on Neurobehavioral Development: Minnesota Symposia on Child Psychology. (pp. 245-280). Mahwah, NJ: Lawrence Erlbaum & Assoc.
- 23. Ashman, S.B., Dawson, G., Panagiotides, H., Yamada, E., & Wilkins, C.W. (2002). Stress hormone levels of children of depressed mothers. Development and Psychopathology, 14(2), 333-349.
- 24. Jones, N.A., Field, T., & Fox, N.A.(1997). EEG activation in 1-month-old infants of depressed mothers. Development and Psychopathology, 9(3), 491-505.
- 25. Gunnar, M., Morison, S.J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. Development and Psychopathology, 13, 611-628.
- 26. Bruce, J., Fisher, P. A., Pears, K. C., & Levine, S. (2009). Morning cortisol levels in preschool-aged foster children: Differential effects of maltreatment type. Developmantal Psychobiology, 51, 14-23.
- 27. Carrion, V.G., Weems, C.F., Ray, R.D., Glaser, B., Hessl, D., & Reiss, A.L.. (2002). Duirnal salivary cortisol in pediatric posttraumatic stress disorder. Biological Psychiatry, 51(7), 575-582.
- 28. De Bellis, M.D., Baum, A.S., Birmaher, B., Keshavan, M.S., Eccard, C.H., Boring, A.M., Jenkins, F.J., & Ryan, N. (1999). Developmental traumatology, Part 1: Biological stress systems. Biological Psychiatry, 9, 1259-1270.
- 29. De Bellis, M.D., Keshavan, M.S., Clark, D.B., Casey, B.J., Giedd, J.B., Boring, A.M., Jenkins, F.J., & Ryan, N. (1999). Developmental traumatology, Part 2: Brain development. Biological Psychiatry, 45, 1271-1284.

- 30. Gunnar, M. (2003). Integrating neuroscience and psychosocial approaches in the study of early experiences. In J.A. King, C. F. Ferris & I. I. Lederhendler (Eds.), Roots of Mental Illness in Children, 1008, 238-247. New York: New York Academy of Sciences.
- Bredy, T.W., Humpartzoomian, R.A., Cain, D.P., & Meaney, M.J.P. (2003). Partial reversal of the effect of maternal care on cognitive function through environmental enrichment. *Neuroscience*, 118(2), 571-576.
- Kamerman, S.,& Kahn,A. (1995). Starting Right: How America neglects its young children and what we can do about it. New York: Oxford University Press.
- 33. Waldfogel, J. (1999). The impact of the Family and Medical Leave Act. *Journal of Policy Analysis and Management*, 18(2), 281-302.
- Waldfogel, J. (2001) International policies toward parental leave and child care. The Future of Children, 11(1), 99-111
- Shonkoff, J.P., & Phillips, D. (Eds.) (2000). From Neurons to Neighborhoods: The science of early childhood development. Committee on Integrating the Science of Early Childhood Development. Washington, D.C.: National Academy Press.
- Phillips, D., Mekos, D., Carr, S., & Abbott-Shim, M. (2000). Within and beyond the classroom door: Assessing quality in child care centers. *Early Childhood Research Quarterly*, 15(4), 475-496.
- NICHD Early Child Care Research Network (1996).
   Characteristics of infant child care: Factors contributing to positive caregiving. Early Childhood Research Quarterly, 11, 296-306.
- NICHD Early Child Care Research Network (2000).
   Characteristics and quality of child care for toddlers and preschoolers. Applied Developmental Science, 4(3), 116-125.
- Gilliam, W.S., & Shahar, G. (2006). Prekindergarten expulsion and suspension: Rates and predictors in one state. *Infants and Young Children*.
- Zito, J.M., Safer, D.J., dosReis, S., Gardner, J.F., Boles, M., & Lynch, F. (2000). Trends in the prescribing of psychotropic medications to preschoolers. *Journal of the American Medical Association*, 283(8),1025-1030.
- 41. Johnson, K., Knitzer, J., & Kaufmann, R. (2002). Making Dollars Follow Sense: Financing early childhood mental health services to promote healthy social and emotional development in young children. New York: National Center for Children in Poverty.
- 42. Melton, G.B., & Thompson, R.A. (2002). The conceptual foundation: Why child protection should be neighborhood-based and child-centered. In G.B. Melton, R.A. Thompson, & M.A. Small (Eds.), Toward a Child-centered, Neighborhood-based Child Protection System: A report of the Consortium on Children, Families, and the Law, (pp. 3-27). Westport, CT: Praeger.
- 43. Brooks-Gunn, J., Berlin, L.J., & Fuligni, A.S. (2000). Early childhood intervention programs: What about the family? In J.P. Shonkoff & S.J. Meisels (Eds.), *Handbook of Early Childhood Intervention* (2nd Ed.) (pp. 549-577). New York: Cambridge University Press.
- 44. Thompson, R.A., & Flood, M.F. (2002). Toward a child-oriented child protection system. In G.B. Melton, R.A. Thompson, & M.A. Small (Eds.) Toward a Child-centered, Neighborhood-based Child Protection System: A report of the Consortium on Children, Families, and the Law, (pp. 155-194). Westport, CT: Praeger.

- Duncan, G.,& Chase-Lansdale, L. (2002). For Better and For Worse: Welfare reform and the well-being of children and families. New York: Russell Sage.
- 46. Huston, A.C. (2002). Reforms and child development. *The Future of Children*, 12(1), 59-77.

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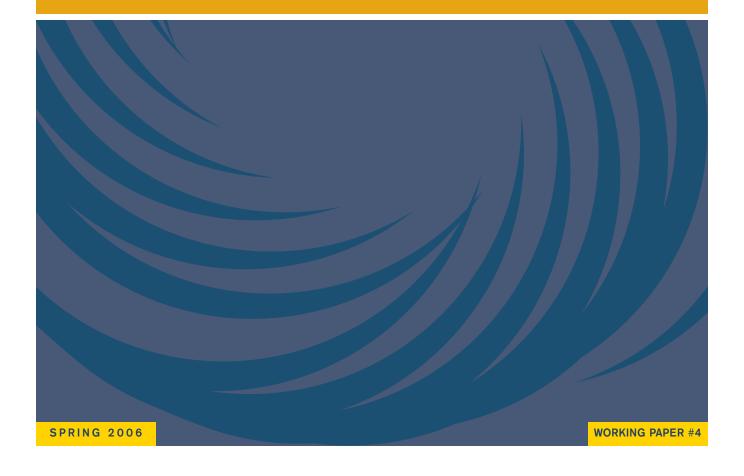
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# EARLY EXPOSURE TO TOXIC SUBSTANCES DAMAGES BRAIN ARCHITECTURE



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is a multidisciplinary collaboration of leading scientists in early childhood and early brain development. Its mission is to bring sound and accurate science to bear on public decision-making affecting the lives of young children.

For more information on the Council and the science of early childhood, please see www.developingchild.net.

#### THE ISSUE

Children develop in an environment of relationships<sup>1</sup>. They also develop in an environment of chemicals. Many of these chemicals, such as the nutrients in a well-balanced diet, are essential for good health. Others, such as lead in drinking water, are poisonous and can cause illness or death. Some chemicals that disrupt brain architecture are produced in our own bodies as a result of severe and prolonged stress<sup>2</sup>. Others enter through contaminants in the air that we breathe, the water that we drink, and the food that we eat.

Brain development begins well before birth and continues through the early adult years. The biology of that process is influenced by the genes that are passed on from the parents to the child, by the environment of the mother's womb, and by the world the child experiences during infancy and childhood, which can either weaken or strengthen the initial blueprint. Thus, brains are built over time, and the circumstances in which they are built are every bit as important as the initial architectural framework handed down by genetics.

Toxic substances have the capacity to disrupt the development of all of the body's organ systems. The nature and severity of that disruption depend upon the type of substance, the level and duration of exposure, and most important, on the timing during the developmental process. Early assaults can lead to a broad range of lifelong problems in both physical and mental health that impose devastating human and financial costs. This paper focuses on the effects of toxic exposures on the architecture of the developing brain. When it is relatively immature, the brain is particularly susceptible to adverse impacts on the formation of its basic circuits. During pregnancy, the developing brain is extremely sensitive to many chemicals. When certain substances reach dangerous levels at particularly sensitive points in time, they can disrupt that developmental process through toxic effects on the general health of brain cells as well as on their ability to perform specialized functions. These toxic influences can weaken the foundational structure of the brain and result in permanent impairment, thereby leading to a wide range of lifelong, adverse impacts on learning, behavior, and health.<sup>3-12</sup>

The magnitude of the challenge of potentially toxic exposures requires sustained review and responsible management of a wide array of potentially threatening substances. Each year 2,000 to 3,000 new chemicals are brought to the U.S. Environmental Protection Agency for review prior to their manufacture. Currently there are more than 15,000 chemicals that are produced in quantities greater than 10,000 pounds per year and 2,800 are produced in quantities that exceed one million pounds annually <sup>13</sup>. Reports indicate that only 43 per cent of these "high volume" chemicals have been tested for human toxicity, and only 7 per cent have been evaluated for their potential effects on development.<sup>14, 15</sup>

Neurotoxicity (i.e., the quality or state of having a poisonous effect on neurons or neural circuits) may produce changes in the architecture and function of the brain as a result of exposure to a variety of biological or chemical agents.<sup>3, 4, 6, 8</sup> Certain prenatal infections, such as rubella, cytomegalovirus, and toxoplasmosis, are examples of biological agents whose neurotoxic properties have been studied extensively. This paper will focus on the wide variety of chemical substances that can harm the developing brain, which we have subdivided into three categories: (1) environmental chemicals, such as lead, mercury, and organophosphates; (2) recreational drugs, such as alcohol, nicotine, and cocaine; and (3) prescription medications, such as anticonvulsants to control seizure disorders and selected drugs for treatment of severe acne. Within each of these categories, exposure that occurs either before or after birth can result in highly toxic effects on the immature brain.

The striking finding from extensive research on neurotoxins is the magnitude of devastation and lifelong impacts they can have on human brain development.<sup>4, 16</sup> An important message for policy makers, however, is the extent to which many scientific findings are not well understood by the general public and do not match popularly held beliefs about safety and risk. This is particularly problematic in the face of widely available substances, such as alcohol, mercury, and many prescription drugs that are highly damaging to the immature brains of embryos (first trimester of pregnancy), fetuses (second and third trimester of pregnancy), and young children at doses that are tolerated with minimal to no adverse effects in adults. Thus, greater public understanding of this often confusing scientific knowledge provides an important opportunity for evidence-based policies that can strengthen our capacity to protect the developing brains of all young children.

#### WHAT SCIENCE TELLS US

Neurotoxins interfere with the natural function of genes, proteins, and other small molecules that build brain architecture. Disruptions of brain development caused by toxic substances, both before and after birth, can result in disorders that are evident immediately after exposure as well as impairments that emerge much later in life.<sup>5, 7, 12, 16</sup> Because fully effective treatments have yet to be developed for many of these disabling conditions, children who are exposed to neurotoxins before or soon after birth often face a lifetime of difficulties, for which all of society pays a continuing price.

- ♠ Environmental influences can be positive or negative in very powerful ways, because they have the capacity to literally change the architecture of the brain as it grows. Although exposure to toxins can result in serious injury, the brain is also resilient as biology protects it over other organ systems and helps it resist the potentially negative impacts of outside threats. Moreover, when given the chance, the brain often demonstrates the capacity to recover from damage. This balance between vulnerability and resilience determines how different environmental conditions affect brain development over time.
- The immature brain is far more vulnerable to toxic exposures than that of an adult. Mature brains have a barrier of cells that restrict the entry of chemicals from the bloodstream into brain tissue, but that protective barrier is absent in the fetus and only reaches maturity in the first year after birth. Thus, the time of greatest brain growth and most intensive construction of brain architecture is also the period that is most vulnerable to the relatively free passage of toxins into its cells.<sup>3, 8, 9, 17, 18</sup> Similar to the impact of disrupting the construction of the foundation of a new house, early exposure to toxic substances has broader and more lasting effects on brain development than exposure later in life.

#### **GLOSSARY**

CELL MEMBRANE: the outer layer of a cell that controls the passage of chemicals between the external environment and the cell's interior

ENZYME: a protein produced by cells that initiates or controls specific biochemical reactions

GLIA: specialized cells that provide a protective and supportive environment for neurons and their connections in the brain

GROWTH FACTOR: a protein or other substance (like vitamins) that promotes the growth of cells

MYELINATION: the process by which specialized brain cells form insulation around nerve fibers, which aids in the more rapid and error-free transmission of signals from one neuron to another

NEURAL CELL MIGRATION: an important part of the early embryonic development of the brain characterized by the movement of nerve cells from the place where they originate to specific locations where they form specific brain structures

NEURAL CIRCUIT: a network of connections among neurons that performs a specific function (e.g., visual circuit) NEURON: a specialized cell that serves as the fundamental information-processing unit of the nervous system

NEUROTOXICITY: the quality or state of having a poisonous effect on neurons or neural circuits

NEUROTRANSMITTER: naturally occurring chemical substances (such as serotonin or dopamine) produced and used by the nervous system to transmit information across a synapse from one neuron to another.

SYNAPSE: the junction between two neurons across which neurotransmitters pass in order to excite or inhibit the next neuron in line.





#### **ENVIRONMENTAL AGENTS**

- Chemicals classified as heavy metals, such as lead, mercury, and manganese, disrupt many of the normal biochemical processes that are necessary to build a sound and durable brain early in life. These substances come from many places, including contaminants in foods (e.g., mercury in fish), chemical waste that accumulates in water and plants, and synthetic materials (e.g., lead in paint, dust, or soil; manganese in unleaded gasoline). Generally speaking, heavy metals are present in complex chemical mixtures that break down over time, leading to the release of individual toxins that enter the bodies of children through eating, skin absorption, or inhalation, as well as through the placenta before birth.<sup>10, 18, 19</sup>
- At levels frequently measured in our environment, heavy metals interfere with the construction of the basic framework of the maturing brain as well as with its function. These toxic effects include disruption of neural cell migration from one part of the brain to another, as well as the formation of synapses (i.e., connections among cells), each of which is essential for building normal brain architecture. Heavy metals also interfere with neurotransmitters, which are the natural body chemicals that carry signals from one cell to another. These neurotransmitters are responsible for all brain functions, including learning, control of emotions, social interactions, and such fundamental processes as movement, vision, hearing, and touch. The most complex of these functions, which involve thinking and feeling, are the most susceptible to disruption by toxic exposures.<sup>20</sup>
- Lead can have adverse effects on several specific aspects of brain development. These include the formation and sculpting of neural circuits (i.e., the networks of connections among brain cells) as well as the process by which fatty tissue forms insulation around nerve fibers (known as myelination) like the insulation around the electrical wires in a house, which facilitates more rapid transmission of signals among brain cells. The disruptive effects of lead are due largely to interference with the normal function of several important neurotransmitters, including dopamine, glutamate, and acetylcholine. The primary functional deficits resulting from lead exposure, which have been demonstrated through repeated studies in both humans and animals, include a range of problems in learning, behavior, and the ability to focus and sustain attention.<sup>3-5, 21-27</sup>
- Mercury disrupts brain development by inhibiting important enzymes and preventing certain cells from dividing to produce more neurons and support cells (called glia). Research shows that mercury also increases the vulnerability of the brain to the adverse effects of other toxins at levels that are otherwise thought to be below dangerous thresholds, thereby producing a so-called "double hit." As for all neurotoxins, the degree to which developing brain architecture is disrupted by mercury ultimately depends upon the timing and level of exposure, each of which is influenced by the source of the toxin. Currently, emissions released by coal-fired power plants are the most important source of environmental mercury in the United States. This chemical is deposited into rivers, streams, and lakes where it is transformed by bacteria into a substance called methyl mercury, which is considered one of its most toxic forms. In recent years, the level of this dangerous chemical has been rising in the food chain, with the highest recordings found in contaminated fish (such as swordfish and tuna) as well as some shellfish, which are now the most significant sources of mercury exposure in the country and the





most harmful to the developing fetus and young child. Direct exposure to other forms of mercury, through contaminated soil or air near industrial sites, is a relatively smaller contributor. Exposure to elemental mercury, through broken thermometers or switches, is also much less common and much less toxic than to methyl mercury.<sup>3-5, 27-33</sup>

Exposure to organophosphates (also called "OPs"), which are common ingredients in insecticides used widely in agricultural regions and by professionals for control of insect infestation in homes and commercial facilities, can cause mild to severe disruption of brain development. The most widely investigated of the organophosphates, chlorpyrifos (CPF), kills neurons, causes defects in neural cell migration, and reduces connections among brain cells. Other organophosphates also affect the production of neurons, supporting cells, and neurotransmitters. Thus, organophosphates disrupt a wide range of processes that are essential for the formation and function of brain circuits. Although animal research demonstrates that organophosphates produce microscopic changes that are difficult to detect, studies of functional outcomes in both animals and children demonstrate that modest changes in brain architecture caused by exposure to CPF can lead to measurable problems in learning, attention, and emotional control. 4, 5, 10, 16, 18, 24, 34-36

#### RECREATIONAL DRUGS

- Both legal and illicit recreational neurotoxins, such as alcohol, nicotine and cocaine, interfere with chemicals that are necessary for the formation of normal brain architecture. Extensive human and animal research indicates that each agent causes different functional deficits that are influenced by the level, duration, and timing of the exposure. Recreational neurotoxins are most damaging during pregnancy because of the heightened susceptibility of the embryonic and fetal brain to developmental disruption. Research designed to pinpoint the precise biological impacts of parental substance abuse on fetal brain development, however, is quite challenging, given the high prevalence of multiple exposures (e.g., cocaine users often smoke cigarettes and consume alcohol) and the difficulties in conducting careful studies of individuals who are addicted to illegal substances. Even more important, it is often difficult to separate the biological impact of fetal exposure to toxic recreational drugs before birth from the physiological effects of environmental stresses facing children whose parents have a substance abuse problem, both of which can harm the developing brain.<sup>2</sup> Nevertheless, despite these research challenges, there is abundant scientific evidence that exposure to dangerous levels of recreational neurotoxins at particularly sensitive times in the developmental process can disrupt the architecture of the brain.<sup>16</sup>
- Of all the recreational neurotoxins studied to date, alcohol produces the most devastating disruptions of early brain development. These changes are most evident in the structure of cell membranes, which contain the proteins that are responsible for the ability of growth factors and neurotransmitters to perform their normal functions. The adverse impacts of alcohol are so powerful that they also can interfere with the development of organs that often are





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spared by other toxic exposures, including those of the cardiovascular, digestive, and musculo-skeletal systems. Thus, high levels of alcohol exposure during pregnancy have been shown to produce a combination of problems that have been characterized as the "fetal alcohol syndrome," which is a serious medical condition involving multiple organ systems. Once again, the timing of the toxic exposure is most important. Alcohol exposure in the embryo and fetus can cause different kinds of disruptions of brain architecture by killing neurons or stalling their migration during critical developmental periods. The potential long term functional outcomes of such disruptions in both human and animal studies include cognitive deficits such as mental retardation, reduced emotional control, problems with attention, and hyperactivity.<sup>3, 16, 27, 37, 38</sup>

- Nicotine exposure from cigarette smoking during pregnancy also has a well documented adverse impact on the structure and function of the fetal brain. Nicotine binds to a membrane protein that is responsible for the function of acetylcholine, a naturally occurring neurotransmitter in the adult brain that also is present during fetal development. When pregnant women smoke, oxygen delivery to the fetus is reduced and high levels of nicotine exposure result in decreased overall growth. Both animal and human studies also have documented cognitive impairments associated with fetal nicotine exposure, although these effects are significantly milder than those resulting from alcohol or other toxic chemicals.⁴ 10, 27, 39
- ©Cocaine, methamphetamine ("speed"), and methylphenidate (Ritalin) are psycho-stimulant substances that have been shown to cause functional impairments in animals and humans who experience prenatal exposure. However, unlike the adverse effects of alcohol and other neurotoxins that are noticeable in early childhood, the damage from prenatal psychostimulant exposure may not be apparent until later in life.<sup>27</sup> Moreover, the specific impact of exposure to psychostimulants in humans has been relatively difficult to investigate, because pregnant women who abuse cocaine or other psychostimulants typically use alcohol and nicotine as well. Psychostimulants act by interfering with the regulation of a class of neurotransmitters (the monoamines) whose activation and inactivation are important for normal function in fetal brain development. Animal studies demonstrate that psychostimulants such as cocaine cause changes in the maturation of brain cells located in specific circuits that affect the ability to focus attention and regulate emotion. Most prospective studies of prenatal cocaine exposure in humans report relatively modest developmental changes in infants and toddlers but measurable problems with attention, hyperactivity, and mood control as the children are followed into their early teen years.<sup>16, 27, 40</sup>

#### **MEDICATIONS**

● A variety of prescription drugs that are safe for adults can cause serious damage to an immature nervous system. For example, both human and animal studies indicate that prenatal exposure to valproate, which is used to treat seizure disorders, can cause neural tube defects (i.e., defects in the spinal cord, such as spina bifida) and substantial disruption of early brain growth and architecture. Moreover, studies of postnatal exposure in animals demonstrate both





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destruction of brain cells and alteration in the formation of neural circuits involved in cognitive and behavioral functions. As expected from this type of developmental disruption, valproate exposure during pregnancy can cause mental retardation, other cognitive deficits, and impaired emotional control.<sup>3, 41</sup>

The extent to which certain nutritional supplements can disrupt the development and function of the immature brain is highly influenced by both the timing and level of exposure. Vitamin A, for example, is a common example of a class of chemicals called retinoids, which are essential to a variety of chemical reactions that are important for normal brain development, including the activity of genes that are necessary for producing brain cells, promoting their specialization, and protecting their survival. Excessive exposure during embryonic or fetal development, however, results in impairments that can be major (e.g., spina bifida) or relatively minor (i.e., mild functional difficulties). Excessive levels of retinoids resulting from maternal use of a compound to treat severe acne during pregnancy can have particularly devastating effects. This provides a striking example of a chemical that is highly neurotoxic for the immature brain of a fetus at doses that are tolerated without serious consequences by adults.<sup>42, 43</sup>



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#### POPULAR MISREPRESENTATIONS OF SCIENCE

Popular beliefs about which chemical substances are more or less toxic to the developing embryo, fetus, infant, and child are most commonly related to their relative abundance and legal status in society. In this context, it is essential that we distinguish scientific facts from widespread misperceptions.

- It is generally assumed that illegal recreational drugs have the most damaging impacts on brain development and function. In fact, extensive research indicates that alcohol is one of the most dangerous neurotoxins that can affect the brain during the period between conception and birth.<sup>3, 37, 38</sup>
- It is generally assumed that the adverse impact of toxic substances on the developing architecture of the brain is an all-or-none phenomenon. In fact, neurotoxins can produce a range of outcomes, from mild to severe impairment, which often lead to confusing conclusions about the linkage between exposure to a specific substance and its consequences.<sup>5-7, 9, 12, 27, 28, 40</sup>
- It is generally assumed that the absence of cognitive or behavioral problems in childhood indicates that an early exposure to a neurotoxin had no adverse effect on brain development. In fact, studies in both animals and humans have demonstrated that some substances cause damage to the brain that is manifested in the delayed onset of learning problems, attention deficits, and changes in emotional regulation, which can have long term consequences into the teenage and adult years.<sup>3, 5, 7-9, 12, 27</sup>
- oIt is generally assumed that the determination of a dangerous level of exposure to a potentially neurotoxic substance is a straightforward scientific question. In fact, this can present a complicated challenge because the developing brain of a young child is typically more susceptible to damage than the mature brain of an adult, and the immature nervous system of an embryo or fetus is even more vulnerable to toxic exposures than is that of an infant. Therefore, there is no credible way to determine a safe level of exposure to a potentially toxic substance without explicit research that differentiates its impact on adults from the greater likelihood of its adverse influences on the developing brain during pregnancy and early childhood.⁴, 6, 7, 9, 11, 12, 16, 24, 26, 36, 44
- There is a popular misperception among some groups that vaccines containing thimerosal (which has been added as a preservative) are linked to the development of autism in susceptible children. In fact, extensive and repeated studies by highly reputable scientific groups have failed to confirm this claim.<sup>45</sup>





#### THE SCIENCE-POLICY GAP

There is no question that exposure to certain chemical substances during the period from conception through the early years of life causes significant and irreversible damage to the developing architecture of the brain. Nevertheless, the importance of determining which substances are safe and specifying thresholds of exposure for those that are dangerous is not yet incorporated into public policy. These tasks are complicated by the fact that policy initiatives in this area are driven largely by popular beliefs, which are influenced primarily by advocacy groups and media reports that often are not updated as new science becomes available. Moreover, these folk beliefs prove especially stubborn to dislodge as they are not subject to rigorous scientific review. Although much remains to be learned about the full breadth of risk during pregnancy and early childhood, there is much that can be done based on what we know now about how to reduce the number of children whose brains are harmed by neurotoxins.

Over the past few decades, effective public policy has been developed to reduce exposure to some of the most widely recognized neurotoxins. The decreased prevalence of lead poisoning is a prominent example. Efforts to educate the public about the harmful effects on children of second hand cigarette smoke provide another example. Between 1994 and 1999, the percentage of homes with a child under age 7 in which someone smoked regularly dropped from 29% to 19%. Consequently, median blood levels of cotinine (a breakdown product of nicotine) were 56% lower in the five year period from 1999 to 2000 compared to levels reported between 1988 and 1991. In 1999-2000, the U.S. Environmental Protection Agency imposed new restrictions on the use of organophosphate pesticides, largely because of concerns about the potential exposure of young children. Subsequently, the percentage of food samples with detectable residues of these pesticides declined from 29% in 1996 to 19% in 2001. In 2001.

Although progress has been made in reducing selected toxins, policies that could restrict the exposure of embryos, fetuses, and infants to other chemicals whose neurotoxicity is well documented, such as mercury and other industrial organic compounds, have been less well formulated. Beyond the moral responsibility to reduce known threats to the health of young children, there are persuasive economic arguments for greater attention to the value of prevention, both as an investment in sound development and as strategy for reducing the continuously escalating treatment costs of disease and disability. The gap between what we know about the potentially devastating effects of neurotoxins and what we do through public policies and programs to protect the developing brain from harm in the early years of life is illustrated by the following examples.

⑤ Because of the highly complex nature of the processes that build brain architecture in the earliest years, the immature brain of an embryo, fetus, or infant is often susceptible to significant damage from exposure to chemicals at levels that appear to be harmless for adults. Despite this well established scientific fact, policy makers generally establish safe levels of exposure to prescription drugs and known neurotoxins through a process that is guided by research findings from studies of mature animals and adult humans.<sup>4,11,16</sup>





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- The absence of overt cognitive and behavioral deficits in infants and toddlers who have been exposed to neurotoxic substances often has a strong influence on establishing priorities for regulatory controls. However, long-term impacts of some early toxic exposures, which can include a so-called "silent period" of normal functioning prior to the appearance of functional deficits, are not well understood. This typically results in public policies that fail to protect developing brains during pregnancy and early infancy.<sup>3, 5, 7-9, 12, 27</sup>
- OAn illustrative example of the science-policy gap can be found in a recent study by the U.S. Environmental Protection Agency which estimated that 8 per cent of women of childbearing age in the United States have dangerously high blood levels of mercury. After concluding that "there is no safe level of methyl mercury in the blood" (p. 59), the report went on to state that 50 per cent of women of childbearing age have blood levels of mercury that reach or exceed 1 part per billion. With these data as a backdrop, research shows that mercury levels in the food chain are increasing, with the greatest concern focused on popular fish such as swordfish and tuna. What makes the science-policy gap particularly striking is evidence that the source of this increasing toxic chemical burden is well known and preventable. The largest production of environmental mercury comes from the emissions of coal-burning power plants and incinerators, despite the fact that technology is available to reduce its atmospheric release. Other sources of contamination are related to inadequate disposal of mercury-containing products, which could be ameliorated through greater public education and the provision of convenient and appropriate mechanisms for recycling and waste management.



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#### IMPLICATIONS FOR POLICY AND PROGRAMS

Beyond the importance of individual responsibility for the care and protection of children, public policies can have a significant impact on promoting health and preventing disease or disability in the entire population. When fluoride is added to public water supplies, children have fewer dental caries. When foods eaten by infants and toddlers are fortified with iron, the prevalence of anemia decreases, and the risk of associated developmental problems goes down. In contrast, when mercury enters the food chain or when young children play in gardens that have been sprayed with neurotoxic insecticides, individual behavior cannot guarantee safety and the resulting disruptions of early brain development that lead to lifelong disabilities could have been prevented by informed public policies.<sup>4, 5, 47, 49</sup>

To this end, the basic science of how early brain development can be disrupted by toxic substances is now sufficiently detailed to inform more effective policies to protect the well-being of human embryos, fetuses, and young children.

- The costs of ignoring the danger of neurotoxins are high. The moral costs of preventable disability and public expenses for special education, medical care, and lost economic productivity incurred for individuals with disabilities that were caused by early chemical damage to the developing brain are considerable. The costs of cognitive impairments due to lead poisoning alone, for example, have been estimated to approach \$43 billion per year, and the costs of mental retardation, autism, and cerebral palsy due to environmental pollutants have been estimated at \$9 billion annually.¹³ The magnitude of this financial burden indicates that the prevention of brain damage by neurotoxic exposures should be assigned a higher priority for policies focused on public health, education, human capital development, and environmental protection.⁵, ¹³, ⁴8, ⁵⁰
- The establishment of safe levels of exposure to toxic substances should be based on scientific data that recognizes the critical link between vulnerability and age. In view of the well-established scientific fact that embryonic, fetal, and early childhood brain development is considerably more susceptible to damage from toxic substances than the mature brain of an adult, the establishment of thresholds for toxic exposures should focus primarily on the best data available for the youngest children. 4, 6, 7, 11, 12, 16, 26, 36
- Nowledge about the potential adverse impacts of prescription drugs and nutritional supplements when used by pregnant women and nursing mothers should be updated continuously and communicated to health care personnel and the general public. The fact that toxic exposures can be most damaging in the earliest weeks of pregnancy, before many women are aware that they are pregnant, underscores the importance of broader public education on this issue. Current efforts to strengthen the ability of nurses to play this important role can be seen in the Environmental Health Nursing Initiative launched by the Agency for Toxic Substances and Disease Registry in the U.S. Department of Health and Human Services (http://www.atsdr.cdc.gov).





- OScientific information should be disseminated more extensively through warning labels and proactive controls on toxic exposures. Information on the toxic effects of organophosphates could be disseminated in a more effective manner by a requirement for clearer warning labels on the packaging of commonly used insecticides. This would enable pregnant women and families with young children to make more informed choices about the products they use. In an effort to move beyond sole reliance on individual behavior, Michigan enacted legislation in 2004 that prohibits the use of any pesticides at a school or child care center unless it has adopted an integrated pest management program that focuses on non-pesticide alternatives to chemical controls. Both the Michigan law and recent legislation in Rhode Island require schools and child care centers to notify parents in advance before pesticides are used on school grounds. More proactive education programs also should be provided for employees of childbearing age who are exposed to substances in the workplace that are not harmful to adults but can be highly toxic to the immature brain. In such cases, chemicals can be transmitted through the placenta of a pregnant woman or brought home on the clothing of a mother or father of a young child.
- Marketing campaigns provide opportunities for enhanced public education. The significant neurotoxicity of prenatal alcohol exposure calls for a focused re-examination of the marketing of alcohol to young adults and other vulnerable populations. This should include efforts targeted at low income neighborhoods and populations with less education, college campuses where alcohol abuse is common, and work sites that have high concentrations of employees of childbearing age.
- The chemical and interpersonal impacts of adult addictive behaviors on child well-being require greater public attention. Routine prenatal care should be augmented by the incorporation of state-of-the-art practices for identifying and treating women who are addicted users of both legal (e.g., alcohol and tobacco) and illegal (e.g., cocaine) substances. This could be reinforced through community-based counseling, targeted education programs in the workplace, and comprehensive therapeutic interventions as needed. Central to such efforts is the need to focus on the fact that parental substance abuse threatens the development of healthy brain architecture through the effects of two kinds of chemicals: (1) abused substances that are taken by the mother and cross the placenta during pregnancy; and (2) elevated stress hormones that are produced by young children themselves who experience highly stressful interactions in the absence of stable and supportive caregiving relationships.
- Increased investments in environmental surveillance are needed to strengthen our capacity to prevent damage to young children's brains. There is a compelling need for greater public efforts to track developmental disorders that are linked to environmental exposures in order to identify disease clusters and determine the causes of disrupted brain architecture.<sup>51</sup> Examples of recent state-level legislation include the California Health Tracking Act of 2003 and the Illinois Children's Environmental Health Officer Act of 2005.

The protection of young children requires a balanced approach to both individual and public responsibility. Extensive research shows that preventive public health measures are most effective





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when they do not depend primarily on individual behavior.<sup>4, 5, 18, 19, 46, 47</sup> Well studied examples include safety caps on medications that decrease child ingestions; lowered hot water temperatures that prevent scalding burns; the removal of lead from gasoline, food cans, and residential paint products which leads to lowered blood lead levels in children; and the fortification of bread, flour, and grain products with folic acid to reduce the incidence of neural tube defects such as spina bifida.

In the final analysis, the prevention of brain damage to embryos, fetuses, and young children as a result of toxic exposures will depend largely on the extent to which effective controls are implemented to lower the levels of known neurotoxins in the environment. Maryland demonstrated such a commitment in 2000 with the creation of a children's environmental health protection advisory panel, which reviews existing regulations, statutes, and proposed regulations to assess whether they provide sufficient protection for children and makes specific recommendations accordingly. The challenge for policy makers and civic leaders is to build working relationships with leading research and public health agencies, educate the general public about the science of neurotoxicity, confront popular misunderstandings and active distortions of that science, and use currently available knowledge to design and implement policies to reduce preventable injuries to the brains of young children, both before and after birth.



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	ON THE DEVELOPING

#### **REFERENCES**

- 1. National Scientific Council on the Developing Child, *Young children develop in an environment of relationships*, 2004, http://www.developingchild.net/reports.shtml
- 2. National Scientific Council on the Developing Child, Excessive stress disrupts the architecture of the developing brain. 2005. http://www.developingchild.net/reports.shtml
- 3. Costa, L.G., et al., *Developmental neuropathology of environmental agents*. Annual Review of Pharmacology and Toxicology, 2004, 44: p. 87-110.
- 4. Etzel, R.A. and S.J. Balk, eds. *Pediatric environmental health.* 2nd ed. 2003, American Academy of Pediatrics, Committee on Environmental Health: Elk Grove Village, IL.
- 5. Lanphear, B.P., R.O. Wright, and K.N. Dietrich, *Environmental neurotoxins*. Pediatrics in Review, 2005. 26(6): p. 191-197.
- 6. Levitt, P., Structural and functional maturation of the developing primate brain. Pediatrics, 2003. 143: p. \$35-\$45.
- 7. National Research Council, *Scientific frontiers in developmental toxicology and risk assessment.* 2000, National Academy Press: Washington, D.C.
- 8. Rice, D. and S. Barone, *Critical periods of vulnerability for the developing nervous system: Evidence from humans and animal models.* Environmental Health Perspectives, 2000. 108(Supplement 3): p. 511-533.
- 9. Weiss, B., *Vulnerability of children and the developing brain to neurotoxic hazards.* Environmental Health Perspectives, 2000. 108(Supplement 3): p. 375-381.
- 10. Needham, L.L., et al., Concentrations of environmental chemicals associated with neurodevelopmental effects in the U.S. population. NeuroToxicology, 2005. 26(4): p. 531-545.
- 11. Pohl, H.R., et al., *Risk assessment of chemicals and pharmaceuticals in the pediatric population: A workshop report.* Regulatory Toxicology and Pharmacology, 2005. 42: p. 83-95.
- 12. Selevan, S.G., C.A. Kimmel, and P. Mendola, *Identifying critical windows of exposure for children's health*. Environmental Health Perspectives, 2000. 108(Supplement 3): p. 451-455.
- 13. Landrigan, P.J., et al., Environmental pollutants and disease in American children: Estimates of morbidity, mortality, and costs for lead poisoning, asthma, cancer, and developmental disabilities. Environmental Health Perspectives, 2002. 110(7): p. 721-728.
- 14. Goldman, L.R. and S. Koduru, Chemicals in the environment and developmental toxicity to children: A public health and policy perspective. Environmental Health Perspectives, 2000. 108(Supplement 3): p. 443-448.
- 15. National Academy of Sciences, *Toxicity testing: Needs and priorities.* 1984, National Academy Press: Washington, D.C.
- 16. Stanwood, G.D. and P. Levitt, *Drug exposure early in life: Functional repercussions of changing neurophar-macology during sensitive periods of brain development.* Current Opinion in Pharmacology, 2004. 4: p. 65-71.
- 17. Houlihan, J., et al., *Body burden: The pollution in newborns.* 2005, Environmental Working Group: Washington, D.C.
- 18.U.S. Environmental Protection Agency, America's children and the environment: Measures of contaminants, body burdens, and illnesses. 2003, U.S. Environmental Protection Agency: Washington, D.C.
- 19. Centers for Disease Control and Prevention, *National report on human exposure to environmental chemicals.* 2001, Centers for Disease Control and Prevention: Atlanta, GA.
- 20. Klaassen, C.D., Heavy metals and heavy-metal antagonists, in Goodman & Gilman's The pharmacological basis of therapeutics, J.G. Hardman and L.E. Limbird, Editors. 1996, McGraw-Hill: New York, NY.





#### EARLY EXPOSURE TO TOXIC SUBSTANCES DAMAGES BRAIN ARCHITECTURE

- 21. Agency for Toxic Substances and Disease Registry, *Toxicological profile for lead (Draft for public comment).* 2005, U.S. Department of Health and Human Services, Public Health Service: Atlanta, GA.
- 22. Centers for Disease Control and Prevention, Screening young children for lead poisoning: Guidance for state and local public health officials. 1997, Centers for Disease Control and Prevention: Atlanta, GA.
- 23. Cory-Slechta, D.A., Interactions of lead exposure and stress: Implications for cognitive dysfunction, in Neurotoxicity and developmental disabilities, P.W. Davidson, G.J. Myers, and B. Weiss, Editors. 2006, Elsevier Academic Press: San Diego, CA.
- 24. Cory-Slechta, D.A., Studying toxicants as single chemicals: Does this strategy adequately identify neurotoxic risk? NeuroToxicology, 2005. 26(4): p. 491-510.
- 25. Canfield, R.L., M.H. Gendle, and D.A. Cory-Slechta, *Impaired neuropsychological functioning in lead-exposed children*. Developmental Neuropsychology, 2004. 26(1): p. 513-540.
- 26. Chiodo, L.M., S.W. Jacobson, and J.L. Jacobson, *Neurodevelopmental effects of postnatal lead exposure at very low levels.* Neurotoxicology and Teratology, 2004. 26(3): p. 359-371.
- 27. Trask, C.L. and B.E. Kosofsky, *Developmental considerations of neurotoxic exposures*. Neurologic Clinics, 2000. 18(3): p. 541-562.
- 28. Agency for Toxic Substances and Disease Registry, *Toxicological profile for mercury.* 1999, U.S. Department of Health and Human Services, Public Health Service: Atlanta, GA.
- 29. Rice, G. and J.K. Hammitt, Economic valuation of human health benefits of controlling mercury emissions from U.S. coal-fired power plants. 2005, Northeast States for Coordinated Air Use Management: Boston, MA.
- 30. U.S. Environmental Protection Agency, *Mercury Study Report to Congress.* 1997, U.S. Environmental Protection Agency: Washington, D.C.
- 31. Mahaffey, K.R., R.P. Clickner, and C.C. Bodurow, *Blood Organic Mercury and Dietary Mercury Intake:* National Health and Nutrition Examination Survey, 1999 and 2000. Environmental Health Perspectives, 2004. 112(5).
- 32. Myers, G.J., P.W. Davidson, and C.F. Shamlaye, Developmental disabilities following prenatal exposure to methyl mercury from maternal fish consumption: A review of the evidence, in Neurotoxicity and developmental disabilities, P.W. Davidson, G.J. Myers, and B. Weiss, Editors. 2006, Elsevier Academic Press: San Diego, CA.
- 33. Davidson, P.W., G.J. Myers, and B. Weiss, *Mercury exposure and child development outcomes*. Pediatrics, 2004. 113(4 Supplement): p. 1023-9.
- 34. Needleman, H.L., *The neurotoxic properties of pesticides,* in *Neurotoxicity and developmental disabilities*, P.W. Davidson, G.J. Myers, and B. Weiss, Editors. 2006, Elsevier Academic Press: San Diego, CA.
- 35. Vidair, C.A., Age dependence of organophosphate and carbamate neurotoxicity in the postnatal rat: Extrapolation to the human. Toxicology and Applied Pharmacology, 2004. 196(2): p. 287-302.
- 36. Weiss, B., S. Amler, and R.W. Amler, Pesticides. Pediatrics, 2004. 113(4 Supplement): p. 1030-1036.
- 37. Burbacher, T.M. and K.S. Grant, *Neurodevelopmental effects of alcohol*, in *Neurotoxicity and developmental disabilities*, P.W. Davidson, G.J. Myers, and B. Weiss, Editors. 2006, Elsevier Academic Press: San Diego, CA.
- 38. Welch-Carre, E., *The neurodevelopmental consequences of prenatal alcohol exposure.* Advances in Neonatal Care, 2005. 5(4): p. 217-229.
- 39. Weitzman, M., M. Kavanaugh, and M.A. Florin, Parental smoking and children's behavioral and cogni-





#### EARLY EXPOSURE TO TOXIC SUBSTANCES DAMAGES BRAIN ARCHITECTURE

tive functioning, in Neurotoxicity and developmental disabilities, P.W. Davidson, G.J. Myers, and B. Weiss, Editors. 2006, Elsevier Academic Press: San Diego, CA.

- 40.Mendola, P., et al., *Environmental factors associated with a spectrum of neurodevelopmental disorders*. Mental Retardation and Developmental Disabilities Research Reviews, 2002. 8: p. 188-197.
- 41. McNamara, J.O., Drugs effective in the therapy of the epilepsies, in Goodman & Gilman's The pharmacological basis of therapeutics, J.G. Hardman and L.E. Limbird, Editors. 1996, McGraw-Hill: New York, NY.
- 42. Guzzo, C.A., G.S. Lazarus, and V.P. Werth, *Dermatological pharmacology*, in *Goodman & Gilman's The pharmacological basis of therapeutics*, J.G. Hardman and L.E. Limbird, Editors. 1996, McGraw-Hill: New York, NY.
- 43. Marcus, R. and A.M. Coulston, Fat-soluble vitamins: Vitamins A, K, and E, in Goodman & Gilman's The pharmacological basis of therapeutics, J.G. Hardman and L.E. Limbird, Editors. 1996, McGraw-Hill: New York, NY.
- 44. Rice, D.C., Assessing the effects of environmental toxicant exposure in developmental epidemiological studies: Issues for risk assessment. NeuroToxicology, 2005. 26: p. 483-489.
- 45. Institute of Medicine and Immunization Safety Review Committee, *Immunization safety review:* Vaccines and autism. 2004, National Academy of Sciences: Washington, D.C.
- 46.U.S. Environmental Protection Agency, *Benefits and costs of the Clean Air Act.* 1997, U.S. Environmental Protection Agency: Washington, D.C.
- 47. Goldman, L.R., et al., *Environmental pediatrics and its impact on government health policy.* Pediatrics, 2004. 113(4): p. 1146-1157.
- 48. Muir, T. and M. Zegarac, Societal costs of exposure to toxic substances: Economic and health costs of four case studies that are candidates for environmental causation. Environmental Health Perspectives, 2001. 109(Supplement 6): p. 885-903.
- 49. Gilbert, S.G., Ethical, legal, and social issues: Our children's future. NeuroToxicology, 2004. 26: p. 521-530.
- 50. National Conference of State Legislatures, *Children's health and environmental fact sheet:* Developmental disabilities. 2004, National Conference of State Legislatures: Denver, CO.
- 51. Needham, L.L., D.B. Barr, and A.M. Calafat, *Characterizing children's exposures: Beyond NHANES*. NeuroToxicology, 2005. 26: p. 547-553.

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### NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD

# The Timing and Quality of **Early Experiences Combine** to Shape Brain Architecture **WORKING PAPER 5**

Center on the Developing Child ## HARVARD UNIVERSITY

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The National Scientific Council on the Developing Child, housed at the Center on the Developing Child at Harvard University, is a multi-disciplinary collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children.

For more information, go to www.developingchild.net.

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## The Issue

THE FOUNDATIONS OF BRAIN ARCHITECTURE ARE ESTABLISHED EARLY IN LIFE THROUGH A CONTINUous series of dynamic interactions in which environmental conditions and personal experiences have a significant impact on how genetic predispositions are expressed.<sup>1-7</sup> Because specific experiences affect specific brain circuits during specific developmental stages—referred to as sensitive periods<sup>8,9</sup>—it is vitally important to take advantage of these early opportunities in the developmental building process. That is to say, the quality of a child's early environment and the availability of appropriate experiences at the right stages of development are crucial in determining the strength or weakness of the brain's architecture, which, in turn, determines how well he or she will be able to think and to regulate emotions.

Just as in the construction of a house, certain parts of the formative structure of the brain need to happen in a sequence and need to be adequate to support the long-term developmental blueprint. And just as a lack of the right materials can result in blueprints that change, the lack of appropriate experiences can lead to alterations in genetic plans. Moreover, although the brain retains the capacity to adapt and change throughout life, this capacity decreases with age. 10-12 Thus, building more advanced cognitive, social, and emotional skills on a weak initial foundation of brain architecture is far more difficult and less effective than getting things right from the beginning.13

The exceptionally strong influence of early experience on brain architecture makes the early years of life a period of both great opportunity and great vulnerability for brain development. An early, growth-promoting environment, with adequate nutrients, free of toxins, and filled with social interactions with an attentive caregiver, prepares the architecture of the developing brain to function optimally in a healthy environment. 14,15 Conversely, an adverse early environment, one that is inadequately supplied with nutrients, contains toxins, or is deprived of appropriate sensory, social, or emotional stimulation, results in faulty brain circuitry. 7,16-19 Once established, a weak foundation can have detrimental effects on further brain development, even if a healthy environment is restored at a later age.

The considerable susceptibility of the young, developing brain to the synergistic effects of environment and experience has enormous implications for policymakers, parents, and society. An abundance of scientific evidence clearly demonstrates that critical aspects of brain architecture begin to be shaped by experience before and soon after birth, and many fundamental aspects of that architecture are established well before a child enters school. 1,7-9,20-22

Critical aspects of brain architecture begin to be shaped by experience before and soon after birth, and many fundamental aspects of that architecture are established well before a child enters school.

Nevertheless, despite increasing public investment in K-12 education, there remains a persistent tolerance in our society for poor quality care and education in the early childhood period. In this context, scientific evidence indicates that for children to reach their full potential, communities need to support the capacity of all families to provide a variety of stimulating and appropriate experiences in the earliest years, when a child's brain is optimally programmed to benefit from specific types of experiences, and then build on that sturdy brain foundation through continuous exposures to high quality, age-appropriate experiences throughout the later school-age years.<sup>23</sup>

### What Science Tells Us

The architecture of the brain depends on the mutual influences of genetics, environment, and experience. *Genetics* supplies a basic plan for brain development, just as an architect supplies a blueprint for building a house. The genetic plan instructs the basic properties of the nerve cells and lays down the basic rules for interconnecting nerve cells within and across circuits. In this manner, genes provide the initial construction plan for the brain's architecture.

The *environment* in which the brain begins to develop can have a profound influence on its initial architecture. Just as the selection of the best building materials enables the realization of the full potential of an architect's blueprint, a healthy environment beginning in the prenatal period allows the full potential of the genetic plan for the brain to be expressed. This includes an abundant supply of nutrients, an absence of toxins, and the healthy personal and social

# Experiences during sensitive periods of development play an exceptionally important role in shaping the capacities of the brain.

habits of the expectant mother. <sup>14,15</sup> Conversely, an environment lacking in critical nutrients, or containing toxins that result from unhealthy behaviors such as excessive maternal alcohol intake during pregnancy or lead ingestion in early childhood, can cause neurons to acquire abnormal properties and aberrant connections with other brain cells. <sup>17,18,22</sup> In addition, an adverse prenatal environment can actually alter the genetic plan for the brain. <sup>19,32</sup> These effects of threatening environmental conditions can cause neural circuits to change in ways that prevent them from functioning well, or at all, even in a subsequent healthy environment.

Experience refers to the interaction of a child with his or her environment. In humans, such experience begins before birth, as the fetus senses and responds to the environment of the womb.<sup>18</sup> This early experience influences the basic architecture of low-level circuits that mature at this early stage. After birth, experience plays an increasingly important role in shaping

the architecture of developing neural circuits so that they function optimally for each individual.<sup>8,15,20,33</sup> Just as a master carpenter modifies the blueprint for a house to adapt to the needs of its setting and the people who will live in it, experience adjusts the genetic plan for the brain and shapes the architecture of its neural circuits according to the needs and distinctive environment of the individual.<sup>2,6,15</sup> Consequently, healthy and stimulating experience results in brain architecture that operates at its full genetic potential, and persistent adversity leads to weak brain architecture with impaired capabilities.

# Early environments and experiences have an exceptionally strong influence on brain architecture.

For most neural circuits, the effects that the environment and individual experience can exert on their architecture are particularly potent just as the circuit is maturing. As a circuit begins to function, its chemical environment and the electrical information that it processes can have an enormous impact on that circuit, causing adjustments in its genetic plan and changing its architecture in fundamental ways. After most circuits have matured, their genetic plans and architecture can still be modified by experience, but the extent of these later modifications tends to be far more limited.

The period of exceptional sensitivity to the effects of environment and experience is called a *sensitive period* for that circuit. Because it is far more difficult to alter neural circuits substantially after their sensitive periods have ended, experiences during these sensitive periods play an exceptionally important role in shaping the capacities of the brain. Some examples of behavioral capacities that have been shown to be affected by sensitive periods of underlying circuitry include vision, 4,34 hearing, 10 language, 35 and responses to social cues. 2,13,15

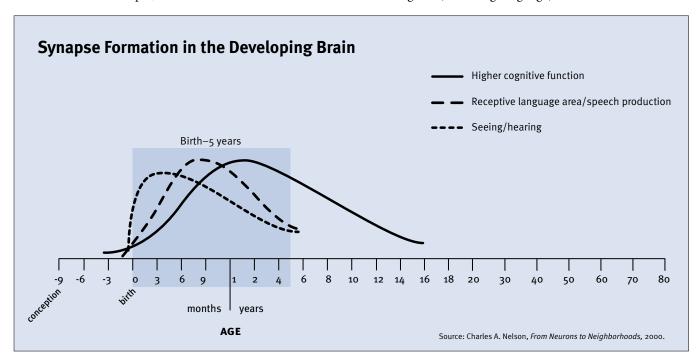
The increased flexibility of the circuitry in a young, developing brain is explained primarily by three factors. First, during its initial stages of formation, the brain develops far more extensive connections than it needs in order to function optimally, and connections that are not useful are pruned away over time.<sup>4</sup> Second, the molecular environment and cellular mechanisms that enable the formation of new connections

and the elimination of incorrect connections are highly active in a circuit while it is maturing.8 Finally, neural circuits are far more flexible before a particular pattern of connections has been shaped and fully activated. Consequently, once a particular circuitry pattern becomes established, it is difficult for the effects of new and different experiences to alter that architecture. 36,37 This means that early experience has a unique advantage in shaping the architecture of developing brain circuits before they are fully mature and stabilized.

Different mental capacities mature at different stages in a child's development. Aspects of mental function are carried out by different hierarchies of neural circuits in the brain. The hierarchies of circuits that analyze visual information are different from those that process auditory information, learn language, remember recent events, plan future actions, or determine emotional responses. Because these various hierarchies mature at different times in a child's life,24 the same environmental conditions will produce different cognitive and emotional experiences for a child, depending on his or her age. 20,25,26

Even within a single hierarchy—such as visual, auditory, or language development—different neural circuits mature at different times. Circuits that process lower-level information mature earlier than those that process higher-level information.<sup>27</sup> For example, in the neural hierarchy that analyzes visual information, low-level circuits that analyze color, shape, or motion are fully mature long before the higher-level circuits that interpret complex stimuli, such as facial expressions, or identify meaningful inputs such as frequently used objects. 26,28-30 For the developing brain, this means that the ability to perceive simple aspects of the world and to make simple emotional and social judgments develops long before the ability to make sophisticated, cognitive analyses. 20,31 Stated simply, children's ability to interpret what they see changes over time as their brain circuitry is built. Thus, it is important that experiences provided in the earliest years are appropriate for the young child's stage of development. Reading a picture book with a toddler who is learning to speak, for example, provides an important opportunity to point to and talk about the pictures, not to focus on the written words. The ability to decode written language comes later, when the appropriate, higherlevel brain circuitry will be built.

Sensitive periods occur at different ages for different parts of the brain. Different neural circuits pass through sensitive periods at different ages. The sensitive periods for neural circuits that perform low-level analyses of sensory stimuli tend to end before or soon after birth. 38,39 In contrast, the sensitive periods for high-level circuits that process sophisticated aspects of the world, such as communication signals (including language)



or the interpretation of facial expressions, end much later in development. <sup>26,35,40</sup>

Because low-level circuits mature early and high-level circuits mature later, different kinds of experiences are critical at different ages for optimal brain development,<sup>41</sup> a concept called *age-appropriate experience*. Soon after birth, basic sensory, social, and emotional experiences are essential for optimizing the architecture of low-level circuits. At later ages, more sophisticated kinds of experiences are critical for shaping higher-level circuits. When adults or communities expect young children to master skills for which the necessary brain circuits have not yet been formed, they waste time and resources, and may even impair healthy brain development by inducing excessive stress in the child.

Stimulating early experiences lay the foundation for later learning. High-level neural circuits that carry out sophisticated mental functions depend on the quality of the information that is provided to them by lower-level circuits. Lowlevel circuits whose architecture was shaped by healthy experiences early in life provide highlevel circuits with precise, high-quality information. High-quality information, combined with sophisticated experiences later in life, allows the architecture of circuits involved in higher functions to take full advantage of their genetic potential. Thus, early learning lays the foundation for later learning and is essential (though not sufficient) for the development of optimized brain architecture. Stated simply, stimulating early experience must be followed by more sophisticated and diverse experiences later in life, when high-level circuits are maturing, in order for full potential to be achieved. 13,20,42,43

Impoverished early experience can have severe and long-lasting detrimental effects on later brain capabilities. Sensitive periods act as double-edged swords. On the one hand, a sensitive period enables a neural circuit to optimize its architecture for the needs and environment of the individual.<sup>33,44</sup> On the other hand, this period of extreme receptivity also makes the circuit vulnerable to the damaging effects of adversity.<sup>16,45</sup> Just as a faulty foundation has far-reaching detrimental effects on the strength and quality of a house, adverse early experience can have far-reaching detrimental effects on the development of brain architecture.

Stressful experiences during sensitive periods alter the function and architecture of specific neural circuits, as these circuits adapt their functional properties to the adversity that has been experienced. 8,10,38 As shown by experiments in which animals have been subjected to significant stress, when the adverse conditions last through the end of a circuit's sensitive period, the changes in the circuit's architecture become stable and tend to persist in the adult brain. 46,47 Subsequently, although the brain's residual capacity for plasticity can mitigate the adverse effects of the altered circuit architecture, 10 the affected neural circuits do not process information as well as they could have if the animal had been exposed to an appropriate experience during the sensitive period. The degraded information that is transmitted by the altered neural circuit can prevent high-level circuits from receiving the information they need to shape their architectures optimally, even after a rich environment has been restored later in life.

Brain plasticity continues throughout life. Neural circuits, particularly those that are specialized for learning, continue to adapt their architecture in response to experience throughout the adult years. 10,11 Even circuits that pass through sensitive periods maintain a degree of flexibility that allows them to adapt their architecture, at least partially, to experience in adulthood. 12,48 The plasticity of many of these circuits in adult animals can be enhanced significantly by intentionally drawing attention to the information that is being processed by the circuit.<sup>10</sup> For example, plasticity in the representation of sound frequencies in the auditory cortex can be induced in adults—long after the appropriate sensitive period has ended-by having adult animals attend to particular sound frequencies to receive a food reward.<sup>49</sup> The residual capacity for plasticity in mature neural circuits thus allows for some recovery of brain capabilities, even in adults. In order for the brain to take full advantage of this plasticity, experience needs to be tailored to activate the relevant neural circuits and the individual's attention must be engaged in the task. The implications for later interventions in development are clear—the task will be harder, more expensive in terms of societal and individual effort, and potentially less extensive and durable.

# **Popular Misrepresentations of Science**

AS ADVANCES IN NEUROSCIENCE HAVE RECEIVED increasing attention, there has been parallel growth in the appetite for information about how to use scientific knowledge to enhance early brain development. This creates both important opportunities for more informed investments in young children, and the danger of unrealistic or misleading applications, sometimes with altruistic intentions and at other times simply for commercial profit. Within this context, it is essential that we differentiate scientific fact from common misperceptions.

Although a great deal of brain architecture is shaped during the first three years, claims that the window of opportunity for brain development closes on a child's third birthday are completely unfounded. Basic aspects of brain function, such as our ability to see and hear effectively, do depend critically on very early experiences. Some aspects of emotional development also conform to this concept. Nevertheless, vast regions of the brain that are responsible for higher order functions—including most cognitive, social, and emotional capacities—have not yet begun to mature by age three or are at extremely early stages of maturation. Thus, although the basic principle of early plasticity generally applies (i.e., "earlier is better than later"), the important time periods for experience depend on the specific function of interest. For most functions, the window of opportunity remains open well beyond age three.

Studies of the adverse effects of deprivation on brain development tell us little about the benefits of enrichment. Much of what we know about the impact of early experience on brain architecture comes from animal or human studies of deprivation. Examples include the negative effect on the development of vision from a cataract present at birth or an untreated strabismus (i.e., "lazy eye") early in life; adverse impacts on language and behavior as a result of delayed detection and intervention for a congenital hearing impairment; and the devastating effects on all aspects of development when a child is brought up in a bleak and neglectful

orphanage. It is important to emphasize, however, that well-documented, scientific evidence of the negative impacts of deprivation on brain circuitry does not necessarily mean that excessive enrichment produces measurable enhancements in brain architecture.

There are no credible scientific data to support the claim that specialized videos or particular music recordings (e.g., "the Mozart Effect") have a positive, measurable impact on developing brain architecture. Beyond recent research that has argued against such claims,<sup>50</sup> evidence from decades of scientific investigation of experience-

Well-documented, scientific evidence of the negative impacts of deprivation on brain circuitry does not mean that excessive enrichment produces measureable enhancements in brain architecture.

induced changes in brain development makes it highly unlikely that the potential benefits of such media would even come close to matching (much less exceeding) the more important influences of attentive, nurturing, and growthpromoting interactions with invested adults. Although a varied array of experiences clearly stimulates learning in the preschool years, promotional statements about the superior brainbuilding impacts of expensive "educational" toys and videos for infants and toddlers have no scientific support. 51,52 Similarly, didactic instruction in skill areas that are developmentally inappropriate for young children (i.e., the underlying neural circuitry necessary to master the particular skill has not developed) is an exercise in futility. Attempting to teach one-year olds to read is an example of such misguided efforts. The issue is not whether the child is "smart enough" or "motivated" to learn, but whether the necessary brain circuitry is sufficiently "wired" to support the specific domains required for that learning.

# The Science-Policy Gap

PRACTICAL EXPERIENCE TELLS US THAT IT IS easier to teach a "slow" first grader how to read than it is to train an illiterate adult for a job that pays a living wage. We don't need sophisticated research to prove that aggressive preschoolers are easier to "rehabilitate" than violent criminals. Common sense tells us that the learning and behavior problems of young children can be fixed more easily and at less cost than those of adolescents and young adults. Neuroscience tells us why these statements are all true.

Scientific evidence about how brains develop makes it very clear that neural circuits are shaped by time-specific experiences, and that the impact of a given experience is influenced by the nature of the circuits that are being formed at that time.

# The convergence of neuroscience and economics tells us that the clock is always ticking, and the costs of ignoring problems keep rising.

Moreover, the convergence of neuroscience and economics tells us that the clock is always ticking, and the costs of ignoring problems keep rising as time passes. Notwithstanding these fundamental principles of biology and human capital formation, the critical importance of time is often ignored in the world of early childhood policy. This striking gap between science and policy is illustrated by the following examples.

The child welfare system is typically characterized by cumbersome and protracted decisionmaking processes that leave young children vulnerable to the adverse impacts of significant stress during sensitive periods of early brain development. The powerful and far-reaching effects of severely adverse environments and experiences on brain development make it crystal clear that time is not on the side of an abused or neglected child whose physical and emotional custody remains unresolved in a slow-moving bureaucratic process. The basic principles of neuroscience indicate the need for a far greater sense of urgency regarding the prompt resolution of such decisions as when to remove a child from the home, when and where to place a child in foster care, when to terminate parental rights, and when to move towards a permanent placement. The window of opportunity for remediation in a child's developing brain architecture is time-sensitive and time-limited.

Education reform efforts that invest significant resources in the training, recruitment, and retention of skilled teachers for K-12 will have greater impact if they also include higher standards and more rigorous professional credentials for preschool programs. Research shows that staff knowledge and skills are among the most important determinants of the impact of early childhood programs.<sup>53,54</sup> Consequently, when model programs that have been proven to be effective are "taken to scale" with less well-compensated personnel who have less expertise, it is not surprising that comparable benefits are often not realized.<sup>23</sup> Stated simply, effective preschool investments require well-trained staff whose knowledge and skills match the needs of the children and families they are asked to serve. Poorly qualified personnel (whose low salaries provide immediate cost savings) compromise the effectiveness of preschool education programs and diminish the ultimate returns that can be achieved from subsequent K-12 investments.

Education policies disregard fundamental concepts of neuroscience when they delay teaching second languages until early adolescence and simultaneously undervalue bilingual programs for **young children.** Beginning at birth, all children have the capacity to learn any of the world's languages. This ability is encoded in our genes and activated by exposure to everyday conversation in an interactive way. Unless a child has a specific disability, the achievement of fluency in any language, as well as the mastery of more than one language at the same time, does not require formal instruction or intervention in the early childhood years. It simply requires ongoing communication with others. Moreover, the younger the brain, the greater its capacity to master more than a single langauge. If education policies were guided by what we know about the development of the brain, second-language learning would be a preschool priority.

# **Implications for Policy and Programs**

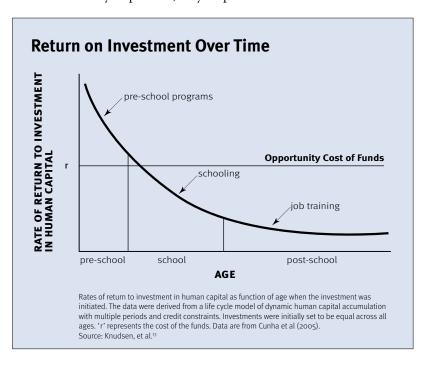
THE SCIENCE OF EARLY BRAIN DEVELOPMENT is sufficiently mature to support a number of evidence-based implications for those who develop and implement policies that affect the health and well-being of young children. Central to this conclusion are the core concepts of sensitive periods and neuroplasticity, which convey three important messages. First, both brain development and behavior are shaped by experience over time. Second, both the architecture of the brain and established patterns of behavior are increasingly difficult to change as individuals get older. Third, it is more effective and more efficient to get things right the first time than to try to fix them later.

There is considerable evidence that public policies can have a significant impact on promoting the healthy development of young children, above and beyond the central importance of family influences. This is particularly compelling for children who experience significant adversity during the early childood years. The following four points are particularly worthy of thoughtful consideration.

The basic principles of neuroscience and the econometrics of human capital development both suggest that early and effective intervention for the most vulnerable children will generate the greatest financial payback. In recent years, a growing body of sophisticated economic analyses has contributed an important new dimension to the public debate about the value to society of investing in the care and education of young children who are at risk for later failure in school and in the workplace. Extensive data now indicate that policymakers can achieve greater return on investments in early education for children from families with low income and limited parent education than they can from investments in remedial programs for adults with limited workforce skills. 13,55 In short, although optimal financial benefits depend on continued investment throughout the middle childhood years, the greatest returns are realized when investments are made in the lives of vulnerable children well before they begin school.

Increasing the availability of evidence-based, two-generation programs that begin immediately after birth (and preferably prenatally), can enhance the experiences of young children in families with limited education and low income. The environment of relationships in which young children live literally shapes the architecture of their brains. Effective programs provide centerbased, growth-promoting experiences for the children, as well as help their parents create a home environment that provides the kind of positive social interactions, rich language exposure, and early literacy experiences that increase the probability that their child will enter school with the social, emotional, and cognitive skills needed to succeed. These supportive interventions can be made available through voluntary associations, community-based organizations, and employer-sponsored initiatives, as well as through government-funded services. Because not all such services are effective, it is essential that funds be invested in programs that have been shown to have measurable impacts.<sup>23</sup>

Enrolling all children who meet the eligibility criteria for early intervention programs as early as possible would help infants and toddlers with developmental delays and disabilities build the foundational skills needed to realize their full **potential.** When compensatory adjustments are facilitated as early as possible, they help build a



sturdier foundation for the later achievement of higher-level skills. This underscores the urgent need to identify sensory impairments as soon after birth as possible, so that corrective devices (e.g., hearing aids and eyeglasses) as well as appropriate habilitative services can be provided during the time that basic neural circuits are being established. Outcomes for children with cognitive impairments are also improved significantly by the facilitation of early learning experiences that build a stronger foundation upon which increasingly higher-level brain circuits and more complex skills can be built over time.

Providing developmental assessments and intervention services for young children experiencing significant adversity *before* they exhibit problems in their behavior or development will increase their chances for more positive life outcomes. Strong and persistent activation of the body's stress response systems (i.e., increases in

heart rate, blood pressure, and stress hormones such as cortisol and cytokines) can result in the permanent disruption of brain circuits during the sensitive periods in which they are maturing. Common causes of such "toxic" stress include child abuse, serious neglect, and prolonged or repeated exposure to violence, which may be associated with deep poverty, parental substance abuse, or maternal mental illness, such as severe depression. The provision of both prevention and early intervention services for the large number of young children and families currently engaged in the nation's child welfare systems offers a compelling and promising place to start. Although this would require significant increases in short-term funding, effective programs for such highly vulnerable, young children are likely to generate a substantial return on investment through significant reductions in the later costs of special education, grade retention, welfare assistance, and incarceration.<sup>23</sup>

# References

- Hensch, T.K. (2005). Critical period mechanisms in developing visual cortex. Current Topics in Developmental Biology, 69, 215-237.
- 2. Horn, G. (2004). Pathways of the past: the imprint of memory. *Nature Reviews Neuroscience*, 5, 108-120.
- Friederici, A.D. (2006). The neural basis of language development and its impairment. *Neuron*, 52, 941-952.
- Katz, L.C. & Shatz, C.J. (1996). Synaptic activity and the construction of cortical circuits. *Science*, 274, 1133-1138.
- Singer, W. (1995). Development and plasticity of cortical processing architectures. *Science*, 270, 758-764.
- Majdan, M. & Shatz, C.J. (2006). Effects of visual experience on activity-dependent gene regulation in cortex. *Nature Neuroscience*, 9, 650-659.
- Grossman, A.W., Churchill, J.D., McKinney, B.C., Kodish, I.M, Otte, S.L., & Greenough, W.T. (2003). Experience effects on brain development: possible contributions to psychopathology. *Journal of Child Psychology and Psychiatry*, 44, 33-63.
- 8. Knudsen, E.I. (2004). Sensitive periods in the development of the brain and behavior. *Journal of Cognitive Neuroscience*, 16, 1412-1425.
- Hess, E.H. (1973). Imprinting: Early experience and the developmental psychobiology of attachment. New York: Van Nostrand Reinhold Company.
- Keuroghlian, A.S. & Knudsen, E.I. (2007). Adaptive auditory plasticity in developing and adult animals. *Progress in Neurobiology*, 82, 109-121.
- Buonomano, D.V. & Merzenich, M.M. (1998). Cortical Plasticity: From Synapses to Maps. Annual Review of Neuroscience, 21, 149-186.
- Karmarkar, U.R. & Dan, Y. (2006). Experience-dependent plasticity in adult visual cortex. *Neuron*, 52, 577-585.

- Knudsen, E.I., Heckman, J.J., Cameron, J.L., & Shonkoff, J.P. (2006). Economic, neurobiological, and behavioral perspectives on building America's future workforce. *Proceedings of the National Academy of Sciences U S A*, 103, 10155-10162.
- 14. Tang, A.C., Akers, K.G., Reeb, B.C., Romeo, R.D., & McEwen, B.S. (2006). Programming social, cognitive, and neuroendocrine development by early exposure to novelty. Proceedings of the National Academy of Sciences U S A, 103, 15716-15721.
- Weaver, I.C., Cervoni N., Champagne F.A., D'Alessio, A.C., Sharma, S., Seckl, J.R., et al. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, 7, 847-854.
- Rice, D. & Barone, S., Jr. (2000). Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. *Environmental Health Perspectives*, 108(Suppl 3), 511-533.
- 17. Levitt, P. (2003). Structural and functional maturation of the developing primate brain. *Journal of Pediatrics*, 143, \$35-45.
- 18. Center on the Developing Child at Harvard University. (2006). Early exposure to toxic substances damages brain architecture, Working Paper No. 4. http://www.developingchild.net/pubs/wp/Early\_Exposure\_Toxic\_Substances\_Brain\_Architecture.pdf.
- Sabatini, M.J., Ebert P., Lewis, D.A., Levitt, P., Cameron, J.L, Mirnics, K. (2007). Amygdala gene expression correlates of social behavior in monkeys experiencing maternal separation. *Journal of Neuroscience*, 27, 3295-3304.
- Kuhl, P.K. (2004). Early language acquisition: cracking the speech code. Nature Reviews Neuroscience, 5, 831-843.

- 21. Matsuzawa, T., Tomonaga, M., & Tanaka, M. (eds.). (2006). Cognitive Development in Chimpanzees. Tokyo: Springer.
- 22. Center on the Developing Child at Harvard University. (2005). Excessive stress disrupts the architecture of the developing brain, Working Paper No. 3. http://www. developingchild.net/pubs/wp/Stress Disrupts Architecture\_Developing\_Brain.pdf.
- 23. National Scientific Council on the Developing Child. (2007). A science-based framework for early childhood policy: Using evidence to improve outcomes in learning, behavior and health for vulnerable children. http://www.developingchild.net/pubs/persp/pdf/Policy\_ Framework.pdf.
- 24. Gogtay, N., Giedd, J.N., Lusk, L., Hayashi, K.M., Greenstein, D., Vaituzis, A.C., et al. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. Proceedings of the National Academy of Sciences USA, 101, 8174-8179.
- 25. Yurgelun-Todd, D. (2007). Emotional and cognitive changes during adolescence. Current Opinion in Neurobiology, 17, 251-257.
- 26. Pascalis, O., de Haan, M., & Nelson, C.A. (2002). Is face processing species-specific during the first year of life? Science, 296, 1321-1323.
- 27. Burkhalter, A., Bernardo, K.L., & Charles, V. (1993). Development of local circuits in human visual cortex. Journal of Neuroscience, 13, 1916-1931.
- 28. Scherf, K.S., Behrmann, M., Humphreys, K., & Luna, B. (2007). Visual category-selectivity for faces, places and objects emerges along different developmental trajectories. Developmental Science, 10, F15-30.
- 29. Golarai, G., Ghahremani, D.G, Whitfield-Gabrieli, S., Reiss, A., Eberhardt, J.L., Gabrieli, J.D, et al. (2007). Differential development of high-level visual cortex correlates with category-specific recognition memory. Nature Neuroscience, 10, 512-522.
- 30. Pascalis, O., Scott, L.S., Kelly, D.J., Shannon, R.W., Nicholson, E, Coleman, M., et al. (2005). Plasticity of face processing in infancy. Proceedings of the National Academy of Sciences U S A, 102, 5297-5300.
- 31. Thompson, R.A. (2001). Development in the first years of life. The future of children, 11, 20-33.
- 32. Weaver, I.C., Champagne, F.A., Brown, S.E., Dymov, S., Sharma, S., Meany, M.J., et al. (2005). Reversal of maternal programming of stress responses in adult offspring through methyl supplementation: altering epigenetic marking later in life. Journal of Neuroscience, 25, 11045-11054.
- 33. DeBello, W.M., Feldman, D.E., & Knudsen, E.I. (2001). Adaptive axonal remodeling in the midbrain auditory space map. Journal of Neuroscience, 21, 3161-3174.
- 34. Hubel, D.H. & Wiesel, T.N. (1977). Ferrier Lecture: Functional architecture of macaque monkey visual cortex. Proceedings of the National Academy of Sciences, 198, 1-59.
- 35. Newport, E.L., Bavelier, D., & Neville, H.J. (2001). Critical thinking about critical periods: Perspectives on a critical period for language acquisition. In E. Doupoux (Ed.), Language, brain and cognitive development: Essays in honor of Jacques Mehler (pp 481-502). Cambridge, MA: MIT Press.
- 36. Feldman, D.E. (2000). Inhibition and plasticity. Nature Neuroscience, 3, 303-304.
- 37. Zheng, W. & Knudsen, E.I. (2001). GABAergic inhibition antagonizes adaptive adjustment of the owl's auditory space map during the initial phase of plasticity. Journal of Neuroscience, 21, 4356-4365.

- 38. Daw, N.W. (1997). Critical periods and strabismus: what questions remain? Optometry and Vision Science, 74, 690-694.
- 39. Jones, E.G. (2000). Cortical and subcortical contributions to activity-dependent plasticity in primate somatosensory cortex. Annual Review of Neuroscience, 23, 1-37.
- 40. Doupe, A.J. & Kuhl, P.K. (1999). Birdsong and Human Speech: Common Themes and Mechanisms. Annual Review of Neuroscience. 22, 567-631.
- 41. Black, J.E. & Greenough, W.T. (1986). Induction of pattern in neural structure by experience: Implications for cognitive development. In M.E. Lamb, A.L. Brown, & B. Rogoff (Eds.), Advances in developmental psychology, Volume 4 (pp 1-50). Hillsdale, NJ:.Lawrence Erlbaum Associates.
- 42. DeBello, W.M. & Knudsen, E.I. (2004). Multiple sites of adaptive plasticity in the owl's auditory localization pathway. Journal of Neuroscience, 24, 6853-6861.
- 43. Nelson, C.A., de Haan, M., & Thomas, K.M. (2006). Neural bases of cognitive development. In W. Damon, R. Lerner, D. Kuhn, & R. Siegler (Eds.), Handbook of Child Psychology, Volume 2. New Jersey: John Wiley & Sons, Inc..
- 44. Antonini, A. & Stryker, M.P. (1993). Rapid remodeling of axonal arbors in the visual cortex. Science, 260, 1819-1821.
- 45. Nelson, C.A. (2007). A neurobiological perspective on early human deprivation. Child Development Perspectives, 1, 13-18
- 46. Linkenhoker, B.A., von der Ohe, C.G., & Knudsen, E.I. (2005). Anatomical traces of juvenile learning in the auditory system of adult barn owls. Nature Neuroscience, 8, 93-98.
- 47. Antonini, A., Fagiolini, M., & Stryker, M.P. (1999). Anatomical correlates of functional plasticity in mouse visual cortex. Journal of Neuroscience, 19, 4388-4406.
- 48. Bergan, J.F., Ro, P., Ro, D., & Knudsen, E.I. (2005). Hunting increases adaptive auditory map plasticity in adult barn owls. Journal of Neuroscience, 25, 9816-9820.
- 49. Polley, D.B., Steinberg, E.E., & Merzenich, M.M. (2006). Perceptual learning directs auditory cortical map reorganization through top-down influences. Journal of Neuroscience, 26, 4970-4982.
- 50. Jones, S.M. & Zigler, E. (2002). The Mozart effect: Not learning from history. Applied Developmental Psychology, 23, 355-372.
- 51. Zimmerman, F.J., Christakis, D.A., & Meltzoff, A.N. (2007). Television and DVD/video viewing in children younger than 2 years. Archives of Pediatrics & Adolescent Medicine, 161, 473-479.
- 52. Zimmerman, F.J., Christakis, D.A., & Meltzoff, A.N. (2007). Associations between media viewing and language development in children under age 2 years. Journal of Pediatrics, 151, 364-368.
- 53. Gormley, W.T., Jr., Gayer, T., Phillips, D., & Dawson, B. (2005). The effects of universal pre-K on cognitive development. Developmental Psychology, 41, 872-884.
- 54. Early, D.M., Maxwell, K.L., Burchinal, M., Alva, S., Bender, R.H., Bryant, D., et al. (2007). Teachers' education, classroom quality, and young children's academic skills: results from seven studies of preschool programs. Child Development, 78, 558-580.
- 55. Cunha, F., Heckman, J., Lochner, L., & Masterov, D. (2005). Interpreting the evidence on life skill formation, Working Paper #10091. Cambridge, MA: National Bureau of Economic Research.

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### The Issue

THE SCIENCE OF EARLY CHILDHOOD DEVELOPMENT TELLS US THAT THE FOUNDATION FOR SOUND mental health is built early in life, as early experiences shape the architecture of the developing brain. These important experiences include children's relationships with parents, caregivers, relatives, teachers, and peers, which play a critical role in shaping social, emotional, and cognitive development. While concerns about cognition and language are already the focus of much public attention, emerging emotional and behavioral difficulties in the early years are also important societal issues that must be addressed. Attention to early mental health concerns is warranted because these kinds of problems impair emerging capacities for learning and relating to others.

Sound mental health provides an essential foundation of stability that supports all other aspects of human development—from the formation of friendships and the ability to cope with adversity to the achievement of success in school, work, and community life. Similar to the way a wobbly table may not function well if the floor is uneven, the legs are not aligned, or the tabletop is not level, the destabilizing consequences of problems in mental health can be caused by many interdependent factors. Just as small "wobbles" in a table can become bigger and more difficult to fix over time, the effective management of mental health concerns in young children requires early identification of the causes and appropriate attention to their source, whether they reside in the environment, the child, or (most frequently) in both. Understanding how emotional well-being can be strengthened or disrupted in early childhood can help policymakers promote the kinds of environments and experiences that prevent problems and remediate early difficulties so they do not destabilize the developmental process.

The scientific evidence is clear: Significant mental health problems can and do occur in young children. In some cases, these problems can have serious consequences for early learning, social competence, and even lifelong health. Children can show clear characteristics of anxiety disorders, attention-deficit/hyperactivity disorder, conduct disorder, depression, posttraumatic stress disorder, and other problems at a very early age.1 And older children often exhibit the emotional legacy of early abuse or neglect.<sup>2</sup> Early mental health problems merit attention because they disrupt the typical patterns of developing brain architecture and impair emerging capacities for learning and relating to others. And regardless of the origin of mental health concerns, new research clearly indicates that early intervention can have a positive impact on the trajectory of common emotional or behavioral problems as well as outcomes for children with serious disorders.

All children experiencing prolonged adversity are at risk for cognitive and mental health problems. Studies show, however, that the long-term impact on physical and mental health is most likely to affect individuals who are genetically more vulnerable to stress. But genetics is neither

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destiny nor "hard-wired"; our genes contain instructions that tell our bodies how to work, but the environment leaves a signature on the genes that authorizes or prevents those instructions from being carried out (or even speeds them up or slows them down). Thus, the interaction between genetic predispositions and sustained, stress-inducing experiences early in life can lay an unstable foundation for mental health that endures well into the adult years. Early exposure to child abuse or neglect, family turmoil, neighborhood violence, extreme poverty, and other threats in a child's environment can prime neurobiological stress systems to become hyperresponsive to adversity.3 Adverse experiences such as these early in life, particularly for vulnerable children, predict the emergence of later physical and mental health problems, including disorders like major depression.4,5

Although mental health challenges for young children share many biological and behavioral characteristics with those of older children and adults, there are at least three ways in which early childhood is a period of special vulnerability. First, psychological health for young children is very strongly influenced by their environment of relationships and the support or risks these relationships confer.6 These relationships can

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buffer the impact of adverse early experiences to make them tolerable. Therefore, to understand the reasons that young children may be at risk for impairments, how best to provide assistance, and the strategies for promoting positive mental health, it is important to look at the quality of their early environments and relationships. To a greater extent than is true of older children and adults, viewing the child alone as the "patient," or the source of the problem, can lead to costly or ineffective policies and practices.

Second, cognitive, social, and emotional capacities are all intertwined within the architecture of the brain, and these capacities are

qualitatively different at different ages, as the brain matures. For example, the area of the brain that enables us to extinguish a learned fear develops later than the area that elicits a fear response.7 So, young children respond to and process emotional experiences and traumatic events in ways that are very different from older children and adults. Children understand, manage, think, and talk about their experiences differently at different ages. These developmental differences are important to understanding the behavioral and emotional disturbances that young children may experience, how the problems are manifested, and how they can be ameliorated.

Third, in early childhood, it can be difficult to distinguish short-lived variations in behavior from persistent problems, or typical differences in maturation from significant developmental delays.8 Although many enduring mental health problems have their origins in the early years, some behavioral or emotional difficulties in children and even adolescents can be transient. 9,10,11 Thus, while a range of strategies is available to treat or prevent difficulties, diagnosis in early childhood can be even more difficult than it is in adults. Caution is needed when evaluating an infant or young child for potential indicators of emotional or behavioral problems and matching those indicators to appropriate treatments.

# What Science Tells Us

Toxic stress early in life can damage the architecture of the developing brain and increase the likelihood of significant mental health problems that may emerge either early or years later. 3,12,13,14,15,16,17,18 Life circumstances associated with family stress in the absence of supportive adult relationships, such as persistent poverty, threatening neighborhoods, and very poor child care conditions, elevate the risk of serious mental health problems and undermine healthy functioning in the early years. 19 Early childhood adversity of this kind also increases the risk of adult physical and mental health problems because of its enduring effects on the developing brain and other maturing organs.20 Young children who experience recurrent abuse or chronic

neglect, regularly witness domestic violence, or live in homes burdened by parental mental health or substance abuse problems are particularly vulnerable. Relationship-based circumstances contributing to early emotional difficulties, such as maternal depression, also have welldocumented adverse effects on developing brain function in the early years. 21,22,23,24,25

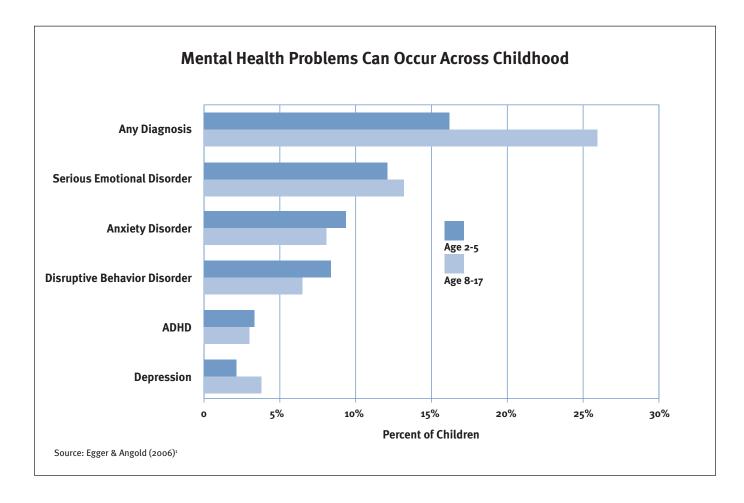
Toxic stress can lead to persistent activation of biological stress response systems. This produces abnormal levels of stress hormones that have the capacity to damage brain architecture if they do not normalize. In the absence of the buffering protection of supportive relationships, these atypical hormone levels interfere with the development of healthy brain architecture. This

poses a serious threat to young children, not only because it undermines their emotional wellbeing, but also because it can impair a wider range of developmental outcomes, including early learning, school readiness, and later academic achievement. 12,18,26,27,28,29,30,31,32

Much impairment in mental health arises as a result of the interaction between a child's genetic predisposition and his or her exposure to significant environmental adversity. Not all stressful experiences are damaging, and those that are positive, like an initial fear of pets, can strengthen adaptive responses to short-lived stress for a lifetime. However, exposure to early traumatic or abusive experiences can be toxic to developing brain circuitry and can combine with differences in individual behavioral styles (which child development researchers call "temperament") to influence the severity of the long-term mental health consequences. A young child with a genetic tendency to fearfulness, for example, is more likely to develop anxiety or depression

than a child without that predisposition. This is particularly powerful in the context of harsh, inconsistent relationships and experiences, such as those associated with deep poverty, poor-quality child care in the family's community, or a depressed mother. In other words, early adversity acts as the "signature" that releases a child's genetic predisposition for anxiety, building a brain architecture that responds to lower levels of stress with excessive fear and anxiety, leading to lifelong consequences for mental health.33

This nature-nurture interaction is illustrated in studies of behavioral inhibition, an earlyemerging pattern of fearful, withdrawn behavior that is a risk factor for later anxiety disorders.<sup>6,34</sup> In a recent report, behavioral inhibition at age 7 was related to the interaction of two earlier influences: (a) a gene that is associated with anxiety and fear in adults, and (b) the mother's report that she lacked social support from others, which may be associated with toxic stress for her children. Put another way, the interaction between a genetic tendency toward anxiety and



the experience of early life stresses best predicted which children remained behaviorally inhibited at age 7.35,36 Such behavioral inhibition may be related to the development of more serious problems later in life, as additional research shows that children who are behaviorally inhibited show different patterns of activation of brain regions related to emotional withdrawal and fear compared with children whose behavior is more typical. 37,38,39,40

The behaviors and characteristics associated with mental health problems in the earliest years of life are often different from those seen in older children and adults with psychological difficulties. 41,42,43 Young children's brains are not fully developed, and they do not respond to stressful events the way adults do. A toddler who is coping with trauma or the loss of a loved one

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acts differently from a traumatized adolescent because of different psychological capabilities, emotional needs, and social experiences at different ages. Young children manifest the symptoms of depression or post-traumatic stress disorder (PTSD) differently than young adults. Some mental health problems, such as attachment-related disorders (i.e., profound disturbances in close relationships with caregivers), are specific to early childhood. Thus, although adult diagnostic approaches can provide some guidance for understanding the kinds of problems that younger children may experience, new approaches to assessment and diagnosis based on the unique developmental needs and characteristics of young children are extremely important.43,44

Over the past few years, researchers have validated diagnostic criteria specific to young children that are useful in identifying early forms of depression, post-traumatic stress disorder, autism, disruptive behavior disorders, anxiety disorders, and attention deficit/hyperactivity disorder. however, the accurate identification of serious mental health disorders during the first three

to four years of life remains a challenging task. Consequently, it is unwise to assume that early problems can be classified simply into one category within a diagnostic system. Indeed, young children, like older children and adults, frequently experience multiple problems (known as "co-morbidity"). This is illustrated by the co-occurrence of depression with oppositional-defiant disorders in early childhood, or the increased prevalence of depression or other emotional problems in children with autism.<sup>1,52,53,54</sup>

If young children are not provided appropriate help, emotional difficulties that emerge early in life can become more serious disorders over time. 55,56,57 Early prevention strategies and efforts to identify and treat emergent mental health concerns are likely to be more psychologically beneficial and cost-effective than trying to treat emotional difficulties after they become more serious at a later age. This field urgently needs treatment strategies that are age-appropriate, support the development of healthy relationships, and are consistent with scientific knowledge about early psychological development. Promising approaches for some early mental health challenges are well-described in the research literature,58,59,60 yet they are not widely available. Other problems have been less wellstudied in very young children. Nevertheless, many disorders can be prevented before they begin by providing access to developmentally appropriate, high-quality early care and education. Systems of support are particularly important, as they assist parents and caregivers in providing warm and secure relationships and in detecting emotional problems before they become more resistant to change. Public policies are also essential to help ameliorate the physical, social, and economic conditions that cause some families to struggle.

Some individuals demonstrate remarkable capacity to overcome the severe challenges of early, persistent maltreatment, trauma, and emotional harm. Yet, there are limits to the capacity of young children to recover psychologically from such adversity. 61,62,63,64 Even under circumstances in which children have been removed from traumatizing conditions and placed in exceptionally nurturing homes, developmental improvements are often

accompanied by continuing problems in self-regulation, emotional adaptability, relating to others, and self-understanding. There also is research indicating that long-term physical health can be affected by early life adversity. This is seen in the form of increased risk of heart disease, diabetes, hypertension, and other physical ailments, as toxic stress can literally be "built" into the body. 5 When children overcome these burdens, they have typically been the beneficiaries of exceptional efforts on the part of supportive adults. These findings underscore the importance of prevention and timely intervention in circumstances that put young children at severe psychological risk.

Serious developmental disabilities can also be associated with significant mental health impairments that are affected by experience and amenable to intervention. Neurodevelopmental disorders, such as autism, fragile X syndrome, and Down syndrome, for example, are the result of strong genetic influences. Nevertheless, genetics is only part of the story. Although disorders such as Down syndrome have a specific genetic cause, mental health outcomes for these children are also affected by the quality of care and support they receive, compared with typically developing children. The possibility of significant improvement in quality of life, as well as in both cognitive and social functioning, as a result of prompt intervention provides a strong argument for the early detection and treatment of these developmental disorders. This is becoming increasingly apparent with respect to early intervention for autism.<sup>65</sup>

It is essential to treat young children's mental health problems within the context of their family, home, and community environments. The powerful influence of a child's early environment of relationships illustrates how much the emotional well-being of young children is directly tied to the emotional functioning of their caregivers and the families in which they live. 66 When these relationships are abusive, threatening, chronically neglectful, or otherwise psychologically harmful, they are a potent risk factor for the development of early mental health problems. In contrast, when positive "serve and return" relationships develop, which occur through reliably responsive and supportive interactions, they can actually buffer young children from the adverse effects of other stressors. 16,61,67,68,69 Addressing the sources of toxic stress affecting a child requires relieving the stressors on his or her family in order to ensure that this broader environment of relationships can be maximally supportive.

For many parents and providers of child health services and early care and education who are faced with children who present problematic behavior, the question of "when to worry" is paramount, yet little evidence exists to answer that question definitively in most circumstances. Although early mental health problems can foreshadow enduring disorders, many difficulties are transient and disappear with appropriate management and further maturation.9,10,11 Generally speaking, clinical experts advise greater concern when children exhibit constellations of problems (e.g., persistent irritability or eating and sleeping problems, combined with defiance) that lead to significant impairments in age-appropriate behavioral skills and relationships.

# **Addressing Common Misconceptions**

AS THE PUBLIC DEVOTES MORE ATTENTION TO the relation between early brain development and the emotional well-being of young children, the risk of misinformation and misleading or irresponsible messages also grows. Within this context, it is essential that we distinguish scientific fact from erroneous fiction. The following two misconceptions are particularly important to set straight.

Contrary to popular belief, young children can and do experience serious mental health problems that are comparable in severity to what we observe in older children and adults, and they can have lasting effects. Although young children are not as psychologically sophisticated as adults, research on early childhood development shows that they are capable of experiencing peaks of joy and elation as well as depths of grief, sadness, hopelessness, intense anger, and rage. Contrary to traditional views, highly negative emotional experiences in early childhood are not "forgotten"; they are built into the architecture of the developing brain and can have a sustained impact that extends well into the adult years, especially when they are severe, persistent, and uncontrollable. Adverse community and family environments can have a similarly enduring emotional impact on young children when they are experienced as toxic stress and not buffered by supportive relationships.

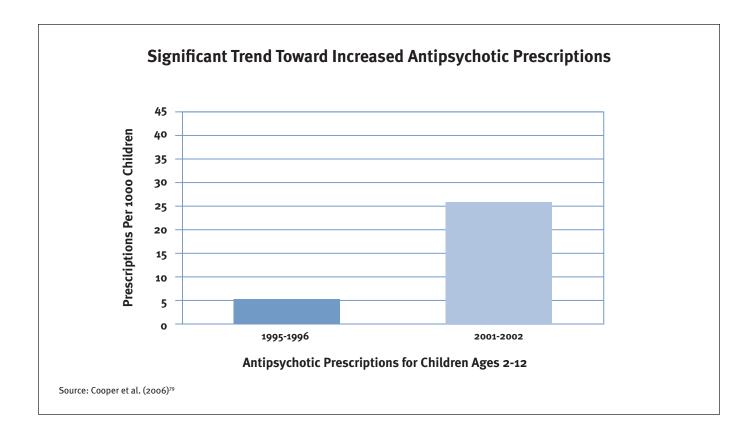
Contrary to popular belief, young children living in highly threatening environments can be

protected from serious emotional or behavioral consequences. Although such conditions increase their risk for serious mental health problems, learning impairments, and long-term physical illnesses, children who experience serious threats to their psychological health, such as those who are physically abused, chronically neglected, or emotionally traumatized, do not inevitably develop significant mental illnesses. These children can be protected through the early identification of their emotional needs and the provision of appropriate assistance in the context of stable, nurturing relationships with supportive and skilled caregivers as well as through preventive mental health services. 62,64,70

# The Science-Policy Gap

THE FACT THAT YOUNG CHILDREN CAN PRESENT challenging behaviors is hardly news to the adults who care for them. It is less well-known that some serious behavior problems in the early years of life may be the first signs of potentially lifelong mental health disorders that are preventable

if treated at a young age. Very young children can experience significant impairments in their mental health that are embedded in the developing architecture of their brains and may have lifelong consequences, according to a rich and growing science base. Yet, relatively little



attention has been paid to the formulation and implementation of strategies to identify children who are at risk for such problems and to provide supports for them and their families that will increase the probability of more favorable outcomes. This gap between what we know and what we do is illustrated by the following three examples.

Professionals who are regularly involved in the lives of infants, toddlers, and preschoolers often lack the knowledge and skills that would help them identify the early signs of mental health problems as well as fully understand the consequences of family difficulties and parent mental health problems for young children's develop**ment.** These professionals include child care providers and preschool teachers (who are often the first people outside the family to identify a child who has serious emotional difficulties), physicians and other health care providers (who often lack a sophisticated understanding of psychological development and early mental health), paraprofessional home visitors, program administrators and personnel in social service, child protection, early intervention, and welfare agencies, among others who regularly serve families with young children.

In most communities, mental health services for young children and their families are often limited, of uneven quality, and difficult to access, and there are relatively few well-trained professionals with expertise in early childhood mental health. Central to this problem is the need to close the gap between the large numbers of young children exhibiting emotional difficulties and/or problematic behavior that cannot be managed adequately by their parents and the limited number of personnel who are skilled in effective intervention approaches that are uniquely suited to this age group.

There has been a dramatic increase in the use of psychoactive drugs for treating young children with behavioral or mental health problems, despite the fact that neither the efficacy nor safety of many of these medications has been studied specifically in children at these early ages.<sup>45</sup> A recent report from the National Survey of Children's Health, for example, reported that children ages 4 to 8 were more likely to be taking medication for attention deficit/hyperactivity disorder than older children and adolescents.71 Of even greater concern, some studies have reported increasing numbers of prescriptions for stimulant medications and antidepressants to treat children as young as age 3.72 In most cases, these medications for young children are prescribed "off label," which means that they have only been approved for treating adults and that there are no scientific data on their immediate or long-term effects on child behavior or early brain development.<sup>45</sup> Until the relevant clinical studies have been completed with the appropriate populations of young children, the use of such medications must be viewed as experimental and their safety and effectiveness as unknown.73,74,75,76

# Implications for Policy and Programs

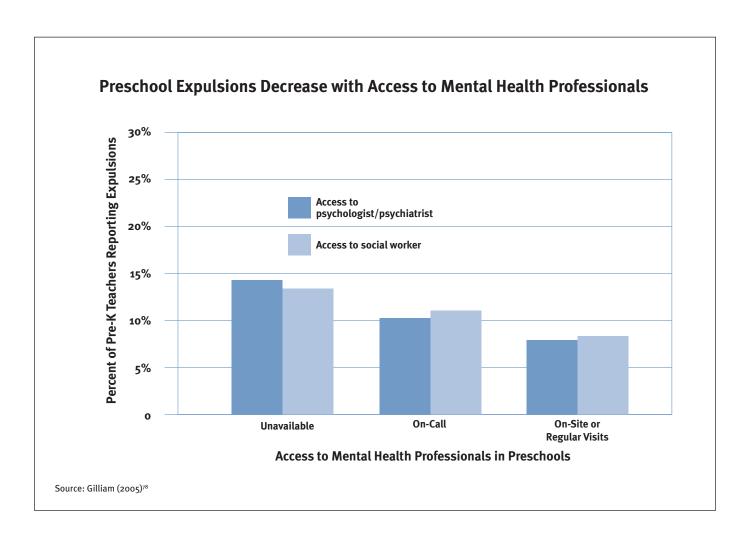
THE SCIENCE OF EARLY CHILDHOOD DEVELOPment, including knowledge about the extent to which serious emotional problems are embedded in the architecture of the developing brain, is sufficiently mature to support a number of evidence-based implications for those who develop and implement policies that affect the health and well-being of young children. Both public and private actions can prevent the kinds of adverse circumstances that are capable of derailing healthy development, as well as increase the likelihood that effective supports and appropriate therapeutic interventions (where

needed) will reduce the long-term consequences of early threats to a child's mental health. The following points are particularly worthy of thoughtful consideration.

Because young children's emotional well-being is tied so closely to the mental health of their parents and non-family caregivers, the emotional and behavioral needs of infants, toddlers, and preschoolers are best met through coordinated services that focus on their full environment of relationships. Multigenerational, family-centered approaches offer the most promising

models for preventing and treating mental health problems in young children. These strategies range from providing information and support to address problematic child behavior to initiating therapeutic interventions to attend to significant parent mental health or substance abuse problems, end domestic violence, or help families to cope with the burdens of persistent poverty. Indeed, sometimes the best intervention strategy for young children with serious behavioral or emotional problems is to focus directly on the primary needs of those who care for them. However, most approaches to funding mental health services are client-specific rather than family-focused, and most programs aimed at such "adult" problems as poverty, domestic violence, substance abuse, or depression do not take into consideration the emotional wellbeing of the children affected by them. More flexible approaches to funding family-based preventive and therapeutic mental health services are needed.

Therapeutic help for a young child with emotional or behavioral problems can be provided through a combination of home- and center-based services involving parents, extended family members, home visitors, providers of early care and education, and/or mental health professionals. The settings, partnerships, and targets of therapeutic assistance for young children with mental health needs are much more diverse than those for adults, because their emotional wellbeing is linked tightly to the quality of their relationships with the important people in their lives. Effective intervention often requires the coordination of services from multiple sources that do not relate easily. These might include early care and education, social service and welfare departments, health care, schools, child welfare agencies, and early intervention programs, to name a few. Reducing barriers to greater coordination often requires attention to a tangle of administrative obstacles. One example would be a change in reimbursement



regulations to allow "mental health funds" to be used to pay for specialized child care for a youngster with emotional and behavioral problems, rather than restricting the funds to only "mental health programs."

Mental health services for adults who are parents of young children would have broader impact if they routinely included attention to the needs of the children as well. Because of the close association between young children's emotional wellbeing and the emotional health and functioning of their caregivers,77 therapeutic assistance to a parent ought to include an automatic assessment of any young children in the family to see how they are experiencing the emotional consequences of their parent's problems. For example, any physician treating a depressed mother ought to understand the consequences of that diagnosis for her young children and therefore assure that they receive timely examinations and appropriate intervention, as needed.

Physicians and providers of early care and education would be better equipped to understand and manage the behavioral problems of young children if they had more intensive professional training focused on this area and easier access to child mental health professionals when needed. Caregivers, teachers, and physicians are often the first to recognize serious emotional difficulties in a child who is in their care, yet their training may include little information regarding an up-to-date understanding of child mental health. The availability of a rich scientific research base provides an opportunity to improve relevant curriculum and training programs. It also is clear that on-site assistance from early childhood mental health specialists can be particularly helpful in providing guidance about how best to respond to the needs of the children, their parents, and providers of early care and education. Preschool teachers with access to mental health consultation, for example, are less likely to expel children with behavioral problems from their programs.<sup>78</sup> Some states have made progress in providing funds for early childhood mental health consultations in early child care settings, often through the coordination of diverse funding streams. Broader attention to early childhood mental health requires attention to the quality of out-of-home care that children typically experience in the early years.

Cultural differences in attitudes and beliefs about mental health need to be recognized and included as factors when developing programs for prevention and intervention. The mental health needs of young children in families from different cultural and ethnic groups would benefit considerably from enhanced practitioner training and flexible service models that incorporate greater content representing a broad variety of populations. Cultural differences include how children are taught to interpret and express their experiences of fear, anger, and shame; the relative reinforcement given to individual achievement versus interdependent behavior; parent and caregiver attitudes about

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mental health and mental illness; and acceptance of therapeutic intervention for very young children by non-family members, among many other concerns. The shifting demographics that are increasing the diversity of the early childhood population in the United States make this a particularly compelling priority for attention. Finally, it is important to understand individual differences within cultural groups related to assimilation of immigrant groups across generations and changes in cultural beliefs and practices over time.

Better investment and coordination of resources to support mental health services for young children will provide a more stable and efficient vehicle for assuring access to effective prevention and treatment programs. Consistent with both current scientific knowledge (i.e., the physiological interrelations among the physical, cognitive, social and emotional dimensions of well-being in young children) and federal legislative support for parity in coverage of physical and mental health care, access to and quality of early childhood mental health services need to improve. Integrating efforts more effectively into a wide range of existing, effective programs is but one solution. Mental health services covered by the Early and Periodic Screening, Diagnosis, and Treatment (EPSDT) program, Children's Health Insurance Program (CHIP), early intervention (IDEA Part C) or maternal and child health programs should better coordinate with related programs such as child welfare, child care, Early Head Start and Head Start, home visiting, and other programs and settings.

FUNDING COORDINATION IS JUST ONE WAY WE could work to improve the environment of relationships and experiences that form the contexts for children's development. Innovative states and communities have been able to design strategies and programs to support such environments, and they have shown significant progress toward solving some of these very difficult problems associated with children's mental health. Our task is to help bring such innovations to scale for children and families in need.

# References

- Egger, H. L., & Angold, A. (2006). Common emotional and behavioral disorders in preschool children: Presentation, nosology, and epidemiology. *Journal of Child Psychology* and Psychiatry, 47(3-4), 313-337.
- Teisl, M., & Cicchetti, D. (2008). Physical abuse, cognitive and emotional processes, and aggressive/disruptive behavior problems. Social Development, 17(1), 1-23.
- Gunnar, M.R. (2007). Stress effects on the developing brain. In D. Romer, E.F. Walker (Eds.) Adolescent psychopathology and the developing brain: Integrating brain and prevention science. (pp. 127-147). New York: Oxford University Press.
- Edwards, V. J., Holden, G. W., Felitti, V. J., & Anda, R.F. (2003). Relationship between multiple forms of child maltreatment and adult mental health in community respondents: Results from the Adverse Childhood Experiences Study. *The American Journal of Psychiatry*, 160(8), 1453-1460.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, 14(4), 245-258.
- Rubin, K., Bukowski, W., & Parker, J. (2006). Peer interactions, relationships, and groups. In W. Damon & R. M. Lerner (Eds.), Handbook of child psychology (6th Edition): Vol. 3: Social, emotional, and personality development (N. Eisenberg, Vol. Ed.) (pp. 571-645). New York: John Wiley & Sons.
- National Scientific Council on the Developing Child. (2010). Persistent fear and anxiety can affect young children's learning and development: Working Paper 9. http://www.developingchild.net.
- Shonkoff, J. P., & Phillips, D. A. (Eds.) (2000). From neurons to neighborhoods: The science of early childhood development. Washington, DC: National Academy Press.
- Peterson, B. S., Pine, D. S., Cohen, P., & Brook, J. S. (2001). Prospective, longitudinal study of tic, obsessive-compulsive, and attention-deficit/hyperactivity disorders in an epidemiological sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40(6), 685-695.
- Pine, D. S., Cohen, P., Johnson, J. G., & Brook, J. S. (2002).
   Adolescent life events as predictors of adult depression.
   Journal of Affective Disorders, 68(1), 49-57.

- 11. Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). The risk for early-adult anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry*, 55(1), 56-64.
- Carrion, V., Weems, C., Ray, R., Glaser, B., Hessl, D., & Reiss, A. (2002). Diurnal salivary cortisol in pediatric posttraumatic stress disorder. *Biological Psychiatry*, 51(7), 575-582.
- 13. De Bellis, M., Baum, A., Birmaher, B., Keshavan, M., Eccard, C., Boring, A., Jenkins, F., & Ryan, N. (1999). A.E. Bennett Research Award: Developmental traumatology, Part I: Biological stress systems. *Biological Psychiatry*, 45(10), 1259-1270.
- De Bellis, M., Keshavan, M., Clark, D., Casey, B., Giedd, J., Boring, A., Frustaci, K., & Ryan, N. (1999). A.E. Bennett Research Award: Developmental traumatology, Part II: Brain development. *Biological Psychiatry*, 45(10), 1271-1284.
- Glaser, D. (2000). Child abuse and neglect and the brain—A review. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 41(1), 97-116.
- Gunnar, M.R., Morison, S.J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology*, 13(3), 611-628.
- Kaufman, J., & Charney, D. (2001). Effects of early stress on brain structure and function: Implications for understanding the relationship between child maltreatment and depression. *Development and Psychopathology*, 13(3), 451-471.
- 18. National Scientific Council on the Developing Child. (2005). Excessive stress disrupts the architecture of the developing brain: Working Paper 3. http://www.developingchild.net.
- Brooks-Gunn, J., & Duncan, G.J. (1997). The effects of poverty on children. The Future of Children, 7(2), 55-71.
- Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. Proceedings of the National Academy of Sciences of the United States of America, 104(4), 1319-1324.
- Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. *Archives of General Psychiatry*, 65(4), 409-416.

- 22. Dawson, G., Ashman, S., Panagiotides, H., Hessl, D., Self, J., Yamada, E., & Embry, L. (2003). Preschool outcomes of children of depressed mothers: Role of maternal behavior, contextual risk, and children's brain activity. Child Development, 74(4), 1158-1175.
- 23. Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The role of chaos in poverty and children's socioemotional adjustment. Psychological Science, 16(7), 560-565.
- 24. Evans, G. W., Kim, P., Ting, A. H., Tesher, H. B., & Shannis, D. (2007). Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. Developmental Psychology, 43(2), 341-351
- 25. Goodman, S., & Gotlib, I. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. Psychological Review, 106(3), 458-490.
- 26. Dawson, G,. & Ashman, S.B. (2000). On the origins of a vulnerability to depression: The influence of the early social environment on the development of psychobiological systems related to risk for affective disorder. In C.A. Nelson (Ed.), The effects of early adversity on neurobehavioral development: Minnesota Symposia on Child Psychology: Vol. 31. Mahwah, NJ: Erlbaum Associates.
- 27. Luby, J., Belden, A., & Spitznagel, E. (2006). Risk factors for preschool depression: The mediating role of early stressful life events. Journal of Child Psychology and Psychiatry and Allied Disciplines, 47(12),1292-1298.
- 28. Osofsky, J. (2004). Community outreach for children exposed to violence. Infant Mental Health Journal, 25(5),
- 29. Rubin, K., Burgess, K., Dwyer, K., & Hastings, P. (2003). Predicting preschoolers' externalizing behaviors from toddler temperament, conflict, and maternal negativity. Developmental Psychology, 39(1),164-176.
- 30. Scheeringa, M., & Zeanah, C. (1995). Symptom expression and trauma variables in children under 48 months of age. Infant Mental Health Journal, 16(4), 259-270.
- 31. Shaw, D., Owens, E., Giovannelli, J., & Winslow, E. (2001). Infant and toddler pathways leading to early externalizing disorders. Journal of the American Academy of Child and Adolescent Psychiatry, 40(1), 36-43.
- 32. Vasey, M., & Dadds, M., Eds. (2001). The developmental psychopathology of anxiety. New York: Oxford University Press.
- 33. National Scientific Council on the Developing Child. (2010). Early experiences can alter gene expression and affect long-term development: Working Paper 10. http:// www.developingchild.net.
- 34. Tincas, I., Benga, O., & Fox, N. (2006). Temperamental predictors of anxiety disorders. Cognitie, Creier, Comportament (Cognition, Brain, Behavior), 10(4), 489-515.
- 35. Fox, N., Hane, A., & Pine, D. (2007). Plasticity for affective neurocircuitry: How the environment affects gene expression. Current Directions in Psychological Science, 16(1), 1-5.
- 36. Fox, N., Nichols, K., Henderson, H., Rubin, K., Schmidt, L., Hamer, D., Ernst, M., & Pine, D. (2005). Evidence for a gene-environment interaction in predicting behavioral inhibition in middle childhood. Psychological Science, 16(12), 921-926.
- 37. Fox, N., Henderson, H., Marshall, P., Nichols, K., & Ghera, M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. Annual Review of Psychology, 56, 235-262.

- 38. Fox, N., Henderson, H., Rubin, K., Calkins, S., & Schmidt, L. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*(1), 1-21.
- 39. Pine, D. S. (2007). Research review: A neuroscience framework for pediatric anxiety disorders. Journal of Child Psychology and Psychiatry, 48(7), 631-648.
- 40. Schwartz, C., Wright, C., Shin, L., Kagan, J., & Rauch, S. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. Science, 300(5627), 1952-1953.
- 41. Doll, B., Brehm, K., Zucker, S., Deaver-Langevin, J., Griffin, J., & Hickman, A. (2000). Contrasting procedures for empirical support of traditional and populationbased mental health services. Psychology in the Schools, 37(5), 431-442.
- 42. Lieberman, A.F., Barnard, K.E., Wieder, S. (2004) Diagnosing infants, toddlers, and preschoolers: The Zero to Three diagnostic classification of early mental health disorders. In R. DelCarmen-Wiggins, & A. Carter (Eds.), Handbook of infant, toddler, and preschool mental health assessment (pp. 141-160). New York: Oxford University Press.
- 43. ZERO TO THREE: National Center for Infants, Toddlers, and Families. (2005). Diagnostic classification of mental health and developmental disorders of infancy and early childhood (DC:0-3R). Washington, DC: Author.
- 44. American Academy of Child & Adolescent Psychiatry (Task Force on Research Diagnostic Criteria: Infancy and Preschool) (2003). Research diagnostic criteria for infants and preschool children: The process and empirical support. Journal of the American Academy of Child & Adolescent Psychiatry, 42(12), 1504-1512.
- 45. Gleason, M. M., Egger, H. L., Emslie, G. J., Greenhill, L. L., Kowatch, R. A., Lieberman, A. F., Luby, J. L., Owens, J., Scahill, L. D., Scheeringa, M. S., Stafford, B., Wise, B., & Zeanah, C. H. (2007). Psychopharmacological treatment for very young children: Contexts and guidelines. Journal of the American Academy of Child and Adolescent Psychiatry, 46(12), 1532-1572.
- 46. Keenan, K., & Wakschlag, L. S. (2002). Can a valid diagnosis of disruptive behavior disorder be made in preschool children? American Journal of Psychiatry,
- 47. Luby, J. L. (Ed.) (2006). Handbook of preschool mental health: Development, disorders, and treatment. New York: Guilford Press.
- 48. Luby, J. L., Mrakotsky, C., Heffelfinger, A., Brown, K., Hessler, M., Spitznagel, E. (2003). Modification of DSM-IV criteria for depressed preschool children. American Journal of Psychiatry, 160(6), 1169-1172.
- 49. Lord, C., Risi, S., DiLavore, P. S., Schulman, C., Thurm, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. Archives of General Psychiatry, 63(6), 694-701.
- 50. Scheeringa, M., Peebles, C. D., Cook, C. A., & Zeanah, C. H. (2001). Toward establishing procedural, criterion, and discriminant validity for PTSD in early childhood. Journal of the American Academy of Child and Adolescent Psychiatry, 40(1), 52-60.
- 51. Scheeringa, M., Zeanah, C. H., Myers, L., & Putnam, F. (2005). Predictive validity in a prospective follow-up of PTSD in preschool children. Journal of the American Academy of Child and Adolescent Psychiatry, 44(9), 899-906.
- 52. Ghaziuddin, M., Ghaziuddin, N., & Greden, J. (2002). Depression in persons with autism: Implications

- for research and clinical care. Journal of Autism and Developmental Disorders, 32(4), 299-306.
- 53. Ghaziuddin, M., & Greden, J. (1998). Depression in children with autism/pervasive developmental disorders: A case-control family history study. Journal of Autism and Developmental Disorders, 28(2), 111-115.
- 54. Kim, J.A., Szatmari, P., Bryson, S.E., Streiner, D.L., & Wilson, F.J. (2000). The prevalence of anxiety and mood problems among children with autism and Asperger syndrome. Autism, 4(2), 117-132.
- 55. Keenan, K., Shaw, D., Delliquadri, E., Giovannelli, J., & Walsh, B. (1998). Evidence for the continuity of early problem behaviors: Application of a developmental model. Journal of Abnormal Child Psychology, 26(6), 441-452.
- 56. Shaw, D.S., Gilliom, M., Ingoldsby, E.M. & Nagin, D.S. (2003). Trajectories leading to school-age conduct problems. Developmental Psychology, 39(2), 189-200.
- 57. Suveg, C., Southam-Gerow, M.A., Goodman, K.L. & Kendall, P.C. (2007). The role of emotion theory and research in child therapy development. Clinical Psychology: Science and Practice, 14(4), 358-371.
- 58. Lieberman, A. F., Ippen, C. G., & Van Horn, P. (2006). Child-parent psychotherapy: 6-month follow-up of a randomized controlled trial. Journal of the American Academy of Child and Adolescent Psychiatry, 45(8), 913-918.
- 59. Peterson, C.A., Luze, G.J., Eshbaugh, E.M., Jeon, H. & Kantz, K.R. (2007). Enhancing parent-child interactions through home visiting: Promising practice or unfulfilled promise? Journal of Early Intervention, 29(2), 119-135.
- 60. Sameroff, A.J., McDonough, S.C., & Rosenblum, K.L. (Eds.) (2004). Treating parent-infant relationship problems: Strategies for intervention. New York: Guilford Press
- 61. Graham-Berman, S.A., & Hughes, H.M. (2003). Intervention for children exposed to interparental violence (IPV): Assessments of needs and research priorities. Clinical Child and Family Psychology Review, 6(3), 189-204.
- 62. Judge, S. (2004). The impact of early institutionalization on child and family outcomes. Adoption Quarterly, 7(3),
- 63. Lowenthal, B. (2001). Abuse and neglect: The educator's guide to the identification and prevention of child maltreatment. Baltimore, MD: Paul H. Brookes Publishing.
- 64. Watts-English, T., Fortson, B.L., Gibler, N., Hooper, S.R. & De Bellis, M.D. (2006). The psychobiology of maltreatment in childhood. Journal of Social Issues, 62(4),
- 65. Faja, S., & Dawson, G. (2006). Early intervention for autism. In J. Luby (Ed.) Handbook of preschool mental health: Development, disorders, and treatment. (pp. 388-416). New York: Guilford Press.
- 66. National Scientific Council on the Developing Child. (2004). Young children develop in an environment of relationships: Working Paper 1. http://www. developingchild.net.
- 67. Bredy, T.W., Humpartzoomian, R.A., Cain, D.P., & Meaney, M.J. (2003). Partial reversal of the effect of maternal care on cognitive function through environmental enrichment. Neuroscience, 118(2), 571-576.
- 68. Francis, D., Diorio, J., Plotsky, P.M., & Meaney, M.J. (2002). Environmental enrichment reverses the effects of maternal separation on stress reactivity. Journal of Neuroscience, 22(18), 7840-7843.

- 69. Sweeney, G.M. (2007). Why childhood attachment matters: Implications for personal happiness, families and public policy. In A.S. Loveless, & T.B. Holman (Eds.), The family in the new millennium: World voices supporting the "natural" clan: Vol. 1 (332-346). Westport, CT: Praeger Publishers.
- 70. Melton, G.B., Thompson, R.A., & Small, M.A. (2002). Toward a child-centered, neighborhood-based child protection system: A report of the Consortium on Children, Families, and the Law. Westport, CT: Praeger Publishers.
- 71. Visser, S. N., Lesesne, C. A., & Perou, R. (2007). National estimates and factors associated with medication treatment for childhood attention-deficit/hyperactivity disorder. Pediatrics, 119, S99-106.
- 72. Zito, J.M., Safer, D.J., dosReis, S., Gardner, J.F., Boles, M., & Lynch, F. (2000). Trends in the prescribing of psychotropic medications to preschoolers. Journal of the American Medical Association, 283(8), 1025-1030.
- 73. Carlezon, W.A. Jr., & Konradi, C. (2004). Understanding the neurobiological consequences of early exposure to psychotropic drugs: Linking behavior with molecules. Neuropharmacology, 47, 47-60.
- 74. Carlezon, W.A. Jr., Mague, S.D., & Andersen, S.L. (2003). Enduring behavioral effects of early exposure to methylphenidate in rats. Biological Psychiatry, 54(12), 1330-1337.
- 75. Bairy, K.L., Madhyastha, S., Ashok, K.P., Bairy, I., & Malini, S. (2007). Developmental and behavioral consequences of prenatal fluoxetine. Pharmacology, 79(1), 1-11.
- 76. Ashman, S., & Dawson, G. (2002). Maternal depression, infant psychobiological development, and risk for depression. In S.H. Goodman & I.H. Gotlib (Eds.), Children of depressed parents: Mechanisms of risk and implications for treatment. (pp. 37-58). Washington, DC: American Psychological Association.
- 77. Lesesne, C. A., Visser, S. N., & White, C. P. (2003). Attention-deficit/hyperactivity disorder in school-aged children: Association with maternal mental health and use of health care resources. *Pediatrics*, 111(5), 1232-1237.
- 78. Gilliam, W. (2005). Prekindergarteners left behind: Expulsion rates in state prekindergarten systems. New Haven, CT: Yale University Child Study Center.
- 79. Cooper, W.O., Arbogast, P.G., Ding, H., Hickson, G.B., Fuchs, D.C., & Ray, W.A. (2006). Trends in prescribing of antipsychotic medications for US children. Ambulatory Pediatrics, 6(2), 79-83.

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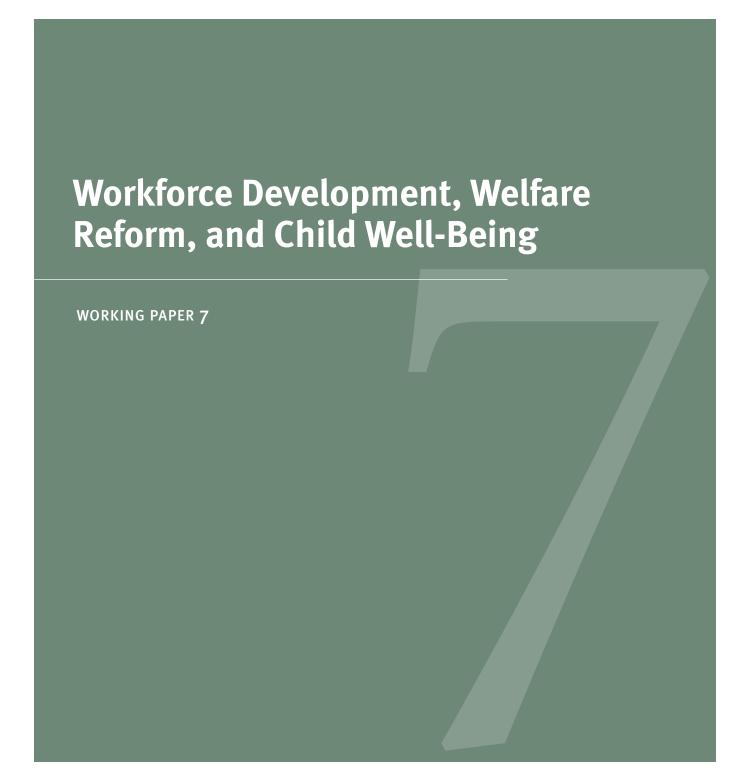
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This collaborative initiative fosters the analysis, synthesis, translation, and dissemination of findings from four decades of early childhood program evaluation studies to learn more about what interventions work best and for whom. Based at the Center on the Developing Child at Harvard University, the Forum involves researchers and data teams from Columbia University, Georgetown University, Harvard University, Johns Hopkins University, Northwestern University, the University of Nebraska, and the University of Wisconsin. Its work includes:

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- conducting rigorous analyses of the findings of well-designed studies of programs designed to improve outcomes for young children and/or provide effective support for their families;
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# The Issue

DEBATE REGARDING WORKFORCE DEVELOPMENT AND WELFARE REFORM OFTEN FOCUSES EXCLUSIVELY on the skills, employment, and economic self-sufficiency of parents. Consequently, little attention has been paid to whether these programs can improve the chances that children in these families will break the intergenerational cycle of poverty. Can policies promoting family self-sufficiency increase *both* parents' incomes *and* their children's school success? What kinds of work supports matter most for improving child well-being? How can these policies play a role in addressing our nation's economic problems?

Transitions from welfare to work may benefit children by placing them in stimulating child care settings, creating positive maternal role models, promoting maternal self-esteem and sense of control, introducing productive daily routines into family life, and eventually, fostering career advancement and higher earnings on the part of both parents and children. On the other hand, efforts to promote employment may overwhelm already stressed parents, force young children into substandard child care, reduce parents' abilities to monitor the behavior of their older children and, for those unable to sustain steady employment, deepen family poverty. Understanding what makes the difference between these positive and negative outcomes for children should be as much the focus of investigation—and public policy—as what improves adult workforce participation.

This Working Paper summarizes recent evidence from a series of evaluations of fam-

ily self-sufficiency programs. These studies show that policies can be successful in achieving both positive economic benefits for parents (increased employment, for example) and positive educational effects on their children. It need

# Can policies promoting family self-sufficiency increase *both* parents' incomes *and* their children's school success?

not be the case that increasing mothers' work effort, for example, simply increases their time away from the family and harms their children. Certain types of economic policies can in fact benefit children's school performance and social behavior.

# What Evaluation Research Tells Us

POLICIES THAT MAKE WORK PAY BY INCREASING both work and total family income boost younger children's school achievement while policies that simply mandate work do not improve child outcomes.

Recent evidence from five large-scale experiments testing 11 different approaches to work and welfare policies holds important lessons for how these policies can influence children's development. The policies tested two overall approaches to encouraging employment— "making work pay" by supplementing earnings and simply mandating employment within the

welfare system but without extra income supplements. Both kinds of policies increased employment and earnings among parents living in poverty. But only the "make work pay" approach increased family income (typically by between \$1,500 and \$2,000 a year). In the case of mandated programs, earnings increased but welfare payments fell by similar amounts, producing little to no net changes in total family income.

These experiments were conducted in several states and localities in the United States, <sup>3,4,5,6,7</sup> as well as two provinces in Canada. <sup>8</sup> In each setting, an approach to encouraging work was im-

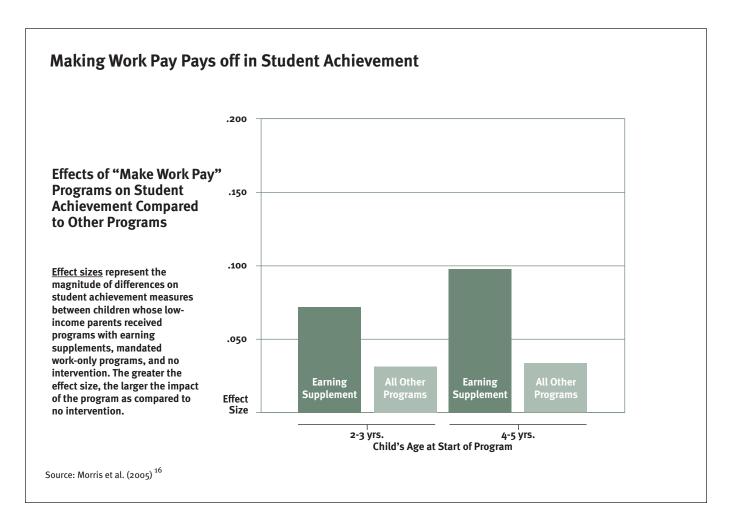
plemented, and effects on children were tested using <u>random-assignment methods</u> and assessment of children's school performance and social behaviors. These experiments enable us to compare policies that increase both work and family income to policies that simply increase work with respect to their effects on children. The results were remarkably consistent across the 11 programs — only the "make work pay" policies improved children's school performance and social behaviors, while policies that simply mandated work did not alter children's outcomes one way or the other.

How large were the effects of the "make work pay" policies? Positive impacts on school performance were small overall, but somewhat larger among younger children making the transition into primary school. For these children, programinduced improvements in school achievement were the equivalent of 1-2 points on an IQ type scale or about 10 points on an SAT-type test. These achievement gains may seem small, but if sustained they can translate into substantially higher

lifetime earnings (see box on page 3 for example of a program where effects were sustained).<sup>9</sup>

A menu of supports that includes child care will most benefit children. The evidence from these studies shows that there is no single best program model for the "make work pay" policies. Some successful programs were implemented in welfare systems, but others were implemented outside of such systems, generally through community-based workforce development organizations. The particular approach to be taken can be tailored to the individualized needs of a community, city, or state. Not surprisingly, the "make work pay" policies cost more than mandates, with additional annual costs ranging from \$2,000 to \$4,000 per family. The types of supports in these policies varied, including such programs as wage supplements, subsidies for health insurance and/or child care, and basic skills or job training for parents.

One "make work pay" program that provided wage supplements was particularly im-



pressive, demonstrating large positive effects on classroom achievement and behavior for boys and smaller effects for girls. This Milwaukeebased program—the New Hope project—provided not only child care subsidies, but also health insurance subsidies, case managers with low caseloads (i.e., 50 families), and temporary community service jobs that paid the minimum wage. 10 By requiring proof of at least 30 hours of weekly work, New Hope garnered the support of the Milwaukee business community (see box below).

Child care supports appear to be a particularly important "effectiveness factor" for such programs, as research shows that quality of care can matter for children's early school success. Those programs that included child care subsidies increased the use of center-based care, the type of care that most consistently improves young children's early school performance.

Programs that focus on basic skills and job training can help children if they are designed to

encourage and support active participation by **parents.** Three of the experimental programs mandated participation in basic skills or vocational training, depending on the needs of the mother.11 Since maternal education is one of the most reliable predictors of children's

Only the "make work pay" policies improved children's school performance and social behaviors, while policies that simply mandated work did not alter children's outcomes one way or the other.

achievement, it was expected that the boost in education from being assigned to these training programs might boost child achievement.

Participation in the program was highly variable. Mothers who participated in education and training activities the most (over an average of 8 months) provided significantly better home learning environments for their children. Perhaps as a

#### **NEW HOPE:**

#### A Cafeteria of Benefits for Families Leads to Classroom Success for Children

In the New Hope program, participants committed themselves to full-time work and New Hope in turn promised a package of work supports to ensure that they would not be poor and that they would be able to afford health insurance and licensed child care. New Hope was open to all low-income adults (family income below 150 percent of the poverty line), regardless of family circumstances.

Specifically, when New Hope participants provided pay stubs or other proof of full-time work (30+ hours per week), they became eligible for three sliding-scale benefits:

- 1) an earnings supplement that raised their income above the poverty line;
- 2) subsidized child care; and
- 3) subsidized health insurance.

Individuals unable to find full-time work would be eligible for:

4) a temporary community-service job.

Taken together, New Hope offered a "cafeteria" of benefits from which participants could pick and choose—a feature that allowed families with diverse needs and circumstances to tailor the program to their own situation. New Hope services were made available in a single office to facilitate the time-consuming and confusing process of dealing with multiple agencies. Although many participants in the original New Hope program were only interested in the program benefits themselves, all had access to help from a caseworker who provided information about jobs, educational opportunities, child care, and other community resources in an atmosphere of respect.

Because its participants were selected by a lottery, New Hope's evaluation resembled a clinical trial of a new drug, with program participants being compared with otherwise similar women and men who were not chosen in the lottery. The results showed that New Hope increased work and reduced poverty. Teachers reported that children in New Hope families performed better in school, were more cooperative and independent, and had fewer behavior problems and loftier schooling expectations than children in the comparison families. Given that boys, particularly black and Hispanic boys, have a higher risk of school failure and behavior problems than girls, it is noteworthy that New Hope was especially successful in improving their school performance and behavior. result, their children's school readiness increased and academic problems (e.g., special education placement or grade retention) declined.<sup>12</sup>

Overall, however, only about half of the mothers assigned to education or training participated at all, and on average the time spent training was sufficiently small to produce no discernable effect on children's school readiness. So while education and training programs for mothers have the *potential* for boosting children's achievement, it has proven difficult to design a program that delivers the required training intensity.

Research on these programs did show that if the program approach (education-first vs. work-first) matched the parents' expressed goals for these activities, effects on children's early school achievement were more positive. <sup>13</sup> This suggests that parents' goals for education and work should be considered in structuring self-sufficiency activities in policies and programs for low income, working families.

Adolescents and children of the most disadvantaged parents need different supports. For adolescents, a different story emerged than that for very young children. Both types of policies produced small negative effects on parents' perceptions of their adolescents' school performance. Some of the negative impacts on adolescents may have been caused by the fact that the increased employment reduced the amount of parental interaction and supervision. It also appeared that some adolescents developed school problems because they were spending more time caring for their younger siblings.

In addition, these programs were most effective for those families at moderate levels of disadvantage.<sup>15</sup> Children of those parents with the most severe barriers to work (such as low levels of education and prior work experience) did not benefit from even the "make work pay" programs. This suggests that the most disadvantaged families in poverty require more intensive services than most work and income support programs provide.

#### **Sustained Benefits of Making Work Pay Boys in New Hope Have Higher Boys in New Hope Rate Higher in School Achievement Over Time Expectations of Completing College** 5 5 **New Hope participants** The effects of New Hope were most pronounced on boys. These graphs show the impact of New Hope **New Hope participants** on boys two and five No intervention years after the program began. Achievement was 3 3 measured by teachers' response on a five-point No intervention achievement-reporting scale, and college expectations by student responses on a five-point scale. Note: Effect sizes, which represent the magnitude of differences between boys participating in the New Hone program and a control group receiving no intervention, are moderate-roughly .3-.4. Program 5 yrs. Program 5 yrs. Program 2 Vrs. Program 2 yrs. **Begins Ends Begins Ends** Source: Huston, et.al. (2003). <sup>7</sup>

# **Implications for Policy and Programs**

CHILD WELL-BEING CAN BE AFFECTED BY BOTH welfare-to-work policies as well as policies that provide supports for already-working families. We discuss each of these kinds of policies and their impacts below.

Welfare policy: Focus on "making work pay," not just making parents work. State policymakers face choices when deciding how best to respond to the changing federal policy demands under Temporary Assistance to Needy Families (TANF). The least expensive option in the short run is to focus on increasing parental work through mandatory employment programs. A more effective option in the long run is to attempt to ensure that "work pays" with earnings supplements, such as state Earned Income Tax Credits and work-conditioned benefits to children, such as child-care subsidies.

Offer a range of work supports. Work-support packages are diverse and can include earnings supplements, child care assistance, health insurance, and even temporary community service jobs. Potential ways to incorporate work supports into federal policies include providing additional funds for the child care block grant, expanding health insurance coverage and participation for children, expanding participation in the Food Stamp program, and expanding the Earned Income Tax Credit (EITC). Evaluation evidence does not point to a single best way of using these kinds of supports to boost child well-being, yet data show successes for children with both earnings supplements alone as well as with the provision of a more comprehensive package of benefits.

Consider implementing proven after-school programs for teens in addition to employment and welfare policies. States should be aware of the likely differential consequences of their policies among children of different ages. Evidence suggests that the school achievement of adolescents is most likely to suffer somewhat when parents are required to work. In response, states may want to consider proven after-school and communitybased programs for adolescents to help support working parents while also supporting these

parents' efforts to keep their children focused on school achievement and positive behavior.

Design training and basic skills programs to encourage and support mothers to actively participate in them. A number of experimental programs have sought to boost the basic or vocational skills of mothers, hoping to enhance their employability and perhaps improve the learning opportunities at home for their children. These programs rarely improved schooling outcomes for children, but for an important

Work-support packages are diverse and can include earnings supplements, child care assistance, health insurance, and even temporary community service jobs.

reason — mothers rarely spend much time in them. Education programs for both children and adults succeed only when instructional time is substantial or when parents express high levels of motivation to pursue their own education. In the case of the training programs, it appeared that the more time mothers spent in their job training classrooms, the more their children benefited.

# References

- Morris, P., Huston, A., Duncan, G., Crosby, D., & Bos, H. (2001). How welfare and work policies affect children: A synthesis of research. New York: MDRC
- Gennetian, L. A., Duncan, G., Knox, V., Clark-Kauffman, B., & Vargas, W. (2002). How welfare and work policies for parents affect adolescents: A synthesis of research. New York: MDRC.
- Bloom, D., Kemple, J.J., Morris, P., Scrivener, S., Verma, N., & Hendra, R. (2000). The family transition program: Final report on Florida's initial time-limited welfare program. New York: MDRC.
- Bloom, D., Scrivener, S., Michalopoulos, C., Morris, P., Hendra, R., Adams-Ciardullo, D., & Walter, J. (2002). Jobs first: Final report on Connecticut's welfare reform initiative. New York: MDRC.
- Gennetian, L. & Miller, C. (2000). Reforming welfare and rewarding work: Final report on the Minnesota family investment program. Volume 2: Effects on children. New York: Manpower Demonstration Research Corporation.
- Hamilton, G., Freedman, S., Gennetian, L.A., Michalopoulos, C., Walter, J., Adams-Ciardullo, D., Gassman-Pines, A., McGroder, S., Zaslow, M., Brooks, J., & Ahluwalia, S. (2001). How effective are different welfare-to-work approaches? Five-year adult and child impacts for eleven programs. Washington, DC: U.S. Department of Health and Human Services, Office of the Assistant Secretary for Planning and Evaluation and Administration for Children and Families, and U.S. Department of Education.
- Huston, A. C., Miller, C., Richburg-Hayes, L., Duncan, G. J., Eldred, C. A., Weisner, T. S., Lowe, E., McLoyd, V. C., Crosby, D. A., Ripke, M. N., & Redcross, C. (2003). The new hope project effects on families and children after five years. New York: MDRC.

- Morris, P. & Michalopoulos, C. (in press). Findings from the self sufficiency project: Effects on children and adolescents of a program that increased employment and income. Applied Developmental Psychology.
- Krueger, A. & Whitmore, D. (2001). The effect of attending a small class in the early grades on collegetest taking and middle school test results: Evidence from project STAR. Economic Journal, Vol. 11, No. 1,
- Duncan, G., Huston, A., & Weisner, T. (2007). Higher ground: New hope for the working poor and their children. New York: Russell Sage Foundation.
- Hamilton et al. (2001).
- Magnuson, K. (2003). The effect of increases in welfare mothers' education on their young children's academic and behavioral outcomes. Madison, WI: University of Wisconsin, Institute for Poverty Research.
- Gassman-Pines, A., Godfrey, E.B., & Yoshikawa, H. (2008). Parental goals moderate the effects of welfare policies on children: A person-environment fit approach. Manuscript under review.
- Gennetian et al. (2002).
- Yoshikawa, H., Magnuson, K., Bos, J. & Hsueh, J. (2003). Effects of earnings-supplement policies on adult economic and middle-childhood outcomes differ for the "hardest to employ." Child Development, 74 (5), 1500-1521.
- Morris, P., Duncan, G., & Clark-Kauffman, E. (2005). Child well-being in an era of welfare reform: The sensitivity of transitions in development to policy change. Developmental Psychology, Vol. 41, No. 6, 919-932.

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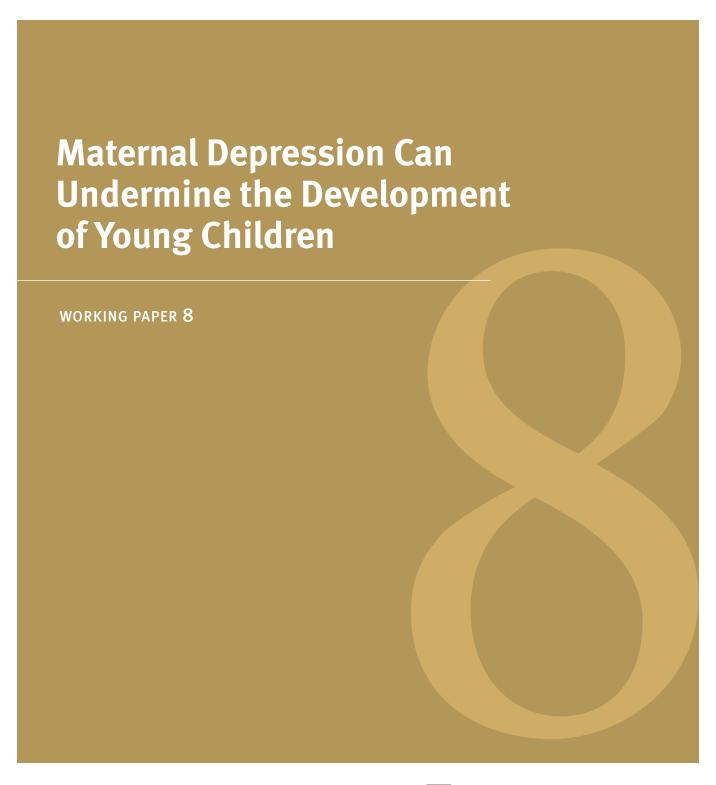
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#### NATIONAL FORUM ON EARLY CHILDHOOD PROGRAM EVALUATION

Center on the Developing Child HARVARD UNIVERSITY



# NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD NATIONAL FORUM ON EARLY CHILDHOOD PROGRAM EVALUATION



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# The Issue

serious depression in parents and caregivers can affect far more than the adults who are ill. It also influences the well-being of the children in their care. Because chronic and severe maternal depression has potentially far-reaching harmful effects on families and children, its wide-spread occurrence can undermine the future prosperity and well-being of society as a whole. When children grow up in an environment of mental illness, the development of their brains may be seriously weakened, with implications for their ability to learn as well as for their own later physical and mental health. When interventions are not available to ensure mothers' well-being and children's healthy development, the missed opportunities can be substantial.

Healthy development depends on the interactive influences of genes and experiences, which shape the architecture of the developing brain. The active ingredient of those experiences can be described as mutual responsiveness or the "serve and return" of young children's interactions with adult caregivers.1 For example, when an infant babbles and an adult responds appropriately with attention, gestures, or speech, this builds and strengthens connections in the child's brain that support the development of communication and social skills. When caregivers are sensitive and responsive to a young child's signals, they provide an environment rich in serve and return experiences, like a good game of tennis or Ping-Pong. However, if depression interferes with the caregiver's ability to regularly provide such experiences, these connections in the child's brain may not form as they should. The difference between a child who grows up in a responsive environment and one who does not can be the difference between the development of strong or weak brain architecture, which serves as a foundation for the learning, behavior, and health that follow.

Maternal depression is particularly worrisome because of its prevalence. An estimated 10 to 20 percent of mothers will be depressed at some time during their lives. <sup>2,3,4,5</sup> About one in eleven infants will experience their mothers' major depression in their first year of life, and the rates are even higher for mothers with previous histories of depression or those

experiencing other stressors, such as financial hardship or social isolation.<sup>6,7</sup> Depression and depressive symptoms are particularly common in disadvantaged populations. Recent data indicate that, in households below the federal poverty threshold, one in four mothers of infants is experiencing moderate-to-severe levels

Because chronic and severe maternal depression has potentially far-reaching harmful effects on families and children, its widespread occurrence can undermine the future prosperity and well-being of society as a whole.

of depressive symptoms. (See graph, page 2.)

Although it is all the same underlying disorder, mothers' *experiences* of depression may differ in timing, severity, and duration.<sup>8,9</sup> For a substantial proportion of mothers, depression comes in spells that may last just a few months; but, for others, depression is more chronic.<sup>9,10,11</sup> Some mothers may experience depression primarily during their children's infant and toddler years; others endure depression that is prolonged or recurs over many years of a child's life.<sup>10,12,13</sup> Although the greatest cause for concern arises when depression begins early and is long-lasting and severe, poor developmental outcomes have

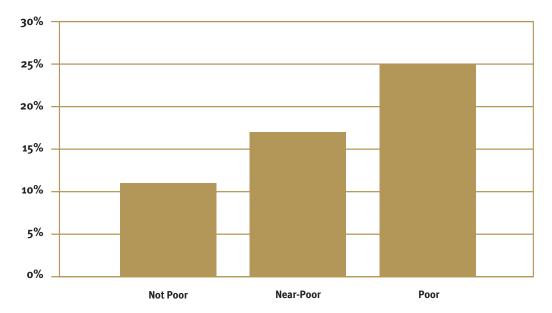
Despite the frequency of depression among new mothers, large numbers of affected individuals may not be identified as having a treatable condition, and only 15 percent obtain professional care.

been documented for children of mothers with declining or low levels of depression, as well.<sup>12,14</sup>

Despite the frequency of depression among new mothers, large numbers of affected individuals may not be identified as having a treatable condition, and only 15 percent obtain professional care.<sup>15</sup> Just as it is essential to treat children's emotional and behavioral problems within the context of their families, <sup>16</sup> it is equally essential for treatments and programs aimed at improving maternal depression and depressive symptoms to consider, treat, and measure their impact on the children. Although depression in fathers or other important caregivers (such as grandparents) also deserves further attention, the effects on children's development are rarely studied, and the research is far less conclusive. <sup>4</sup>

It is not normal for women to suffer major depression after having a baby. Major depression is very different from (but sometimes confused with) the emotional swings experienced by many mothers shortly after child-birth. <sup>17</sup> Characterized by a low mood and loss of interest in usually enjoyable activities, depressive symptoms include difficulty sleeping and

#### Maternal Depression Affects Children in Low-Income Families Disproportionately



Percent of mothers with a 9-month-old infant who are moderately or severely depressed

Source: Calculations based on analyses of the Early Childhood Longitudinal Study, Birth Cohort 9-month restricted-use data (NCES 2006-044) by Danielle Crosby, PhD, University of North Carolina Greensboro. Maternal Depression is measured by the 12-item version of the Center for Epidemiological Studies' Depression Scale (CES-D). Elevated symptoms of moderate to severe depression are identified by scores of 10 points or higher on a scale that ranges from 0-36. Analytic weights (W1RO) were applied to ensure data were nationally representative of mothers with 9-month-old infants born in 2001. Poor refers to family income less than or equal to 100% Federal Poverty Threshold (FPT). Near poor refers to family income greater than 200% of FPT but less than 200% of FPT. Not poor refers to income greater than 200% of FPT.

concentrating, loss of appetite, feelings of worthlessness or guilt, and low energy. In the face of major clinical depression, the drive, energy, and enjoyment needed to build and maintain positive family relationships recedes. Especially when combined with other, related adversities, deep depression is debilitating, making it difficult for mothers to effectively carry out requisite caregiving tasks and responsibilities and to build and maintain nurturing relationships with their children. This may explain why, when raised by a chronically depressed mother, children perform lower, on average, on cognitive, emotional, and behavioral assessments than children of nondepressed caregivers, and they are at risk for later mental health problems, social adjustment difficulties, and difficulties in school.<sup>18</sup> Such patterns may also forecast difficulties in adult life across a variety of important domains, including employment and health.

As the magnitude and societal consequences of this problem have become better understood, increasing numbers of clinicians and policymakers have begun to realize that it is unacceptable to ignore what science tells us and have made the prevention and treatment of maternal depression an important goal. In

Children who experience maternal depression early in life may suffer lasting effects on their brain architecture and persistent disruptions of their stress response systems.

order to maximize the impact of such investments on the well-being of children as well as mothers, it's important for policymakers to start from a common understanding of what we know-and what we don't know-about the effects of maternal depression on children as well as the effectiveness of programs designed to treat or prevent this serious condition.

# What Neuroscience and Developmental Research Tell Us

Chronic depression can manifest itself in two types of problematic parenting patterns that disrupt the "serve and return" interaction that is essential for healthy brain development: hostile or intrusive, and disengaged or withdrawn. 19 When parents are hostile and/or intrusive, it is as if the parent is "serving" the ball in ways that make it difficult for the child to "return." Conversely, if a parent is withdrawn or disengaged, the child may serve the ball, but the parent doesn't return it. In both cases, depressed mothers are less likely to respond to their infants' cues (i.e., vocalizations and actions) or to engage with their infants and young children in positive, harmonious interactions.<sup>20,21</sup> When caregivers are hostile or withdrawn for prolonged periods of time, the game of serve and return falls apart, and the architecture of the developing brain may be affected adversely. Such patterns are particularly worrisome because, once negative parent-child interactions are established, they may persist even after a mother's depression has improved

and may make the child more likely to have negative interactions with other important adults, as well.22,23 When infants and young children interact with a hostile, irritable caregiver, this creates feelings of fear and anxiety in the child, which may result in the increased production of potentially harmful stress chemicals.24 Such a recurring physiological reaction can affect brain development, interfere with young children's ability to learn, and increase the risk of emotional disorders.25

Children who experience maternal depression early in life may experience lasting effects on their brain architecture and persistent disruptions of their stress response systems. Studies of children of depressed mothers show patterns of brain activity (as observed on an electroencephalogram, or EEG) that are similar to those found in adults with depression.<sup>26</sup> These patterns are more pervasive when the mother is both depressed and withdrawn from her infant<sup>27</sup>

and when deep depression occurs during the child's second and third year, the time at which the brain systems that generate the depression-associated pattern of brain waves are developing rapidly.<sup>24</sup> Living with a depressed mother may also shape the development of a child's stress biology.<sup>28</sup> Indeed, there is increasing evidence that effects on stress response systems are one mechanism linking maternal depression to the child's

research has found that prenatal depression can be linked to the silencing of a gene that controls the over-production of stress chemicals.<sup>35</sup> Thus, by the time of birth, the infant of a seriously depressed mother may have sustained effects on his or her stress response and immune systems that make the child even more vulnerable than average to irritable, intrusive, or withdrawn maternal care.

# Depressed women produce higher levels of stress chemicals during pregnancy, which reduce fetal growth and are associated with an increased risk for premature labor.

own risk of developing depression and other emotional disorders.<sup>29,30</sup> When mothers are depressed, both early in a child's life and later, their children tend to produce higher and more fluctuating levels of stress chemicals such as cortisol.<sup>31,32</sup> Chronic maternal depression over many years of a child's life also predicts cardiovascular patterns suggestive of emergent hypertension in childhood and abnormal stress chemical patterns in response to laboratory testing.<sup>29</sup>

Maternal depression may begin to affect brain development in the fetus before birth. Depressed women produce higher levels of stress chemicals during pregnancy, which reduce fetal growth and are associated with an increased risk for premature labor.<sup>33</sup> Depressive symptoms in an expectant mother also have been shown to be associated with altered immune functioning in her baby after birth.<sup>34</sup> Even more striking, recent

Depression often occurs in the context of other family adversities, which makes it challenging to treat successfully. When mothers have good social supports, adequate income, and environments free of stress and conflict, they are better able to provide the developmentally appropriate interactions that their children need. However, depression often coincides with a constellation of other adversities that may further undermine child development. For example, mothers experiencing depression are often also young, socially isolated, economically or educationally disadvantaged, and burdened by more family conflict and stressful life events than mothers who are not depressed. 11,36 Mothers who experience deep or chronic depression are also more likely to have experienced intimate partner violence,<sup>37</sup> to be in poorer health, and to have problems with anxiety or substance abuse.4 Indeed, evidence suggests that 75 percent of adults diagnosed with major depression have at least one other mental health diagnosis.38 When maternal depression co-occurs with other serious adversities, not only are standard treatments less likely to be successful in reducing depressive symptoms, but the children are at even greater risk for poor outcomes, as these related risk factors also reduce the likelihood that they will experience environments that foster healthy development.4,39

# What Program Evaluation Research Tells Us

GIVEN THE POTENTIAL NEGATIVE CONSEQUENCES of depression for both mothers and their children, a variety of interventions has been designed to prevent and treat it as well as to buffer children from its harmful effects. By intervening early, before these effects can accrue, we increase

the likelihood that children of depressed mothers will grow into healthy, capable, fully contributing members of society.

Although a few studies address the more general treatment of maternal depression, much of the research on prevention is focused on the

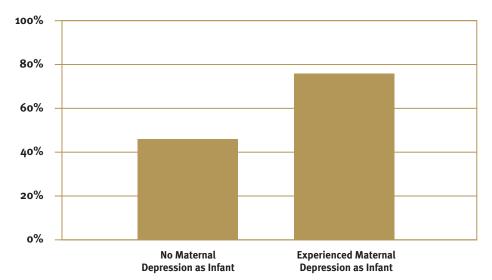
specific condition of postpartum depression (PPD). Interventions that have been employed differ as to whether they take a biological approach, such as the prescription of antidepressant medications, or a psychosocial approach grounded in therapeutic and psycho-educational strategies. In cases of PPD, the use of medication for preventive purposes has been limited because of concerns about the potentially harmful effects of antidepressants that can pass directly into the fetus through the placenta or into the infant through breast milk.40 Alternatively, the range of psychosocial interventions that have been employed includes strategies directed either solely to mothers or to the motherchild dyad.

Intensive intervention efforts that focus specifically on mother-child interactions have shown promising results in several recent studies. One program of weekly toddler-parent

By intervening early, we increase the likelihood that children of depressed mothers will grow into healthy, capable, fully contributing members of society.

psychotherapy that lasted over a year produced improved cognitive development among children of depressed mothers.<sup>46</sup> Another successful intervention targeted young, economically disadvantaged mothers of infants and provided a comprehensive set of daily supports, including education opportunities for the mothers, massage therapy for both mothers and infants, and mother-infant interaction coaching.<sup>47</sup> Several recent studies have shown improvements in important dimensions of mother-child

# **Exposure to Maternal Depression in Infancy Causes Stress Hormone** Levels to Become More Extreme in Adolescence



Percentage of Adolescents with Extremely High Cortisol Levels

(Above 90th percentile for gender) on 1 or more days out of 10 measured

Source: Halligan, Herbert, Goodyer, and Murray (2004).

interaction. 40,48 Collectively, these examples demonstrate that intensive, well-designed interventions for depressed mothers and their children can improve both parenting behaviors in the mothers and developmental outcomes in the children. What these studies cannot tease

also provide suggestive evidence that programs that explicitly promote positive parenting practices among depressed parents may have benefits for children and families.

apart, however, is whether the key ingredient in the program's success was its focus on the mother-child dyad or the intensity of the treatment. Nevertheless, interventions with families and older children also provide suggestive evidence that programs that explicitly promote positive parenting practices among depressed parents may have benefits for children and families. 4,49 Yet, the high cost of intensive interventions and the fact that most studies have been conducted on relatively small samples present serious challenges to the task of replicating successful models and taking them to scale.

Research indicates that various combinations of psychotherapy and educational treatments that are focused exclusively on adults can be effective in reducing depressive symptoms in mothers but appear to have limited impacts on the development of their young children. 41,42,43,44 A few good studies of therapeutic interventions

have demonstrated reductions in maternal depression after treatment, but no differences were documented in long-term child outcomes, such as cognitive functioning and behavior. These findings have led several researchers to argue that therapies should not only treat the mother but should also focus on the mother-child relationship.<sup>45</sup>

Successful efforts to prevent maternal depression before it develops have thus far been more elusive than effective treatments. Several models of educational and psychological interventions to prevent postpartum depression have shown promise.41,50,51 These programs are diverse in terms of when services are initiated, how and by whom services are delivered, and the likelihood of depression in the population they serve. Most of these prevention programs, however, are short-term, often delivered through a small number of individual or group educational sessions or psychotherapy, midwife care, or home visits. The documented success of a program in which poor women participated in four prenatal sessions of group psychotherapy suggests that preventing PPD may be possible.<sup>52</sup> Yet, the small, voluntary, and select nature of the sample, as well as the lack of child outcome measures, indicate that more research is necessary. Biological approaches to preventing PPD also have been studied, but successful results are rare, 41,53 and, as noted previously, biological prevention approaches are often not well received by mothers because of concerns about the pass-through effects of medication on fetuses or infants. The extent to which maternal depression often coexists with other mental health problems (e.g., anxiety disorders or substance abuse) further complicates the challenge of designing effective prevention programs.

# **Correcting Common Misconceptions**

Contrary to popular belief, professional treatment is needed to help mothers overcome major depression. Although many mothers experience emotional adjustments and mood swings in the immediate period following childbirth—sometimes known as the "baby blues"—this is very

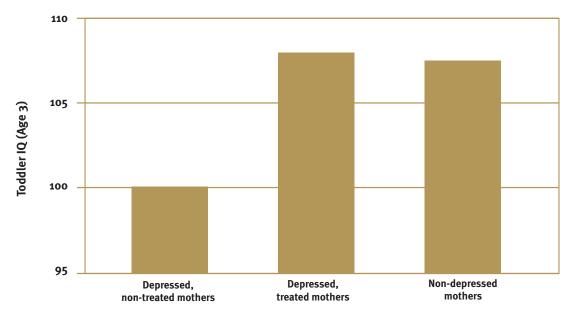
different from major depression, which is much more severe and can be long-lasting.<sup>17</sup> Major depression should be understood as a serious medical condition that affects brain functioning and typically limits one's ability to carry out everyday activities. Mothers of infants are more likely than other women to experience such depression, particularly as they experience high caregiving demands, yet they are less likely to get professional help.15 This is most unfortunate, because there is evidence that a range of treatment approaches may reduce depressive symptoms among these mothers.54

It is not commonly understood that even very young children are likely to be affected by their mother's depression and these effects may be lasting. Adverse effects may even begin during pregnancy. As noted earlier, maternal depression in the prenatal period is linked to alterations in the stress response and immune systems of the fetus, which can increase the chance that an infant will be more vulnerable to irritable, intrusive, or withdrawn maternal care than the average baby.34,35 Moreover, ongoing depression

after childbirth is linked to patterns of parenting that may disrupt the normal "serve and return" interactions between an infant and mother, thus potentially harming the child's developing brain architecture and emerging skills.19 Finally, hostile or withdrawn parenting has been linked to patterns of child brain activity associated with anxious and withdrawn emotions, which may persist over time.24

Contrary to what is frequently assumed, reducing mothers' depressive symptoms alone does not necessarily lead to improvements in parenting and children's development. This erroneous assumption has been called into question by treatments that have improved mothers' depressive symptoms but have not had measurable effects on children's development. 42,43,44 Even when successful, short-term treatments that focus only





Mothers with a major depressive disorder were randomly selected to participate in Toddler-Parent Psychotherapy as a preventive intervention for their children, age 20 months at entry to program. Children's scores on Bayley Mental Developmenet index did not differ at age of entry, but significant differences appeared in IQ tests given at age 3.

Source: Cicchetti, Rogosch, and Toth (2000).

on reducing mothers' depression may miss the opportunity to also improve their parenting skills and their view of their children.<sup>43</sup> Limited but promising evidence suggests that treatments designed to improve child well-being must

attend both to relieving mothers' depression and to focusing on parenting behavior and interactions with the child as central dimensions of the intervention.<sup>9</sup>

# The Science-Policy Gap

Postpartum depression has several characteristics that make it a particularly compelling target for preventive intervention, yet little has been done in the U.S. to determine which interventions work. These characteristics include a clear time of onset (childbirth), a distinct risk period (up to six months postdelivery), and an identifiable population of at-risk women (expectant mothers).<sup>51</sup> Despite this striking opportunity, most studies of programs designed to prevent PPD have been conducted outside of the United States, where differences in health service delivery systems may make their findings less applicable to the U.S. context. In view of the fact that maternal depression can set in motion detrimental patterns of parenting and developmental processes that may be difficult to change as time passes, the limited availability of effective prevention programs in the United States represents an important missed opportunity to improve children's development.

Enhanced treatment programs for mental health problems in parents need to address adult behavior toward young children as well as the programs' impacts on children's developmental outcomes. This presents a major challenge, as very few studies to date have measured the impacts of interventions for maternal depression on either mother-child interaction or child wellbeing. Evidence that depression interferes with a mother's ability to engage in and benefit from a variety of services adds to that challenge, particularly for disadvantaged populations.4 Several settings have been identified as promising vehicles through which prevention, screening, and treatment services for maternal depression might be embedded, including primary health care, home visiting, and early care and education.<sup>4,55</sup> Each of these types of programs provides an underutilized opportunity to identify and engage depressed mothers, yet they all face distinctive challenges in developing and implementing effective services.4,56

# **Implications for Policy and Programs**

Maternal depression matters for children, families, and society. Maternal depression affects a large number of families and can have potentially far-reaching, adverse effects on parenting and the development of children. These consequences have implications for society as a whole, as children who are affected adversely become the next generation of parents and workers. Untreated maternal depression may lead to more hostile or withdrawn parenting, which can, in turn, have harmful effects on young children's developing brains, leaving them at higher risk for later cognitive and socio-emotional problems. Insensitive, hostile, or withdrawn parenting is associated with the development of

patterns of brain activity associated with anxious, withdrawn emotions in children and adults. Depressed mothers engage in less stimulation of their children, potentially reducing the strength of brain circuits involved in learning and memory. It is therefore important for the well-being of society to find effective ways to prevent and treat this disorder.

Evidence suggests that intensive therapies that focus on both mothers and their young children together can improve child outcomes. Not only are access and use of mental health services by mothers important, but equally important is the need for policymakers and clinicians to work

together to establish and support a model of care that simultaneously addresses mothers' own mental health needs as well as their caregiving roles and their children's healthy development. Because healthy brain architecture is built by positive interactions with responsive caregivers over time, short-term therapies of low intensity that focus solely on mothers may be effective at reducing their depressive symptoms, but they are unlikely to improve child outcomes. In view of the magnitude of the problem, creative policymaking would be well served by support for promising pilot projects that focus on mothers' interactions with their infants and are linked to strong evaluation designs.

Innovation, evaluation, and continuous improvement are needed to better understand what works for both children and their mothers and to bring such interventions to scale. Findings from program evaluations indicate that the challenge of treating maternal depression is not simply a matter of increased funding. Because there is still much to be learned, two kinds of investments are important for policymakers to consider. The first would focus on replication and expansion of interventions that have been evaluated and shown to have positive effects on young children. The second and equally important kind of investment would provide support for innovative program models that focus on the needs of mothers and their children, guided by advances in neuroscience and developmental research and committed to rigorous evaluations. In short, the prevalence of maternal depression, the growing evidence of its potential adverse impacts on young children, and the lack of interventions that focus on the well-being of the young children of affected mothers all call for increased investment in the design, testing, and continuous improvement of more effective two-generation interventions.

The development and testing of more successful models for prevention of maternal depression, particularly for women who are at increased risk for the disorder, should be an important policy **priority.** Evaluations of prevention programs for maternal depression thus far have been disappointing. This does not mean that prevention cannot work, but it does indicate that we still

The development and testing of more successful models for prevention of maternal depression, particularly for women who are at increased risk for the disorder, should be an important policy priority.

have much to learn. The magnitude of risk facing affected mothers and young children from all social classes, particularly those who have low incomes and limited education, underscores the need for policymakers, neuroscientists, doctors, program developers, and evaluation specialists to work together in the search for new and more effective prevention strategies.

IN SUMMARY, THE PREVALENCE AND MULTIPLE costs of maternal depression and the growing development of conceptual and practical approaches for protecting children whose mothers have the disorder all provide a compelling rationale for testing and evaluating promising practices and new intervention strategies. The continuing failure to address the consequences of depression for large numbers of vulnerable, young children presents a missed opportunity to help families and children in a way that could have far-reaching implications for the productivity, health, and well-being of the next generation.

# References

- National Scientific Council on the Developing Child. (2007). The science of early childhood development. Retrieved November 1, 2009, from www.developingchild. harvard.edu.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., Rush, A. J., Walters, E. E., & Wang, P. S. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *Journal of the American Medical Association*, 289, 3095–3105.
- 3. Mian, A. I. (2005). Depression in pregnancy and the postpartum period: Balancing adverse effects of untreated illness with treatment risks. *Journal of Psychiatric Practice*, 11(6), 389-396.
- 4. National Research Council and Institute of Medicine (2009). Depression in parents, parenting, and children: Opportunities to improve identification, treatment, and prevention. Committee on Depression, Parenting Practices and the Healthy Development of Children, Board on Children, Youth and Families, Division on Behavioral and Social Sciences and Education. Washington, DC: The National Academies Press.
- 5. O'Hara M. W., & Swain, A. M. (1996). Rates and risk of postpartum depression a meta-analysis. *International Review of Psychiatry*, 8, 37-54.
- Miller, L. J. (2002). Postpartum depression. Journal of the American Medical Association, 287, 762-765.
- 7. Administration for Children and Families. (2002). Making a difference in the lives of children and families: The impacts of Early Head Start programs on infants and toddlers and their families. Washington, DC: U.S. Department of Health and Human Services.
- 8. Cooper, C., Jones, L., Dunn, E., Forty, L., Haque, S., Oyebode, F., Craddock, N., & Jones, I. (2007). Clinical presentation of post-natal and non-postnatal depressive episodes. *Psychological Medicine*, *37*, 1273-1280.
- Whiffen, V. E., & Gotlib, I. H. (1993). Comparison of postpartum and nonpostpartum depression: Clinical presentation, psychiatric history, and psychosocial functioning. *Journal of Consulting and Clinical Psychology*, 61, 485-494.
- Campbell, S., Cohn, J. F., & Meyers, T. (1995). Depression in first-time mothers: Mother-infant interaction and depression chronicity. *Developmental Psychology*, 31, 349-357.
- Horowitz, S. M., Briggs-Gowan, M. J., Storfer-Isser, A., & Carter, A. (2007). Prevalence, correlates and persistence of maternal depression. *Journal of Women's Health*, 16, 678-691.
- Ashman, S. B., Dawson, G., & Panatogiotides, H. (2008). Trajectories of maternal depression over seven years: Relations with child psychophysiology and behavior and role of contextual risks. *Development and Psychopathol*ogy, 20, 55-77.
- 13. Phillips, J., Sharpe, L., Matthey, S., & Charles, M. (in press). Subtypes of postnatal depression? A comparison of women with recurrent and de novo postnatal depression. *Journal of Affective Disorders*.
- 14. Campbell, S., Morgan-Lopez, A. A., Cox, M., McLoyd, V. & NICHD ECCRN (2009). A latent class analysis of maternal depressive symptoms over 12 years and offspring adjustment in adolescence. *Journal of Abnormal Psychology*, 3, 479-493.

- Vesga-Lopez, O., Blanco, C., Keyes, K., Olfson, M., Grant, B., & Hasin, D. (2008). Psychiatric disorders in pregnant and postpartum women in the United States. Archives of General Psychiatry, 65, 805-815.
- 16. National Scientific Council on the Developing Child. (2008). Mental health problems in early childhood can impair learning and behavior for life. Working Paper No. 6. Retrieved November 1, 2009, from www.developingchild. harvard.edu.
- Sohr-Preston, S.L., & Scaramella, L. V. (2006). Implications of maternal depressive symptoms for early cognitive language development. Clinical Child and Family Psychology Review, 9, 65-83.
- Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review*, 106, 458-490.
- Lovejoy, M. C., Graczyk, P. A., O'Hare, E., & Neuman, G. (2000). Maternal depression and parenting behavior: A meta-analytic review. *Clinical Psychology Review*, 20(5), 561-592.
- Field, T., Pickens, J., Prodromidis, M., Malphurs, J., Fox, N., & Bendell, D. (2000). Targeting adolescent mothers with depressive symptoms for early intervention. *Adolescence*, 35, 381-414.
- 21. Gladstone, T. R. G., & Beardslee, W. R. (2002). Treatment, intervention and prevention with children of depressed parents: A developmental perspective. In S. H.Goodman & I. H. Gotlib (Eds.). Children of depressed parents: Mechanisms of risk and implications for treatment (pp. 277-305). Washington, DC: American Psychological Association.
- Seifer, R., Dickstein, S., Sameroff, A. J., Magee, K. D., & Hayden, L. C. (2001). Infant mental health and variability of parental depression symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*, 40, 1375–1382.
- Tronick, E., & Reck, C. (2009). Infants of depressed mothers. Harvard Review of Psychiatry, 17, 147-156.
- 24. Dawson, G., & Ashman, S. (2000). On the origins of a vulnerability to depression: The influence of early social environment on the development of psychobiological systems related to risk for affective disorder. In C. A. Nelson (Ed.), The effects of early adversity on neurobehavioral development: The Minnesota Symposia on Child Psychology, Vol. 31 (pp. 245-278). New York: Erlbaum & Associates.
- National Scientific Council on the Developing Child. (2005). Excessive stress disrupts the architecture of the developing brain. Working Paper No. 3. Retrieved November 1, 2009, from www.developingchild.harvard.edu
- Dawson, G., Frey, K., Panagiotides, H., Osterling, J., & Hessl, D. (1997). Infants of depressed mothers exhibit atypical frontal brain activity: A replication and extension of previous findings. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 38(2), 179-186.
- 27. Diego, M. A., Field, T., Jones, N. A., & Hernandez-Reif, M. (2006). Withdrawn and intrusive maternal interaction style and infant frontal EEG asymmetry shifts in infants of depressed and non-depressed mothers. *Infant Behavior and Development*, 29, 220-209.

- 28. Ronsaville, D.S., Municchi, G., Laney, C., Cizza, G., Meyer, S.E. & Haim, A. (2006). Maternal and environmental factors influence the hypothalamic-pituitaryadrenal axis response to corticotropin-releasing hormone infusion in offspring of mothers with or without mood disorders. Development & Psychopathology, 18, 173-194.
- 29. Gump, B. B., Reihman, J., Stewart, P., Lonky, E., Darvill, T., Granger, D. A., & Matthews, K. A. (2009). Trajectories of maternal depressive symptoms over her child's life span: Relation to adrenocortical, cardiovascular, and emotional functioning in children. Development & Psychopathology, 21, 207-225.
- 30. Halligan, S. L., Herbert, J., Goodyer, I., & Murray, L. (2007). Disturbances in morning cortisol excretion in association with maternal postnatal depression predict subsequent depressive symptomatology in adolescents. Biological Psychiatry, 62, 40-46.
- 31. Essex, M. J., Klein, M., Cho, E., & Kalin, N. H. (2002). Maternal stress beginning in infancy may sensitize children to later stress exposure: Effects on cortisol and behavior. Biological Psychiatry, 52, 776-784.
- 32. Halligan, S. L., Herbert, J., Goodyer, I. M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. Biological Psychiatry,
- 33. Diego, M. A., Field, T., Hernandez-Reif, M., Schanberg, S., Kuhn, C., & Gonzalez-Quintero, V. H. (2009). Prenatal depression restricts fetal growth. Early Human Develop-
- 34. Mattes, E., McCarthy, S., Gong, G., van Eekelen, J. A., Dunstan, J., & Foster, J. (2009). Maternal mood scores in mid-pregnancy are related to aspects of neonatal immune function. Brain Behavior & Immunity, 23(3), 380-388.
- 35. Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A. M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. Epigenetics, 3, 97-106.
- 36. Lorant, V., Deliege, D., Eaton, W., Robert, A., Philippot, P., and Ansseau, M. (2003). Socioeconomic inequalities in depression: A meta-analysis. American Journal of Epidemiology, 157, 98-112.
- 37. Golding, J. M. (1999). Intimate partner violence as a risk factor for mental disorders: A metaanalysis. Journal of Family Violence, 14, 99-132.
- 38. Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., and Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry, 62, 593-602.
- 39. Talati, A., Wickramaratne, P. J., Pilowsky, D. J., Alpert, J. E., Cerda, G., Garber, J., Hughes, C. W., King, C. A., Malloy, E., Sood, A. B., Verdeli, H., Trivedi, M. H. Rush, A. J., & Weissman, M. M. (2007). Remission of maternal depression and child symptoms among single mothers: A STAR\*D-Child report. Social Psychiatry and Psychiatric Epidemiology, 42, 962-971.
- 40. Clark, R., Tluczek, A., & Wenzel, A. (2003). Psychotherapy for postpartum depression: A preliminary report. American Journal of Orthopsychiatry, 4, 441-454.
- 41. Chabrol, H., & Callahan, S. (2007). Prevention and treatment of postnatal depression. Expert Review of Neurotherapeutics, 7(5), 557-576.
- 42. Cooper, P. J., Murray, L., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of postpartum depression: I.

- Impact on maternal mood. British Journal of Psychiatry, 182, 412-419.
- 43. Forman, D. R., O'Hara, M. W., Stuart, S., Gorman, L. L., Larsen, K., & Coy, K. C. (2007). Effective treatment for postpartum depression is not sufficient to improve the developing mother-child relationship. Development and Psychopathology, 19, 585-602.
- 44. Murray, L., Cooper, P. J., Wilson, A., & Romaniuk, H. (2003). Controlled trial of the short- and long-term effect of psychological treatment of postpartum depression: II. Impact on the mother-child relationship and child outcome. British Journal of Psychiatry, 182, 420-427.
- 45. Nylen, K. J., Moran, T. E., Franklin, C. L., & O'Hara, M. W. (2006). Maternal depression: A review of relevant treatment approaches for mothers and infants. Infant Mental Health Journal, 27(4), 327-343.
- 46. Cicchetti, D., Rogosch, F. A., & Toth, S.L. (2000). The efficacy of toddler-parent psychotherapy for fostering cognitive development in offspring of depressed mothers. Journal of Abnormal Psychology, 28(2), 135-148.
- 47. Field, T. (2002). Prenatal effects of maternal depression. In S. H. Goodman & I. H. Gotlib (Eds.), Children of depressed parents: Mechanisms of risk and implications for treatment (pp. 59-88). Washington, DC: American Psychological Association.
- 48. Toth, S. L., Rogosch, F. A., Manly, J. T., & Cicchetti, D. (2006). The efficacy of toddler-parent psychotherapy to reorganize attachment in the young offspring of mothers with major depressive disorder: A randomized preventive trial. Journal of Consulting and Clinical Psychology, 74, 1006-1016.
- 49. Beardslee, W. R., Wright, E. J., Gladstone, T. R. G., & Forbes, P. (2008). Long-term effects from a randomized trial of two public health preventive interventions for parental depression. Journal of Family Psychology, 21, 703-713.
- 50. Dennis, C. (2005). Psychosocial and psychological interventions for prevention of postnatal depression: Systematic review. British Medical Journal, 331(7507), 15.
- 51. Ogrodniczuk, J. S., & Piper, W. E. (2003). Preventing postnatal depression: A review of research findings. Harvard Review of Psychiatry, 11(6), 291-307.
- 52. Zlotnick, C., Johnson, S. L., Miller, I. W., Pearlstein, T., Howard, M. (2001). Postpartum depression in women receiving public assistance: Pilot study of an interpersonal-therapy-oriented group intervention. American Journal of Psychiatry, 158, 638-640.
- 53. Howard, L. M., Hoffbrand, S. Henshaw, C., Boath, L., & Bradley, E. (2005). Antidepressant prevention of postnatal depression. Cochrane Database of Systematic Reviews, Issue 2. Article No.: CD004363. DOI: 10.1002/14651858. CD004363.pub2.
- 54. Dennis, C. L., & Hodnett, E. D. (2009). Psychosocial and psychological interventions for treating postpartum depression (Review). The Cochrane Library, 2.
- 55. Chazen-Cohen, R., Ayoub, C., Pan, B. A., Roggman, L., Raikes, H., McKelvey, L., Whiteside-Mansell, L., & Hart, A. (2007). It takes time: Impacts of Early Head Start that lead to reductions in maternal depression two years later. Infant and Mental Health Journal, 28, 151-170.
- 56. Tandon, S. D., Parillo, K. M., Jenkins, C., & Duggan, A. K. (2005). Formative evaluation of home visitors' role in addressing poor mental health, domestic violence, and substance abuse among low-income pregnant and parenting women. Maternal and Child Health Journal, 9, 273-283.

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## The Issue

ENSURING THAT YOUNG CHILDREN HAVE SAFE, SECURE ENVIRONMENTS IN WHICH TO GROW, LEARN, and develop healthy brains and bodies is not only good for the children themselves but also builds a strong foundation for a thriving, prosperous society. Science shows that early exposure to circumstances that produce persistent fear and chronic anxiety can have lifelong consequences by disrupting the developing architecture of the brain. Unfortunately, many young children are exposed to such circumstances. While some of these experiences are one-time events and others may reoccur or persist over time, all of them have the potential to affect how children learn, solve problems, and relate to others.

All children experience fears during childhood, including fear of the dark, monsters, and strangers. These fears are normal aspects of development and are temporary in nature. In contrast, threatening circumstances that persistently elicit fear and anxiety predict significant risk for adverse long-term outcomes from which children do not recover easily. Physical, sexual, or emotional abuse; significant maltreatment of one parent by the other; and the persistent threat of violence in the community are examples of such threatening circumstances in a child's environment.

Studies show that experiences like abuse and exposure to violence can cause fear and chronic anxiety in children and that these states trigger extreme, prolonged activation of the body's stress response system. In studies with animals, this type of chronic activation of the stress system has been shown to disrupt the efficiency of brain circuitry and lead to both immediate and long-term physical and psychological problems. This is especially true when stress-system overload occurs during sensitive periods of brain development. While much of the evidence for the effects of stress on the development of brain architecture comes from animal studies, strong similarities in the processes of brain development across species indicate that experiences of persistent fear and chronic anxiety likely exert similarly adverse impacts on the developing brain in humans. Thus, stress-system overload can significantly diminish a child's ability to learn and engage in typical social interactions across the lifespan.

Many policymakers, educators, and even medical professionals are unaware of the potentially significant, long-term risks of exposure to fear-provoking circumstances in children and lack information about the prevalence of these situations in their communities. Critically, 1 in every 7 children, and nearly 1 out of every 40 infants, in the United States experience some form of maltreatment, including chronic neglect or physical, emotional, or sexual abuse. Child maltreatment has been shown to occur most often in families that face excessive levels of stress, such as that associated with community violence, parental drug abuse, or significant social isolation. Research also tells us that nearly half of children living in poverty witness violence, or are indirectly victims of violence. Clearly, for children in these circumstances, the frequent and repetitive threats around them

Science shows that exposure to circumstances that produce persistent fear and chronic anxiety can have lifelong consequences by disrupting the developing architecture of the brain.

create the potential for heightened fear and chronic anxiety.

Behavioral neuroscience research in animals tells us that serious, fear-triggering experiences elicit physiological responses that affect the architecture of the brain as it is developing. These experiences cause changes in brain activity and have been shown to have long-term, adverse consequences for learning, behavior, and health. Studies show that solutions for children are available through programs that effectively prevent specific types of fear-eliciting events, such as physical or sexual abuse. The timely

implementation of such interventions can prevent and treat the harmful effects of exposure to extreme, fear-eliciting circumstances. In addition to these preventive measures, there also are effective treatments for children experiencing high levels of anxiety or chronic fear that result from serious emotional trauma. Despite this rapidly increasing knowledge base, however, significant gaps continue to exist in how society responds to the developmental needs of children who regularly experience serious, fearinducing events.

## What Science Tells Us

SOME TYPES OF FEAR ARE NORMAL ASPECTS OF development. Infants begin to experience feelings of fear and differentiate them from other emotions between 6 and 12 months of age.<sup>4,5</sup> Over the course of the early childhood period, toddlers and preschoolers typically express fear of a wide variety of events or individuals. For example, it is not unusual for a young child to react with wariness or distress when greeted by an unfamiliar adult. Such responses are often called "stranger anxiety" and typically first emerge at around 9 or 10 months of age. This hesitancy toward unfamiliar people generally continues throughout childhood, but diminishes over time, as children's social worlds expand and they interact with increasing numbers of caregivers, relatives, neighbors, and other familiar adults.

Later in early childhood, it is common for children to express fear of both imagined and real circumstances. The emergence and development of imagination, for example, may lead to fear of monsters or the dark. These reactions are

The emergence and course of typical childhood fears are different from the fears and anxiety elicited by traumatic situations such as physical or sexual abuse or exposure to violence.

> typical and usually peak between 4 and 5 years of age. Generally speaking, normal preschool fears do not disrupt a child's life, and they dissipate by age 7 or 8. That is, while children may express these fears at certain times (e.g., bedtime) or in response to certain events (e.g., being surprised by a clown at a birthday party), their overall behavior does not otherwise suggest that they are generally fearful or distressed.

Scientific research provides an explanation

for why children outgrow these normative fears. Many fears are a result of the difficulty young children have in distinguishing between the real and the imaginary. As they get older, children get better at understanding what is real and what it means for something to be "make believe." At the same time, they develop a growing sense of control and predictability over their immediate environment, so that even very young children are less frightened by events if they have some control over them. For example, a toy that scares 12-month-olds because it is loud and unpredictable will elicit less fear if the children are shown how to turn it on and off and are allowed to do so.6 As they get older, children develop the cognitive and social skills needed to better understand predictability in their environment and, therefore, gain a greater sense of control. As these developmental capacities are mastered, many of the normal fears of childhood begin to disappear. Thus, the emergence and course of typical childhood fears are different from the fears and anxiety elicited by traumatic situations such as physical or sexual abuse or exposure to violence: While typical fears disappear with age, the fear and anxiety elicited by maltreatment and other threatening circumstances do not.

Early exposure to extremely fearful events affects the developing brain, particularly in those areas involved in emotions and learning. A large and growing body of research, including animal studies as well as recent neuroimaging studies of human adults, has revealed groundbreaking insights into the brain circuitry that underlies how we learn to be afraid 7,8 and how we come to associate a specific event or experience with negative outcomes.<sup>9,10</sup> Two extensively studied structures located deep in the brain—the amygdala and the hippocampus are involved in fear conditioning.9,10 The amygdala detects whether a stimulus, person, or event is threatening 9,10 and the hippocampus links the fear

response to the context in which the aversive stimulus or threatening event occurred.11 Studies also show that both the amygdala and the hippocampus play an important role in how the body then responds to this threat. Elevated stress hormones such as cortisol have been shown to affect the growth and performance of the hippocampus and the activity of the amygdala in rodents and nonhuman primates, and early and persistent activation of the stress response system adversely affects brain architecture in these critical regions.

Beyond its impact on these two brain structures, heightened stress has also been shown in animals to impair the development of the prefrontal cortex, the brain region that, in humans, is critical for the emergence of executive functions—a cluster of abilities such as making, following, and altering plans; controlling and focusing attention; inhibiting impulsive behaviors; and developing the ability to hold and incorporate new information in decisionmaking. These skills become increasingly important throughout the school years and into adulthood. Behavioral neuroscience research in animals tells us that the prefrontal cortex is highly sensitive to the detrimental effects of excessive stress exposure and that its developing architecture is vulnerable to the negative effects of chronic fear.12

When young children experience serious feartriggering events, they learn to associate that fear with the context and conditions that accom**panied it.** Very young children can actually *learn* to be fearful through a process called "fear conditioning," which is strongly connected to the development of later anxiety disorders. 13,14,15,16 In the typical circumstances of early childhood, fear responses are activated quickly and then dissipate. However, when young children are chronically exposed to perceived or real threat, fear-system activation can be prolonged. In research studies, fear conditioning involves the pairing of a neutral stimulus (e.g., a tone or a light) that normally does not elicit a negative emotional response with an aversive stimulus (e.g., pain) that produces fear. As this conditioning evolves, it solidifies the relation between the two stimuli and then generalizes the fear response to other neutral stimuli that may share similar characteristics with the aversive stimulus. Conditioned fear is apparent when individuals come to experience and express fear

within the context in which the learning occurred. For example, a child who is physically abused by an adult may become anxious in response to both the person and the place where the fear

For young children who perceive the world as a threatening place, a wide range of conditions can trigger anxious behaviors that then impair their ability to learn and to interact socially with others.

learning occurred. Over time, the fear elicited and the consequent anxiety can become generalized, and subsequent fear responses may be elicited by other people and places that bear sometimes only small resemblances to the original conditions of trauma. Consequently, for young children who perceive the world as a threatening place, a wide range of conditions can trigger anxious behaviors that then impair their ability to learn and to interact socially with others. The extent to which these problems affect physical and mental health is influenced by the frequency of the stressful exposure and/or the emotional intensity of the fear-eliciting event.

## Science tells us that unlearning fear is a fundamentally different process from fear learning.

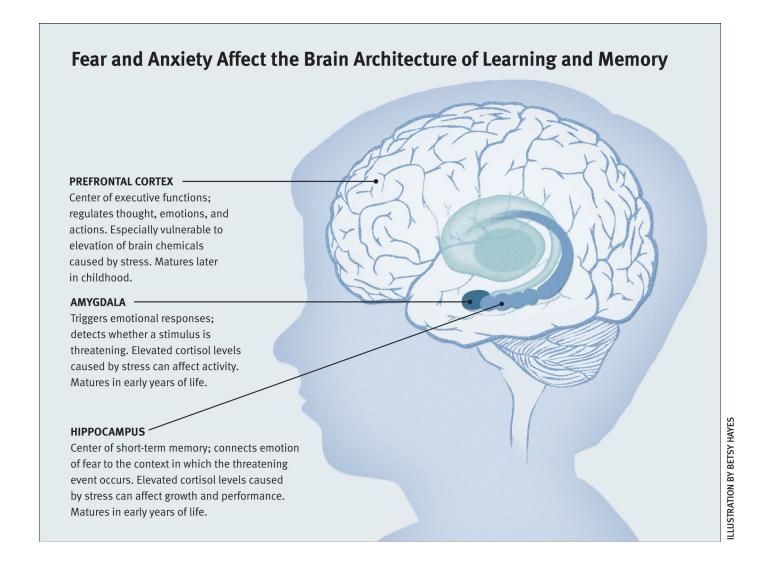
The process of unlearning conditioned fear is called "extinction" and actually involves physically separate and distinct areas of the brain's architecture from those into which fear responses are first incorporated. Generally speaking, the unlearning process involves activity in the prefrontal cortex, which decreases the fear response by regulating the activity of the amygdala. 17,18,19,20 Research tells us that fears are not just passively forgotten over time, but they must be actively unlearned. Studies show that fear learning can occur relatively early in life, 21,22,23 whereas fear unlearning is only achieved later, when certain structures in the brain have matured.<sup>24,25</sup> Consequently, early fear learning can have a significant impact on the physical and mental health of a young child that can take years to remediate.

This understanding of how fear unlearning occurs can be helpful in designing interventions for anxious and fearful children. For example, research has shown that unlearning negative fear responses to specific stimuli such as

animals, insects, heights, or social situations can be accomplished successfully by presenting the aversive stimulus or circumstance at a low level of intensity while the fearful individual is in a safe context. This therapeutic approach is called cognitive behavioral therapy. Providing additional explanations for anxious behavior during these controlled exposures has proven to be particularly successful for reducing anxiety in older children with excessive fears, as their ability to understand these explanations develops. Such interventions work well with specific phobias, as well as social or generalized anxiety, but are not effective in remediating the effects of abuse or neglect.

Chronic and intense fear early in life affects the development of the stress response system and influences the processing of emotional memories.<sup>26,27</sup> When an individual is confronted with

a threat, stress systems are activated and elevate the levels of several different stress chemicals that are circulating throughout the body. 28,29 An increase in one of those chemicals, cortisol, can have a dramatic impact on how memories are processed and stored.<sup>29,30</sup> The production of cortisol and adrenalin (as well as noradrenaline in the brain) in a normal stress response leads to memory formation for events and places that generate danger. More specifically, elevated cortisol levels can strengthen the formation of memories of emotional events,31,32 block the ability to unlearn fear memories,33 and enhance the formation of memories of the surrounding context in which the fearful event occurred.34 Interestingly, too much cortisol can also have the opposite effect and actually impair memory and learning in non-threatening contexts.<sup>35</sup> Thus, the biological response to stress is intimately involved in both fear learning and unlearning.



Fear learning can form emotional memories that are extremely powerful and long lasting. These memories are relived by individuals who experienced a traumatic event when cues in the environment activate those memories. This repeated recall or retrieval of the memory makes emotional memories both more easily activated and more resistant to being forgotten.<sup>29,30</sup> The repeated recall of a traumatic event can lead to additional release of cortisol, even in the absence of the actual event. Behavioral neuroscience research with animals has shown that chronic elevation of cortisol can have a number of detrimental effects, including increased damage to brain cells in areas that support learning, thereby leading to increased impairment in subsequent memory formation. 30,31

Persistent fear can distort how a child perceives and responds to threat. Fear learning typically takes place in specific contexts and results in those fears becoming associated with the places where the learning occurred. Children may also express fear in response to situations that are similar (not identical) to those initially learned or to situations that are similar to the contexts in which the original learning occurred. These are called "generalized" fear responses, and they are thought to underlie the expression of later anxiety disorders, including post-traumatic stress disorder (PTSD). 15,36,37 Thus, although all individuals display a heightened fear response when faced with threatening contexts, 36,38 individuals with anxiety disorders show this same increased fear response when faced with similar contexts that are known to be safe. 36,38,39 Indeed, children who have had chronic and intense fearful experiences often lose the capacity to differentiate between threat and safety. This impairs their ability to learn and interact with others, because they frequently perceive threat in familiar social circumstances, such as on the playground or in school. These responses inhibit their ability to learn and often lead to serious anxiety disorders. 40,41

Young children who have been exposed to traumatic circumstances also have difficulty identifying and responding to different expressions of emotions, and, therefore, have trouble forming healthy relationships. 42,43,44,45,46 These deficits lead to general problems with social interaction, such as understanding others' facial expressions and emotions. For example, children raised in physically abusive households show heightened sensitivity (compared with non-abused children) to angry faces, which negatively affects their brain function and behavior. 47,48,49,50 Learning to identify anger quickly and successfully—in order to avoid being harmed is a highly adaptive and appropriate response to an abusive environment. However, an increased tendency to assume someone is angry when his or her facial expression is ambiguous can be inappropriate and maladaptive

# Children who have had chronic and intense fearful experiences often lose the capacity to differentiate between threat and safety.

in a typical, non-threatening social setting and even dangerous in unfamiliar social settings.<sup>51</sup> This "attention bias" to threat is associated with interpreting ambiguous information in a negative fashion, and it is linked to greater vulnerability to stress and anxious behaviors as well as to a greater likelihood to respond aggressively as a form of self-defense in neutral circumstances that are erroneously viewed as threatening. Thus, the extent to which children with a heightened attention bias to threat view the world as a hostile and threatening place can be viewed as both a logical adaptation to an abusive environment and a potent risk factor for behavior problems in later childhood, adolescence, and adult life.

Early exposure to intense or persistent feartriggering events affects children's ability to learn. There is extensive and growing scientific evidence that prolonged and/or excessive exposure to fear and states of anxiety can cause levels of stress that can impair early learning and adversely affect later performance in school, the workplace, and the community. Multiple studies in humans have documented problems in cognitive control and learning as a result of toxic stress. 52,53 These findings have been strengthened by research evidence from non-human primates and rodents that is expanding our understanding of the brain mechanisms underlying these difficulties.

The brain region in animals that appears highly vulnerable to adversity in this regard is the prefrontal cortex, which is the critical area for regulating thought, emotions, and actions as well as for keeping information readily

accessible during the process of active learning. For example, researchers have found that elevations in brain chemicals like noradrenline, an important neurotransmitter, can impair functions that are controlled by the prefrontal region by altering the activity of neurons in that area of the brain. In a related fashion, humans

experiencing chronic stress have been shown to perform poorly on tasks related to prefrontal cortex functioning (such as working memory or shifting attention), and their ability to control their emotions is typically impaired.<sup>12</sup>

# **Correcting Popular Misrepresentations of Science**

THERE ARE A NUMBER OF WIDESPREAD MISCONceptions about how children experience, respond to, and learn fear. Many of these assumptions derive from overgeneralizations of what fears are typical at specific developmental stages as well as misunderstandings about what children can simply "outgrow" as they mature. Being afraid of strangers and monsters are common examples of typical fears. In contrast, research has demonstrated convincingly that excessive fear and anxiety caused by experiences such as abuse and neglect can affect the developing child in very different ways from the fear experiences that characterize a typical childhood.

Contrary to popular belief, serious fear-triggering events can have significant and long-lasting impacts on the developing child, beginning in infan**cy.** Science tells us that young children *can* perceive threat in their environment but, unlike adults, they do not have the cognitive or physical capacities to regulate their psychological response, reduce the threat, or remove themselves from the threatening situation. Research also shows that very young infants can learn to fear certain places, events, or people. These learned fear responses may disrupt the physiology of the stress response system, making it more difficult for the body to respond appropriately to typical, mild stress in everyday contexts later in life. Furthermore, when fear is learned, normal situations and circumstances can elicit responses that are harmful to a child's development.

Children do not naturally outgrow early learned fear responses over time. Fear learning early in life can often be adaptive—think about how a young child learns to stay away from hot surfaces. Thus, fear learning and associated

memories that occur early in life get built into our brain architecture and do not dissipate with age. During typical development, children learn to regulate their responses to mild threats and stresses. However, if young children are exposed to persistent fear and excessive threat during particularly sensitive periods in the developmental process, they may not develop healthy patterns of threat/stress regulation. When they occur, these disruptions do not naturally disappear.

Simply removing a child from a dangerous environment will not by itself undo the serious consequences or reverse the negative impacts of early fear learning. There is no doubt that children in harm's way should be removed from a dangerous situation. However, simply moving a child out of immediate danger does not in itself reverse or eliminate the way that he or she has learned to be fearful. The child's memory retains those learned links, and such thoughts and memories are sufficient to elicit ongoing fear and make a child anxious. Science clearly shows that reducing fear responses requires active work and evidence-based treatment. Children who have been traumatized need to be in responsive and secure environments that restore their sense of safety, control, and predictability—and supportive interventions are needed to assure the provision of these environments. Thus, it is critical that communities be equipped to address the sources of fear in children's lives. Where indicated, children with anxiety can benefit from scientifically proven treatments, such as cognitive behavioral therapy, which have been shown to reduce anxiety and fear.

# The Science-Policy Gap

Advances in the science of child development tell us that significant fear-eliciting experiences early in life can disrupt the typical development of stress regulation as well as learning, memory, and social behavior, yet there is still widespread resistance in the policy arena to fully addressing the needs of young children who have been traumatized. Building on decades of evidence from behavioral research, it is now abundantly clear that young children who are exposed to circumstances that produce persistent fear are at heightened risk for anxiety disorders and other mental health problems that persist into adulthood. Concurrently, a variety of prevention and early intervention programs have been developed to address the needs of young children who have been exposed to such fearful situations as physical abuse or family violence. The limited availability of these kinds of programs for very young children represents a striking failure to relieve immediate distress as well as prevent serious and costly long-term disability.

The lack of availability of adequate health insurance to cover the cost of therapeutic treatment for young children who are experiencing persistent fear and chronic anxiety represents a significant lost opportunity to ameliorate preventable impairments in physical and mental health that can have lifelong implications. The science of child development points the way toward effective approaches to the treatment of children with excessive anxiety and fear. These methods, if administered early, can reduce the incidence of anxiety disorders in children and prevent the kinds of elevated stress responses that lead to physical and mental health impairments later in life. Addressing the current gaps among what science knows about effective treatments, what is available in health care and early childhood settings, what is covered by health insurance, and the availability of coverage for all children needs to be an important policy priority.

# **Policy Implications**

Programs and policies that are designed to address domestic violence, substance abuse, and mental health problems in adults who have (or are expecting) children would have considerably stronger impacts if their focus also included the children's developmental needs, beginning in the prenatal period. Extensive scientific evidence shows that significant mental health problems in parents can be a source of fear and stress in children and have negative effects on a child's development. Through reduced caregiving capacities, the co-occurrence of child neglect or abuse, and exposure to other sources of fear and stress, parental mental health conditions have direct consequences for the health and well-being of their children. The fear that abuse and neglect elicits in children can lead to serious dysregulation of their emotions and behavior control. That said, there are promising interventions that have been shown to be effective in preventing abuse and neglect. Prenatal home visiting for

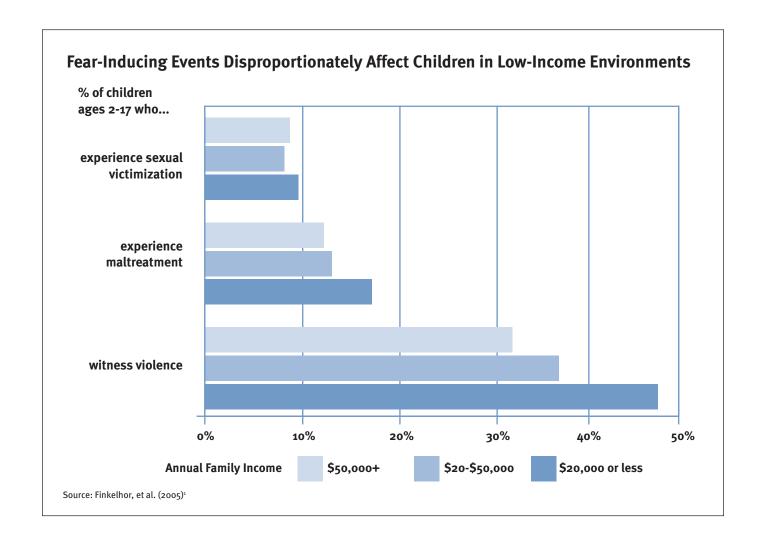
first-time mothers provided by trained nurses is one example of a program whose effectiveness has been documented by randomized controlled trials in multiple locations.<sup>54,55</sup> Other promising approaches include specific training for

**Extensive scientific evidence shows that** significant mental health problems in parents can be a source of fear and stress in children and have negative effects on a child's development.

professionals who work with families experiencing trauma and the incorporation of developmental interventions for young children in programs that address domestic violence.<sup>56</sup> When hard evidence of program effectiveness is available, the imperative of providing appropriate preventive or intervention services is clear. When program evaluation data on successful prevention or interventions for specific threats to child well-being are limited or nonexistent, the compelling evidence of potential harm to children's development calls for serious investment in the design and testing of new strategies for prevention and treatment that are grounded in sound scientific principles, subjected to rigorous evaluation, and improved continuously over time.

Child welfare policies and programs that are mandated to assess and intervene in cases of suspected and/or confirmed abuse or neglect must address the full range of children's developmental needs, not just focus on their physical safety. All states have established systems that require the reporting of suspected child maltreatment and the provision of protective services for children whose health or well-being

is threatened. These services focus largely on issues related to physical safety, reduction of repeated injury, and child custody. Advances in neuroscience now indicate that evaluations of maltreated children that rely solely on physical examination and screening for broken bones are insufficient and must be augmented by comprehensive developmental assessments and appropriate intervention by skilled professionals as needed. To this end, it is important to note that early intervention programs for children with developmental delays or disabilities have the expertise to provide many of the services needed by maltreated children, and these programs are already available in all states under a federal entitlement specified in the Individuals With Disabilities Education Act (IDEA). Moreover, the most recent reauthorizations of the relevant federal legislation for both the child welfare and early intervention systems (the Keeping Children and Families Safe Act and IDEA,



respectively) include requirements for regularized referrals of newly established child protective cases from the child welfare agency to the early intervention system for developmental screening. Notwithstanding its strong, sciencebased rationale, the implementation of this linkage has been limited to date and requires immediate attention. The evidence that significant "fear learning" with long-term consequences can occur as early as the first year of life—and that the capabilities for effective "fear unlearning" do not fully emerge until later-underscores the extent to which the limited child development expertise available within the nation's child welfare system can no longer be justified.

Early identification and treatment for anxiety and post-traumatic stress disorders in young children should be routinely available through existing services for families, as they can significantly affect the future mental and physical health of children. Advances in neuroscience, behavioral and developmental studies, and clinical research have all converged to contribute to a shared understanding of both the reality of early childhood mental health impairment and the parameters of successful preventive intervention and effective treatments. Early in infancy and childhood, intervention and treatment should focus on programs that provide families with necessary services, supports, and expertise, while later in development, supports should be focused more on children themselves.

The critical importance of intervening early in the lives of young children who experience excessive fear and anxiety is evident in two domains: the need to relieve current suffering and the opportunity to prevent enduring impairment that can lead to a lifetime of poor mental and physical health, diminished economic productivity, and antisocial behavior. With these high stakes in mind, all of society

would benefit from a greater capacity to address the problem of excessive fear and anxiety in young children across a broad array of service systems, including health care, child welfare programs, school- and child care-based health services, and the foster care system, among others.

Policies with a broad mandate to reduce poverty and neighborhood violence would likely have greater long-term impacts if they also included explicit and focused attention on the prevention of fear and anxiety overload in young chil**dren.** Children who live in violent communities have been shown to have more behavior problems, greater evidence of post-traumatic stress

All of society would benefit from a greater capacity to address the problem of excessive fear and anxiety in young children across a broad array of service systems.

disorder, and increased physical symptoms such as headaches and stomachaches, as well as lower capacity for empathy and diminished selfesteem.<sup>57</sup> Programs focused on the reduction of poverty, domestic violence, substance abuse, and neighborhood violence are examples of the kinds of community-based services whose impacts could be enhanced by incorporating targeted interventions to explicitly address the emotional needs of young children living under these conditions. When delivered effectively, such interventions could have a multiplier effect into the next generation by reducing both the individual and societal costs of the negative developmental effects of persistent fear, including mental health impairments, antisocial behavior, physical disease, and violent crime.

## References

- Finkelhor, D., Ormrod, R., Turner, H., & Hamby, S. L. (2005). The victimization of children and youth: A comprehensive, national survey. *Child Maltreatment*, 10(1), 5-25
- U.S. Department of Health and Human Services, Administration on Children, Youth, and Families. (2009). *Child maltreatment 2007*. Washington, DC: U.S. Government Printing Office.
- Centers for Disease Control and Prevention (2009). Understanding child maltreatment. Retrieved from http:// www.cdc.gov/violenceprevention/pdf/CM-FactSheet-a. pdf
- Lewis, M. & Michalson, L. (1983). Children's emotions and moods: Developmental theory and measurement. New York: Plenum Press.
- Nelson, C. A., & De Haan, M. (1996). Neural correlates of infants' visual responsiveness to facial expressions of emotion. *Developmental Psychobiology*, 29(7), 577-595.
- Gunnar-von Gnecheten, M. R. (1978). Changing a frightening toy into a pleasant toy by allowing the infant to control its actions. *Developmental Psychology*, 14, 157-162.
- Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. *Neuron*, 48, 175-187.
- Delgado, M. R., Olsson, A., & Phelps, E. A. (2006). Extending animal models of fear conditioning to humans. *Biological Psychology*, 23, 39-48.
- 9. LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23, 155-184.
- LeDoux, J. E. & Phelps, E. A. (2008). Emotional networks in the brain. In M. Lewis, J. M. Haviland-Jones, & L. F. Barrett, (Eds.), *Handbook of emotions* (pp. 159-179). New York: Guilford Press.
- 11. Kim, J. J., & Fanselow, M. S. (1992). Modality-specific retrograde amnesia of fear. *Science*, 256, 675-677.
- Arnsten, A. F. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10, 410-422.
- Watson, J. B., & Rayner, R. (1920). Conditioned emotional reactions. *Journal of Experimental Psychology*, 3(1), 1-14.
- Pavlov, I. (1927). Conditioned reflexes. London: Oxford University Press.
- Grillon, C., & Morgan, C. A. I. (1999). Fear-potentiated startle conditioning to explicit and contextual cues in Gulf War veterans with posttraumatic stress disorder. *Journal of Abnormal Psychology*, 108, 134-142.
- Pine, D. S. (1999). Pathophysiology of childhood anxiety disorders. *Biological Psychiatry*, 46, 1555-1566.
- Quirk, G. J., Garcia, R., & Gonzalez-Lima, F. (2006). Prefrontal mechanisms in extinction of conditioned fear. *Biological Psychiatry*, 60, 337-343.
- Sotres-Bayon, F., Bush, D. E. A., & LeDoux, J. E. (2009).
   Emotional perseveration: An update on prefrontal-amygdala interactions in fear extinction. *Learning & Memory*, 11, 525-535.
- Morgan, M. A., Romanski, L. M., & LeDoux, J. E. (1993). Extinction of emotional learning: Contribution of medial prefrontal cortex. *Neuroscience Letters*, 163, 109-113.
- 20. Phelps, E. A., Delgado, M., Nearing, K., & LeDoux, J.

- (2004). Extinction learning in humans: Role of the amygdala and vmPFC. *Neuron*, 43(6), 897-905.
- Sullivan, R.M., Landers, M., Yeaman, B., & Wilson, D. A. (2000). Neurophysiology: Good memories of bad events in infancy. *Nature*, 407, 38-39.
- Prather, M.D., Lavenex, P., Mauldin-Jourdain, M. L., Mason, W. A., Capitanio, J. P., Mendoza, S. P., & Amaral, D. G. (2001). Increased social fear and decreased fear of objects in monkeys with neonatal amygdala lesions. *Neuroscience*, 106(4), 653-658.
- Rudy, J. W. (1993). Contextual conditioning and auditory cue conditioning dissociate during development. *Behavioral Neuroscience*, 107, 887-891.
- Carew, M. B., & Rudy, J. W. (1991). Multiple functions of context during conditioning: A developmental analysis. *Developmental Psychobiology*, 24, 191-209.
- 25. Kim, J. H., & Richardson, R. (2008). The effect of temporary amygdala inactivation on extinction and reextinction of fear in the developing rat: Unlearning as a potential mechanism for extinction early in development. *Journal of Neuroscience*, 28, 1282-1290.
- Sanchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology*, 13(3), 419-449.
- Nemeroff, C. B. (2004). Neurobiological consequences of childhood trauma. *Journal of Clinical Psychiatry*, 65(1), 18-28.
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, 87, 873-904.
- 29. de Kloet, E. R., Karst, H., & Joëls, M. (2008). Corticosteroid hormones in the central stress response: Quick-andslow. *Frontiers in Neuroendocrinology*, 29(2), 268-272.
- Wiegert, O., Joels, M., & Krugers, H. J. (2008). Corticosteroid hormones, synaptic strength and emotional memories: Corticosteroid modulation of memory a cellular and molecular perspective. *Progress in Brain Research*, 167, 269-271.
- Roozendaal, B., Barsegyan, A., & Lee, S. (2008). Adrenal stress hormones, amygdala activation, and memory for emotionally arousing experiences. *Progress in Brain Re*search, 167, 79-97.
- McGaugh, J. L., Cahill, L., & Roozendaal, B. (2006). Involvement of the amygdala in memory storage: Interaction with other brain systems. *Proceedings of the National Academy of Sciences USA*, 93, 13508-13514.
- 33. Yang, Y. L., Chao, P. K., Ro, L. S., Wo, Y. Y. P., & Lu, K. T. (2007). Glutamate NMDA receptors within the amygdala participate in the modulatory effect of glucocorticoids on extinction of conditioned fear in rats. *Neuropsychopharmacology*, 32, 1042-1051.
- 34. Brinks, V., de Kloet, E. R., & Oitzl, M. S. (2008). Strain specific fear behaviour and glucocorticoid response to aversive events: Modelling PTSD in mice. *Progress in Brain Research*, 167, 257-261.
- Roozendaal, B., McEwen, B. S., & Chattarji, S. (2009).
   Stress, memory, and the amygdala. *Nature Reviews Neuroscience*, 10, 423-433.

- 36. Grillon, C. (2002). Startle reactivity and anxiety disorders: aversive conditioning, context, and neurobiology. Biological Psychiatry, 52, 958-975.
- 37. Davis, M. (2006). Neural systems involved in fear and anxiety measured with fear-potentiated startle. American Psychologist, 61(8), 741-756.
- 38. Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., & Pine, D. S. (2005). Classical fear conditioning in the anxiety disorders: A meta-analysis. Behaviour Research & Therapy, 43(11): p. 1391-1424.
- 39. Lissek, S., Biggs, A. L., Rabin, S. J., Cornwell, B. R., Alvarez, R. P., Pine, D.S., & Grillon, C. (2008). Generalization of conditioned fear-potentiated startle in humans: Experimental validation and clinical relevance. Behaviour Research & Therapy, 46(5), 678-687.
- 40. Grillon, C., Dierker, L., & Merikangas, K. R. (1998). Fearpotentiated startle in adolescent offspring of parents with anxiety disorders. Biological Psychiatry, 44, 990-997.
- 41. Reeb-Sutherland, B. C., Helfinstein, S. M., Degnan, K. A., Perez-Edgar, K., Henderson, H. A., Lissek, S., Chronis-Tuscano, A., Grillon, C., Pine, D. S., & Fox, N. A. (2009). Startle response in behaviorally inhibited adolescents with a lifetime occurrence of anxiety disorders. Journal of the American Academy of Child and Adolescent Psychiatry, 48(6), 610-617.
- 42. Wismer Fries, A. B., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuro-peptides critical for regulating social behavior. Proceedings of the National Academy of Sciences USA, 102(47), 17237-17240.
- 43. Zeanah, C. H., Smyke, A. T., & Dumitrescu, A. (2002). Attachment disturbances in young children II: Indiscriminate behavior and institutional care. Journal of the American Academy of Child and Adolescent Psychiatry, 41(8),
- 44. Zeanah, C. H., Smyke, A. T., & Koga, S. F. (2005). Attachment in institutionalized and community children in Romania. Child Development, 76(5), 1015-1028.
- 45. O'Connor, T. G., Rutter, M., & The English and Romanian Adoptees (ERA) Study Team (2000). Attachment disorder behavior following early severe deprivation: extension and longitudinal follow-up. Journal of the American Academy of Child and Adolescent Psychiatry, 39(6), 703-712.
- 46. O'Connor, T. G., Bredenkamp, D., Rutter, M., & The English and Romanian Adoptees (ERA) Study Team. (1999). Attachment disturbances and disorders in children exposed to early severe deprivation. Infant Mental Health Journal, 20(1), 10-29.
- 47. Pollak, S. D., Messner, M., Kistler, D. J., & Cohn, J. F. (2009). Development of perceptual expertise in emotion recognition. Cognition, 110(2), 242-247.
- 48. Pollak, S. D. & Kistler, D. J. (2002). Early experience is associated with the development of categorical representations for facial expressions of emotion. Proceedings of the National Academy of the Sciences USA, 99(13), 9072-9076.
- 49. Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. Developmental Psychology, 36(5),
- 50. Pollak, S. D., & Tolley-Schell, S. A. (2003). Selective

- attention to facial emotion in physically abused children. Journal of Abnormal Psychology, 112(3), 323-338.
- 51. Pollak, S. D. (2008). Mechanisms linking early experience and the emergence of emotions: illustrations from the study of maltreated children. Current Directions in Psychological Science, 17, 370-375.
- 52. National Scientific Council on the Developing Child. (2005). Excessive stress disrupts the architecture of the developing brain (Working Paper No. 3). Retrieved from http://www.developingchild.net
- 53. Shonkoff, J., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. Journal of the American Medical Association 301(21), 2252-2259.
- 54. MacMillan, H. L., Wathen, C. N., Barlow, J., Fergusson, D. M., Leventhal, J. M., & Taussig, H. N. (2009). Interventions to prevent child maltreatment and associated impairment. Lancet, 373(9659), 250-266.
- 55. Donelan-McCall, N., Eckenrode, J., & Olds, D. L. (2009). Home visiting for the prevention of child maltreatment: Lessons learned during the past 20 years. Pediatric Clinics of North America, 56, 389-403.
- 56. Poole, A., Beran, T., & Thurston, W. (2008). Direct and indirect services for children in domestic violence shelters. Journal of Family Violence, 23, 679-686.
- 57. Huth-Bocks, A. C., Levendosky, A. A., & Semel, M. A. (2001). The direct and indirect effects of domestic violence on young children's intellectual functioning. Journal of Family Violence, 16(3), 269-290.
- 58. Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. Arch Gen Psychiatry, 65(4), 409-415.

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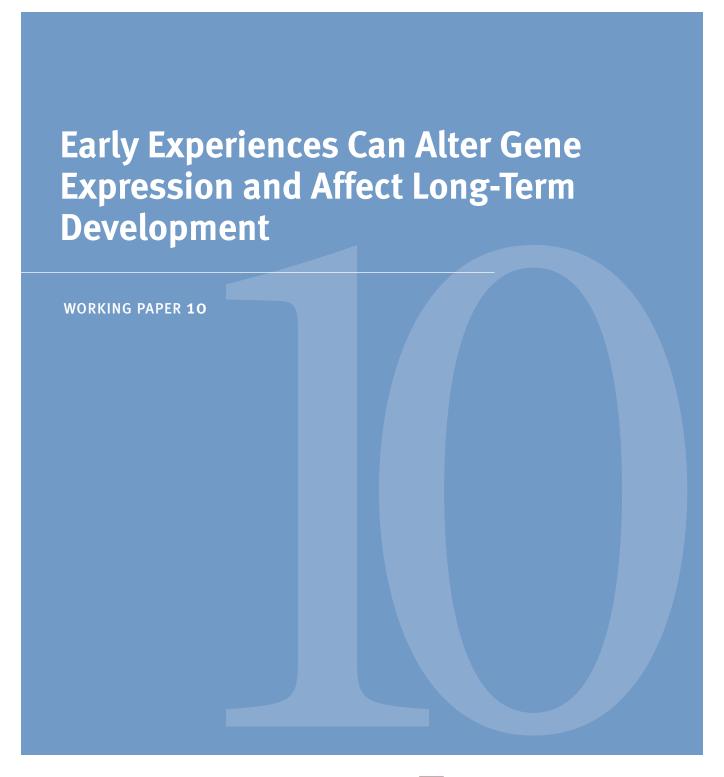
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## The Issue

NEW SCIENTIFIC RESEARCH SHOWS THAT ENVIRONMENTAL INFLUENCES CAN ACTUALLY AFFECT whether and how genes are expressed. Thus, the old ideas that genes are "set in stone" or that they alone determine development have been disproven. In fact, scientists have discovered that early experiences can determine how genes are turned on and off and even whether some are expressed at all. <sup>1,2,3</sup> Therefore, the experiences children have early in life—and the environments in which they have them—shape their developing brain architecture and strongly affect whether they grow up to be healthy, productive members of society. This growing scientific evidence supports the need for society to re-examine the way it thinks about the circumstances and experiences to which young children are exposed.

The approximately 23,000 genes that children inherit from their parents form what is called the "structural genome." Scientists liken the structural genome to the hardware of a computer both determine the boundaries of what's possible, but neither works without an operating system to tell it what to do. In the genome, that operating system is called the epigenome.4 Like the software in an operating system, the epigenome determines which functions the genetic "hardware" does and does not perform.5 This system is built over time as positive experiences, such as exposure to rich learning opportunities, or negative influences, such as environmental toxins or stressful life circumstances, leave a chemical "signature" on the genes. These signatures can be temporary or permanent, and both types affect how easily the genes are switched on or off. For example, even though identical twins have the same structural genomes, their different experiences result in different epigenomes.<sup>6</sup> These differing experiences leave signatures on the epigenome that cause some genes to be expressed differently. This explains why genetically identical twins, though similar in many ways, can exhibit different behaviors, skills, health, and achievement in both school and, later, in the workplace.

The field of epigenetics is relatively new and at the cutting-edge of the biological sciences. To date, scientists have found that *temporary* epigenetic chemical modifications control when and where most of our genes are turned on and off. This, however, is not the entire story. Certain experiences can also cause *enduring* epigenetic modifications in hundreds of genes that have already been identified, and the list is growing.<sup>7,8</sup> Increasing

evidence shows that experience-driven, chemical modifications of these latter genes appear to play particularly key roles in brain and behavioral development. This new knowledge has motivated scientists to look more closely at the factors that shape the epigenome and to study whether interventions can reverse these modifications when negative changes occur.

Nutritional status, exposure to toxins and drugs, and the experiences of interacting with varied environments can all modify an individual's epigenome. Epigenetic instructions that change how and when certain genes are turned on or off can cause temporary or

Like the software in a computer's operating system, the epigenome determines which functions the genetic "hardware" does and does not perform.

enduring health problems. Moreover, research in both animals and humans shows that some epigenetic changes that occur in the fetus during pregnancy can be passed on to later generations, affecting the health and welfare of children, grandchildren, and their descendents. 10,11,12 For example, turning on genes that increase cell growth, while at the same time switching off genes that suppress cell growth, has been shown to cause cancer. 13,14 Repetitive, highly stressful experiences can cause epigenetic changes that damage the systems that manage one's response to adversity later in life. 2,3,15 On the other hand, supportive environments and rich learning experiences generate positive epigenetic

signatures that *activate* genetic potential.<sup>16</sup> In this second case, the stimulation that occurs in the brain through active use of learning and memory circuits can result in epigenetic changes that establish a foundation for more effective learning capacities in the future.<sup>17,18</sup>

As we get older, new experiences can continue to change our epigenome. However, science tells us that the chemical signatures imprinted on our genes during fetal and infant development can have significant influences on brain architecture that last a lifetime. Stated simply, the discovery of the epigenome provides an explanation, at the molecular level, for why and how early positive and negative experiences can have lifelong impacts.<sup>2,3,19,20</sup>

Policymakers can use this knowledge to inform decisions about the allocation of resources for interventions that affect the life circumstances of young children—knowing that effective interventions can literally alter how children's genes work and, thereby, have long-lasting effects on their mental and physical health, learning, and behavior. In this respect, the epigenome is the crucial link between the external environments that shape our experiences and the genes that guide our development.

## What Science Tells Us

OVER THE PAST 50 YEARS, EXTENSIVE RESEARCH has demonstrated that the healthy development of all organs, including the brain, depends on how much and when certain genes are expressed. When scientists say that genes are "expressed," they are referring to whether they are turned on or off—essentially whether and when genes are activated to do certain tasks. Research has shown that there are many non-inherited environmental factors and experiences that have the power to chemically mark genes and control their functions. These influences create a new genetic landscape, which scientists call the epigenome. Some of these experiences lead to chemical modifications that change the expression of genes temporarily, while increasing numbers have been discovered that leave chemical signatures that result in an enduring change in gene expression.

Early prenatal or postnatal experiences and exposures influence long-term outcomes by chemically altering the structure of genes. Known as *epigenetic* modification (from the Greek root *epi*, meaning upon or over), these chemical signatures are written on top of the gene without actually altering the genetic code itself.<sup>21</sup> Instead, the signatures attract or repel other chemicals that help the genes produce the proteins that are the building blocks our brains and bodies need to develop. Research tells us that some genes can only be modified epigenetically during certain periods of development, defined as

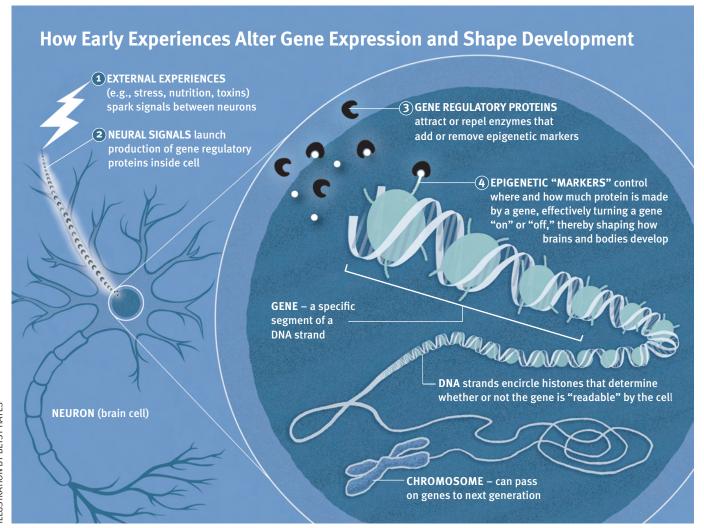
*critical periods* of modification, while other genes are open to alterations throughout life. <sup>2,3,22,23,24,25</sup>

Epigenetic modification typically occurs in cells that comprise organ systems, thereby influencing how these structures develop and function. Experiences that change the epigenome early in life, when the specialized cells of organs such as the brain, heart, or kidneys are first developing, can have a powerful impact on physical and mental health for a lifetime.26 We are also learning from new scientific discoveries in both animals and humans that environmental factors, such as certain drugs or the nutritional status of the mother, have the potential to cause epigenetic changes to genes in egg or sperm cells in the fetus. When such changes occur, this new chemical signature of the DNA is enduring and can be inherited by future generations. 27,28,29

The brain is particularly responsive to experiences and environments during early development, which influences how well or poorly its architecture matures and functions. We know from extensive research that the physiological activity created by experience is powerful in shaping brain architecture and actually changes the chemistry that encodes the genes in brain cells.<sup>30</sup> Put simply, the brain adapts to the experiences it has. Certain types of adaptations result in healthy systems, such as effective learning and memory, and other adaptations lead to the development of unhealthy systems, such as setting a stress response activation level too high

or too low. The physiological activity caused by positive mastery experiences can lead to epigenetic changes that control the expression of genes in brain cells that are essential for successful learning. 17,18,31 In a parallel fashion, exposure to damaging levels of stress early in life can lead to long-lasting epigenetic changes in brain cells that direct how our bodies respond to adversity throughout the lifespan.<sup>32</sup> In short, early experiences cause epigenetic adaptations in the brain that influence whether, when, and how genes build the capacity for future skills to develop.

Modification of the epigenome caused by stress during fetal and child development affects how well or poorly we respond to stress as adults and can result in increased risk of adult disease. Some of our genes provide instructions for how our bodies respond to stress, and research has shown that these genes are clearly subject to epigenetic modification. For example, research in animals has shown that stressful experiences to which the pregnant mother is exposed, or to which the offspring is exposed soon after birth, can produce epigenetic changes that chemically modify the receptor in the brain that controls the stress hormone cortisol and, therefore, determines the body's response to threat (the fight-or-flight response). 19,33,34 Healthy stress responses are characterized by an elevation in blood cortisol followed by a return to baseline to avoid a highly activated state for a prolonged period of time. If young children or pregnant mothers experience toxic stress—as a result of serious adversity (such as chronic neglect, abuse, or exposure to violence) in the absence of protective relationships-persistent epigenetic changes can result.32 These modifications have been shown to cause prolonged stress responses, which can be likened to revving a car engine for long periods of time. Excessive stress has been correlated with changes in brain architecture and chemistry as well as animal behaviors that resemble anxiety and depression in



humans. 35,36,37,38,39,40 Human studies have found connections between highly stressful experiences in children and increased risk for later mental illnesses, including generalized anxiety disorder and major depressive disorder. 41,42,43 Atypical stress responses over a lifetime can also result in increased risk for physical ailments, such as asthma, hypertension, heart disease and diabetes. 29,32,41,42,43,44,45,46,47,48

In addition to adverse experiences, a wide variety of chemicals, nutrients, and drugs are also capable of modifying the epigenome for longlasting effects on gene expression. Epigenetic modification caused by exposure to toxic substances can either turn genes off or on, and both conditions have been linked to increased risk for mental and physical illnesses. Certain dietary supplements (such as excessive amounts of folic acid, choline, or vitamin B<sub>12</sub>) and chemicals (such as bisphenol A, or BPA, which is found in some plasticware) can turn genes off. 1,8,49,50 Some genes that can be turned on by heavy metals, such as cadmium, nickel, and lead, have been linked to the cellular over-activity that results in an increased risk for certain kinds of cancer.<sup>28,51,52</sup> Many organs, including the brain, are most vulnerable to the influence of these substances on gene expression during the period of fetal and infant development, when basic organ systems are being built. These resulting differences in gene expression, in turn, can lead to fundamental changes in brain architecture and the biological systems that govern how well we function later in life.

Recent research demonstrates that even after the epigenome has been modified, there may be ways to alter it again that actually can reverse negative changes and restore functioning. Experiments in animals have shown that certain types of epigenetic modifications that were thought to be permanent can be reversed under certain conditions.35,53 Most recently, researchers found that stressful experiences during early postnatal development resulted in epigenetic modification that caused exaggerated stress responses in adult animals, and that subsequent drug treatment of the adults could eliminate these adverse DNA changes. In this case, reversing the chemical modification resulted in increased expression of genes that control the stress response, and when exposed to subsequent stress, the treated adult animals had a normal response (that is, the adverse effect of the early postnatal experience had, indeed, been reversed). We now know that the same types of epigenetic chemical modifications can occur in adult humans who endured extreme stress as children, such as from physical abuse.<sup>19</sup> This possibility of reversibility is generating a flurry of research activity, because it has direct implications for developing new interventions for physical and mental illnesses that we now know are due in part to epigenetic modification. As promising as this work appears to be, further research is needed to determine if and how the reversal of epigenetic modifications can be achieved in humans.

# **Correcting Popular Misrepresentations of Science**

UNTIL RECENTLY, THE INFLUENCES OF GENES were thought to be set, and the effects of children's experiences and environments on brain architecture and long-term physical and mental health outcomes remained a mystery. That lack of understanding led to several misleading conclusions about the degree to which negative and positive environmental factors and experiences can affect the developing fetus and young child. The following misconceptions are particularly important to set straight.

Contrary to popular belief, the genes inherited from one's parents do not set a child's future development in stone. Variations in DNA sequences between individuals certainly influence the way in which genes are expressed and how the proteins encoded by those genes will function. But that is only part of the story—the environment in which one develops, before and soon after birth, provides powerful experiences that chemically modify certain genes which, in turn, define how much and when they are expressed. Thus, while genetic factors exert

potent influences, environmental factors have the ability to alter family inheritance.

Although frequently misunderstood, adverse fetal and early childhood experiences can-and do-lead to physical and chemical changes in the brain that can last a lifetime. Injurious experiences, such as malnutrition, exposure to chemical toxins or drugs, and toxic stress before birth or in early childhood are not "forgotten," but rather are built into the architecture of the developing brain through the epigenome. The "biological memories" associated with these epigenetic changes can affect multiple organ systems and increase the risk not only for poor physical and mental health outcomes but also for impairments in future learning capacity and behavior.

Despite some marketing claims to the contrary, the ability of so-called enrichment programs to enhance otherwise healthy brain development is not known. While parents and policymakers might hope that playing Mozart recordings to newborns will produce epigenetic changes that enhance cognitive development, there is absolutely no scientific evidence that such

exposure will shape the epigenome or enhance brain function. What research has shown is that specific epigenetic modifications do occur in brain cells as cognitive skills like learning and memory develop and that repeated activation of brain circuits dedicated to learning and memory through interaction with the environment, such as reciprocal "serve and return" interaction with adults,54 facilitates these positive epigenetic modifications. We also know

Adverse fetal and early childhood experiences can-and do-lead to physical and chemical changes in the brain that can last a lifetime.

that sound maternal and fetal nutrition, combined with positive social-emotional support of children through their family and community environments, will reduce the likelihood of negative epigenetic modifications that increase the risk of later physical and mental health impairments.

# The Science-Policy Gap

THE FACT THAT THE GENOME IS VULNERABLE TO modification by toxic stress, nutritional problems, and other negative influences underscores the importance of providing supportive and nurturing experiences for young children in the earliest years, when brain development is most rapid. From a policy perspective, it is in society's interest to strengthen the foundations of healthy brain architecture in all young children to maximize the return on future investments in education, health, and workforce development. In this context, the epigenome is the chemical signature that explains how early life experiences become embedded in the circuitry of the developing brain and are associated with lifelong consequences. Research now shows that interaction between adverse environments and the genes we inherit—through the epigenome can increase the risk for long-term negative mental and physical health outcomes. Nevertheless, many policy decisions do not yet reflect this growing knowledge, which logically calls for reducing the exposure of pregnant women and young children to environments and experiences that can have significant negative effects on the epigenome (and therefore powerful, indirect influences on genes). This gap between what we know and what we do is illustrated by the following four examples:

Child welfare. Because threatening or harmful environments can produce epigenetic modifications that affect a child adversely for a lifetime, the management of child abuse or neglect cases by child protective services is extremely time-sensitive. This sense of urgency is particularly important with respect to decisions about custody arrangements that involve non-family placements. The biology of adversity also tells us that assessment and planning for the mental health and broad developmental needs of vulnerable children-with the assurance of nurturing, protective, and stable relationships as the highest priority—require attention in the child welfare system comparable to the conventional focus placed primarily on physical safety.

Mandated maternal employment and public assistance. Whether low-income mothers with very young children should be required to work in order to be eligible for public assistance is a political decision. Policies informed by scientific knowledge about early brain development and epigenetics, however, would link mandated maternal employment to a parallel investment in high-quality early care and education programs for affected children. When policies view child care simply as a custodial service whose primary purpose is to facilitate maternal work outside the home, they reflect a lack of understanding of extensive scientific evidence about how the developing architecture of the brain is shaped by epigenetic influences associated with the quality of adult-child interactions in the early childhood years. In contrast, policies that view high-quality child care and early education programs as strategic interventions to improve the life prospects of children whose parents have limited education and low income are more likely to increase the prosperity of communities across generations.

Prenatal and newborn health care. The fetal period is a highly active time for organ development and epigenetic modification, yet investment in prenatal services remains uneven. Policies that assure the availability, accessibility, and affordability of individualized support and monitoring of all pregnancies create a safety net that prevents harm and detects problems at a point when appropriate interventions can reduce the negative consequences of toxic stress and other adverse environmental exposures.

**Support for new parents.** The United States is one of very few developed nations that does not provide some amount of paid family leave for all parents after the birth or adoption of a baby. For parents of newborns who do not have the economic resources to make ends meet in the absence of paid employment, the supportive relationships that promote positive epigenetic changes and help very young children to manage stress can be compromised by a premature return to work and the inability to secure highquality care for a young infant. Family leave is one way of helping parents build these critical relationships, but other policies that support parents during this important transition time can also have important, short-term effects on the quality of family life as well as long-term impacts that bring high returns to all of society.

# **Implications for Policy and Programs**

BECAUSE EARLY EXPERIENCES CAN ALTER THE epigenome and influence developing brain architecture, policies affecting the life circumstances of pregnant women and young children can have enormous implications for all of society. The varied effects of environments on the epigenome are evident from the time of early embryonic development and extend into the early childhood years. Science tells us that children can be helped to reach their full potential through both appropriate experiences in the earliest years and the reduction of sources of toxic stress that can alter the epigenome and increase the risk of long-term problems in physical and mental health. Thus, public policies that harness the basic principles of neuroscience and epigenetics to address the needs of young children are likely to also generate long-term benefits, such as healthier communities and a more prosperous society.

The documented effects of toxic stress on negative epigenetic adaptations demonstrate the urgent need to alleviate sources of significant adversity as early as possible in the lives of children who live in threatening environments. In order to be maximally effective, programs and services targeting the precipitants of excessive stress—such as child abuse and neglect, violent neighborhoods and families, and caregiver mental illness—must have prompt access to specific expertise in the target areas in order to move quickly to protect young children from epigenetic changes that can lead to lifelong problems

in learning, behavior, and health. Because the clock is always ticking when the basic architecture of the brain is developing, informed policymakers understand that a delayed response to the needs of young children who are experiencing significant adversity jeopardizes their individual well-being as well as the broader human capital needs of society.

Epigenetic changes caused by the exposure of pregnant women, infants, and toddlers to environmental toxins, prescription drugs, alcohol, and illicit substances require an urgent look at what safeguards can be implemented to prevent such exposures. Lead paint laws are a good example of public policy that has been successful in reducing the harmful consequences of environmental toxins. Less aggressive policies with respect to mercury and organophosphate insecticides, on the other hand, are just two examples of many missed opportunities to mitigate the well-documented, adverse effects of environmental hazards on pregnant women and young children. The serious and continuing impact of prenatal exposure to alcohol and a wide variety of chemical substances (including prescription drugs) on child health and development calls for a more vigorous approach to environmental policies and public education. In view of the well-established scientific fact that embryonic, fetal, and early childhood brain development is considerably more susceptible to damage from neurotoxins than the mature brain of an adult, the establishment of safe levels of exposure to toxic substances should be based on scientific data that recognize the critical link between vulnerability and age, and that focus primarily on the best information available for the youngest children.55

Because prenatal and early postnatal experiences can affect long-term outcomes through epigenetic influences, the provision of highquality health services and nutritional support for all pregnant women, infants, and toddlers would be likely to reduce preventable diseases across the lifespan-as well as the costly treatments for them. A range of currently available programs and systems has been established to meet these needs. Assuring access to appropriate, affordable, high-quality services that are well implemented, however, remains an elusive goal, particularly for many of the most

disadvantaged families and those whose primary language is not English. Policies that succeed in connecting all pregnant women and young children to medical and nutrition services that match their individualized needs will produce measurable benefits in population health.

Certain epigenetic changes in humans can be transferred across generations, thereby underscoring important, long-term implications for policies that affect the circumstances in which young children are raised. Effective policies and programs that address conditions associated with economic hardship with or without other sources of adversity—especially those targeted to help families during pregnancy—can not only improve birth outcomes and short-term conditions for young children but should also be viewed as investments in building a stronger foundation for healthy communities and future prosperity across generations. When policymakers support positive environments for pregnant women and very young children, they reduce the risk of intergenerational transfer of negative epigenetic changes that can lead to impaired health, diminished learning capacity, and poor parenting of the next generation.

Because discoveries about the epigenome and its lifelong effects are so recent, a multi-faceted education campaign could bring important new information to a wide range of important audiences, including health professionals, judges and lawyers, educators, caregivers, families, and the general public. A broad-based understanding of cutting-edge, developmental science by people who influence the experiences and environments of young children could provide a framework for greater alignment and integration of the current patchwork of services and supports provided to families. In this vein, greater understanding of how toxic stress, poor nutrition, and toxic chemical and drug exposures can increase lifelong risks for physical and mental health impairments by changing the chemistry of our children's DNA would provide a powerful foundation for more effective public action to address the needs of young children—and all of society—for generations to come.

## References

- Meaney, M. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, 81(1), 41–79.
- 2. Szyf, M. (2009a). Early life, the epigenome and human health. *Acta Paediatrica*, 98(7), 1082-1084.
- Szyf, M. (2009b). The early life environment and the epigenome. *Biochimica Biophysica Acta (BBA)*, 1790(9), 878–885
- Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature*, 150, 563-565.
- Dolinoy, D. C., Weidman, J. R., & Jirtle, R. L. (2007). Epigenetic gene regulation: Linking early developmental environment to adult disease. *Reproductive Toxicology*, 23(3): 297-307.
- Kaminsky, Z. A., Tang, T., Wang, S., Ptak, C., Oh, G. H. T., Wong, A. H. ... & Petronis, A.. (2009). DNA methylation profiles in monozygotic and dizygotic twins. *Nature Genetics*, 42, 240-245.
- Crews, D. (2008). Epigenetics and its implications for behavioral neuroendocrinology. Frontiers in Neuroendocrinology, 29(3), 344-357.
- 8. Dolinoy, D. C. & Jirtle, R. L. (2008). Environmental epigenomics in human health and disease. *Environmental and Molecular Mutagenesis*, 49(1), 4-8.
- Bernstein, B. E., Meissner, A., & Lander, E. S. (2007). The mammalian epigenome. *Cell*, 128(4), 669-81.
- Anway, M. D., Cupp, A. S., Uzumcu, M., & Skinner, M. K. (2005). Epigenetic transgenerational actions of endocrine disruptors and male fertility. *Science* 308, 1466-1469.
- Champagne, F. A. (2010). Epigenetic influences of social experiences across the lifespan. *Developmental Psychobiol*ogy, 1-13.
- Newbold, R. R., Padilla-Banks, E., & Jefferson, W. N. (2006) Adverse effects of the model environmental estrogen diethylstilbestrol are transmitted to subsequent generations. *Endocrinology*, 146, S11-S17.
- 13. Smith, L. T., Otterson, G. A., & Plass, C. (2007). Unraveling the epigenetic code of cancer for therapy. *Trends in Genetics*, 23(9), 449-456.
- Giacinti, L., Vici, P., & Lopez, M. (2008). Epigenome: A new target in cancer therapy. *Clinica Terapeutica*, 159(5), 347-360.
- Bagot, R. C., van Hasselt, F. N., Champagne, D. L., Meaney, M. J., Krugers, H. J., & Joels, M. (2009). Maternal care determines rapid effects of stress mediators on synaptic plasticity in adult rat hippocampal dentate gyrus. Neurobiology of Learning and Memory, 92(3), 292-300.
- Curley, J. P. (2009). Social enrichment during postnatal development induces transgenerational effects on emotional and reproductive behavior in mice. Frontiers in Behavioral Neuroscience, 3, 1-14.
- Sweatt, J. D. (2007). An atomic switch for memory. *Cell*, 129(1), 23-4.
- Sweatt, J. D. (2009). Experience-dependent epigenetic modifications in the central nervous system. *Biological Psychiatry*, 65(3), 191-7.
- McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonte, B., Szyf, M. ... & Meaney, M. J. (2009).

- Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience*, 12(3), 342-348.
- Roth, T. L., Lubin, F. D., Funk A., & Sweatt, J. (2009).
   Lasting epigenetic influence of early-life adversity on the BDNF gene. *Biological Psychiatry*, 65(9), 760-769.
- 21. Mellor, J., Dudek, P., & Clynes, D. (2008). A glimpse into the epigenetic landscape of gene regulation. *Current Opinion in Genetics & Development*, 18(2), 116-122.
- Isles, A. R. & Wilkinson, L. S. (2008). Epigenetics: What is it and why is it important to mental disease? *British Medi*cal Bulletin, 85(1), 35-45.
- Jirtle, R. L. (2008). Randy L. Jirtle, PhD: Epigenetics a window on gene dysregulation, disease. Interview by Bridget M. Kuehn. *Journal of the American Medical Association* (*JAMA*), 299(11): 1249-1250.
- 24. Nafee, T. M., Farrell, W. E., Carroll, W. D., Fryer, A. A., & Ismail, K. M. (2008). Epigenetic control of fetal gene expression. *BJOG: An International Journal of Obstetrics & Gynaecology*, 115(2), 158-168.
- Sinclair, D. A. & Oberdoerffer, P. (2009). The ageing epigenome: Damaged beyond repair? *Ageing Research Reviews*, 8(3), 189-198.
- Das, R., Hampton, D. D., & Jirtle, R. L. (2009). Imprinting evolution and human health. *Mammalian Genome*, 20(9-10), 563.
- 27. Bocock, P. N. & Aagaard-Tillery, K. M. (2009). Animal models of epigenetic inheritance. *Seminars in Reproductive Medicine*, 27(5), 369-79.
- Suter, M. A. & Aagaard-Tillery, K. M. (2009). Environmental influences on epigenetic profiles. Seminars in Reproductive Medicine, 27(5), 380-390.
- Swanson, J. M., Entringer, S., Buss, C., & Wadhwa, P. D. (2009). Developmental origins of health and disease: Environmental exposures. Seminars in Reproductive Medicine, 27(5), 391-402.
- 30. Levitt, P. (2003). Structural and functional maturation of the developing primate brain. *Journal of Pediatrics*, 143(4), 25, 45
- Miller, C. A., Campbell, S. L., & Sweatt, J. D. (2008). DNA methylation and histone acetylation work in concert to regulate memory formation and synaptic plasticity. *Neu*robiology of Learning and Memory, 89(4), 599-603.
- Shonkoff, J. P., Boyce, W. T., & McEwen, B S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA*, 301(21), 2252-2259.
- Meaney, M. J., Szyf, M., & Seckl, J. R. (2007). Epigenetic mechanisms of perinatal programming of hypothalamicpituitary-adrenal function and health. *Trends in Molecular Medicine*, 13(7), 269-277.
- Weaver, I. C. G., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., & Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neu*roscience, 7(8), 847-854.

- 35. Champagne, F. A. & Curley, J. P. (2009). Epigenetic mechanisms mediating the long-term effects of maternal care on development. Neuroscience and Biobehavioral Reviews, 33(4), 593-600.
- 36. Champagne, F. A., Weaver, I. C., Diorio, J., Dymov, S., Szyf, M., & Meaney, M. J. (2006). Maternal care associated with methylation of the estrogen receptor-alpha1b promoter and estrogen receptor-alpha expression in the medial preoptic area of female offspring. Endocrinology, 147(6), 2909-2915.
- 37. Chen, Y., Dube, C. M., Rice, C. J., & Baram, T. Z. (2008). Rapid loss of dendritic spines after stress involves derangement of spine dynamics by corticotropin-releasing hormone. The Journal of Neuroscience, 28(11), 2903-2911.
- 38. Moriceau, S. & Sullivan, R. M. (2006). Maternal presence serves as a switch between learning fear and attraction in infancy. Nature Neuroscience, 9, 1004-1006.
- 39. Rice, C. J., Sandman, C. A., Lenjavi, M. R., & Baram, T. Z. (2008). A novel mouse model for acute and long-lasting consequences of early life stress. Endocrinology, 149(10),
- 40. Thompson, J. V., Sullivan, R. M., & Wilson, D. A. (2008). Developmental emergence of fear learning corresponds with changes in amygdala synaptic plasticity. Brain Research, 1200, 58-65.
- 41. Bradley, R. G., Binder, E. B., Epstein, M. P., Tang, Y., Nair, H. P., Liu, W. ... & Ressler, K. J. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. Archives of General Psychiatry, 65(2), 190-200.
- 42. Gillespie, C. F., Bradley, B., Mercer, K., Smith, A., Conneely, K., Gapen, M. ... & Ressler, K. (2009). Trauma exposure and stress-related disorders in inner city primary care patients. General Hospital Psychiatry, 31(6), 505-514.
- 43. Hovens, J. G., Wiersma, J. E., Giltay, E. J., van Oppen, P., Spinhoven, P., Penninx, B. W. & Zitman, F. G. (2009). Childhood life events and childhood trauma in adult patients with depressive, anxiety and comorbid disorders vs. controls. Acta Psychiatrica Scandinavica. Oct 30 [epub]
- 44. Jovanovic, T., Blanding, N. Q., Norrholm, S. D., Duncan, E., Bradley, B., & Ressler, K. J. (2009). Childhood abuse is associated with increased startle reactivity in adulthood. Depression and Anxiety, 26(11), 1018-1026.
- 45. Krupanidhi, S., Sedimbi, S. K., Vaishnav, G., Madhukar, S. S., & Sanjeevi, C. B. (2009). Diabetes-role of epigenetics, genetics, and physiological factors. Zhong Nan Da Xue Xue Bao Yi Xue Ban, 34(9), 837-845.
- 46. Quas, J. A., Carrick, N., Alkon, A., Goldstein, L., & Boyce, W. T. (2006). Children's memory for a mild stressor: The role of sympathetic activation and parasympathetic withdrawal. Developmental Psychobiology, 48(8), 686-702.
- 47. Weidman, J. R., Dolinoy, D. C., Murphy, S. K., & Jirtle, R. L. (2007). Cancer susceptibility: Epigenetic manifestation of environmental exposures. Cancer Journal, 13,(1), 9-16.
- 48. Wilson, A. G. (2008). Epigenetic regulation of gene expression in the inflammatory response and relevance to common diseases. Journal of Periodontology, 79(8): 1514-1519.
- 49. Dolinoy, D. C., Huang, D., & Jirtle, R. L. (2007). Maternal nutrient supplementation counteracts bisphenol

- A-induced DNA hypomethylation in early development. Proceedings of the National Academy of Sciences of the United States of America, 104(32), 13056-13061.
- 50. Waterland, R. A. & Jirtle, R. L. (2004). Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. Nutrition, 20(1), 63-68.
- 51. Waisberg, M., Joseph, P., Hale, B., & Beyersmann, D. (2003). Molecular and cellular mechanisms of cadmium carcinogenesis. Toxicology, 192(2-3), 95-117.
- 52. Salnikow, K. & Zhitkovich, A. (2008). Genetic and epigenetic mechanisms in metal carcinogenesis and cocarcinogenesis: nickel, arsenic and chromium. Chem. Res. Toxicol, 21, 28-44.
- 53. Szyf, M. (2009c). Epigenetics, DNA methylation, and chromatin modifying drugs. Annual Review of Pharmacology & Toxicology, 49, 243-263.
- 54. National Scientific Council on the Developing Child (2007). The timing and quality of early experiences combine to shape brain architecture: Working paper no. 5. http://www.developingchild.net
- 55. National Scientific Council on the Developing Child (2006). Early exposure to toxic substances damages brain architecture: Working paper no. 4. http://www. developingchild.net

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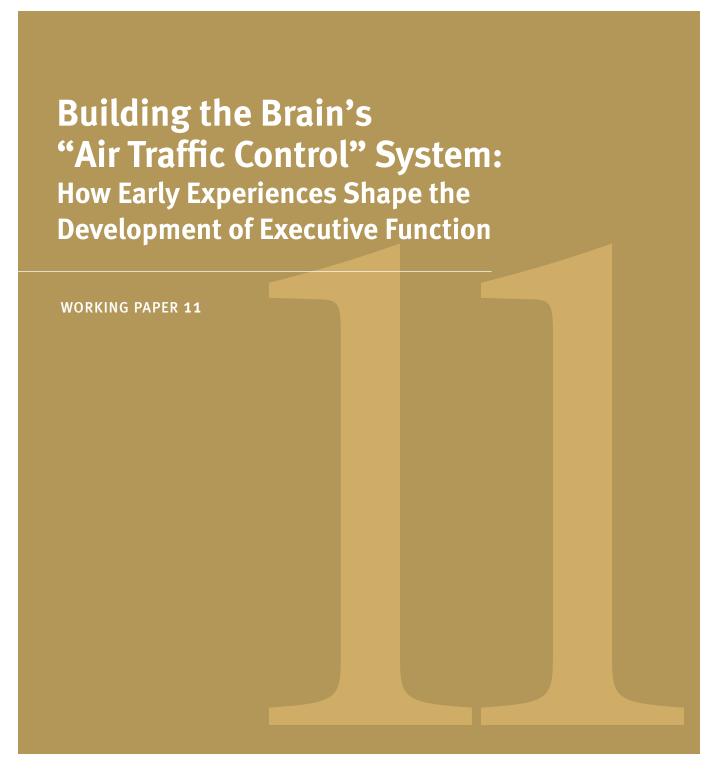
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# NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD NATIONAL FORUM ON EARLY CHILDHOOD POLICY AND PROGRAMS



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## The Issue

we prepare dinner while simultaneously helping our children with their homework and making notes about appointments we need to schedule for the week. We focus on our jobs when we need to and our families when they need us. We remember the phone number that our neighbor just gave us so we can write it down as soon as we find a pen. We take a deep breath, rather than honk, if the car in front of us fails to move immediately when the light turns green. As adults, our capacities to multitask, to display self-control, to follow multiple-step directions even when interrupted, and to stay focused on what we are doing despite ever-present distractions are what undergird the deliberate, intentional, goal-directed behavior that is required for daily life and success at work. And while there are cognitive limits to anyone's ability to multi-task effectively, we need and rely on these basic skills in all areas of our lives. Without them, we could not solve complicated problems and make decisions, persist at tedious but important tasks, make plans and adjust them when necessary, recognize and correct mistakes, control our impulsive behavior, or set goals and monitor our progress toward meeting them. Children need to develop these skills, too, in order to meet the many challenges they will face on the road to becoming productive, contributing members of their communities.

As essential as they are, we aren't born with the skills that enable us to control impulses, make plans, and stay focused. We are born with the potential to develop these capacities—or not-depending on our experiences during infancy, throughout childhood, and into adolescence. Our genes provide the blueprint, but the early environments in which children live leave a lasting signature on those genes. This signature influences how or whether that genetic potential is expressed in the brain circuits that underlie the executive function capacities children will rely on throughout their lives. (See Working Paper 10, "Early Experiences Can Alter Gene Expression and Affect Long-Term Development.") These skills develop through practice and are strengthened by the experiences through which they are applied and honed. Providing the support that children need to build these skills at home, in child care and preschool programs, and in other settings they experience regularly is one of society's most important responsibilities.

Being able to focus, hold, and work with information in mind, filter distractions, and switch gears is like having an air traffic control system at a busy airport to manage the arrivals and departures of dozens of planes on multiple runways. In the brain, this air traffic control mechanism is called executive function. This

refers to a group of skills that helps us to focus on multiple streams of information at the same time, monitor errors, make decisions in light of available information, revise plans as necessary, and resist the urge to let frustration lead to hasty actions. Acquiring the early building blocks of these skills is one of the most important and challenging tasks of the early childhood years, and the opportunity to build further on these rudimentary capacities is critical to healthy

Having executive function in the brain is like having an air traffic control system at a busy airport to manage the arrivals and departures of dozens of planes on multiple runways.

development through middle childhood and adolescence. Just as we rely on our well-developed personal "air traffic control system" to make it through our complex days without stumbling, young children depend on their emerging executive function skills to help them as they learn to read and write, remember the steps in performing an arithmetic problem, take part in class discussions or group projects, and

# **What Are Executive Functions?**

Completing most tasks requires the successful orchestration of several types of executive function skills. Among scientists who study these functions, three dimensions are frequently highlighted: Working Memory, Inhibitory Control, and Cognitive or Mental **Flexibility**.<sup>1,2,3</sup> In most real-life situations, these three functions are not entirely distinct, but, rather, they work together to produce competent executive functioning.

WORKING MEMORY is the capacity to hold and manipulate information in our heads over short periods of time. It provides a mental surface on which we can place important information so that it is ready to use in the course of our everyday lives. It enables us to remember a phone number long enough to dial it, to return to our place in a magazine article before a friend interrupted us, and to recall whether we had added the salt to what we were cooking before we had to help our child find a missing shoe. It enables children to remember and connect information from one paragraph to the next, to perform an arithmetic problem with several steps, to keep track of the moves and make a logical next step in a game of checkers, and to follow multiple-step instructions without reminders ("go to your cubbies, put away your storybooks, bring back your arithmetic books, and open them to page 30"). It also helps children with social interactions, such as planning and acting out a skit, taking turns in group activities, or easily rejoining a game after stepping away to get a drink of water.

**INHIBITORY CONTROL** is the skill we use to master and filter our thoughts and impulses so we can resist temptations, distractions, and habits and to pause and think before we act. It makes possible selective, focused, and sustained attention, prioritization, and action. This capacity keeps us from acting as completely impulsive creatures who do whatever comes into our minds. It is the skill we call on to push aside daydreams about what we would rather be doing so we can focus on important tasks. It is the skill we rely on to help us "bite our tongue" and say something nice, and to control our emotions at the same time, even when we are angry, rushed, or frustrated. Children rely on this skill to wait until they are called on when they know the answer, to be good at games like "Simon Says" and "Red Light/Green Light," to stop themselves from yelling at or hitting a child who has inadvertently bumped into them, and to ignore distractions and stay on task in school.

**COGNITIVE OR MENTAL FLEXIBILITY** is the capacity to nimbly switch gears and adjust to changed demands, priorities, or perspectives. It is what enables us to apply different rules in different settings. We might say one thing to a co-worker privately, but something quite different in the public context of a staff meeting. If a friend asks if we like her new haircut and we don't, we are able to flexibly shift to the social convention that governs not hurting people's feelings. Likewise, we teach our children about "outside voices" and "inside voices" and the different situations in which they should use each. As the author of *The* Executive Brain, Elkhonon Goldberg, notes, "The ability to stay on track is an asset, but being 'dead in the track' is not." Stated differently, self-control and persistence are assets, rigidity is not. Cognitive flexibility enables us to catch mistakes and fix them, to revise ways of doing things in light of new information, to consider something from a fresh perspective, and to "think outside the box." If the "church in two blocks" where we were told to turn right is actually a school, we adjust and turn anyway. If we are missing a recipe ingredient, we call a neighbor or make a substitution. Children deploy this skill to learn exceptions to rules of grammar, to approach a science experiment in different ways until they get it to work, or to try different strategies when they are working out a conflict with another child.

enter into and sustain play with other children. The increasingly competent executive functioning of childhood and adolescence enable children to plan and act in a way that makes them good students, classroom citizens, and friends. Children who do not have opportunities to use and strengthen these skills, and, therefore, fail to become proficient—or children who lack the capacity for proficiency because of disabilities or, for that matter, adults who lose it due to brain injury or old age—have a very hard time managing the routine tasks of daily life. Studying, sustaining friendships, holding down a job, or managing a crisis pose even bigger challenges.

The process of development is sometimes portrayed as one in which children gradually manage more and more aspects of their environments and lives on their own. We would not trust two-year-olds to stop going after a ball just because it rolled into the street, get ready in the morning (brush their teeth, pick out their clothes, and get dressed) by themselves, or even clean up their toys without reminders. Adults set up the framework (i.e., establishing routines, providing cues, breaking big tasks into smaller chunks) that helps children use the executive function skills they are developing to the best of their abilities. We call these techniques "scaffolding." Just as a scaffold supports workers while a building is being erected, adults can use these activities to support the emergence of children's executive function skills until they can practice and perform them on their own. And, just as construction workers remove the scaffolding when the building itself can support them, over time we can reduce scaffolding activities and allow children to organize themselves and get their tasks done without constant reminders and direction.

Elementary school teachers are keenly aware of executive function skills. It is often within the group setting of a classroom and the demands of schoolwork that delays or deficits in the development of age-expected executive function skills are first noted. Teachers identify problems with paying attention, managing emotions, completing tasks, and communicating wants and needs verbally as major determinants of whether a child is ready to succeed in the school setting. In many ways, coming to school with a solid base of these foundational executive function skills is more important than whether children know their letters and numbers.5,6 Imagine a classroom of first graders in which some children are unable to control their impulses, wait their turn, stay focused on their work, or remember instructions. Even when only a couple of the children have underdeveloped executive function abilities, an entire classroom can become disorganized, and precious time will be diverted from productive learning activities. This can have a profound impact on the overall climate of the classroom

# Executive function skills are crucial building blocks for the early development of both cognitive and social capacities.

and is often reported by teachers as a source of exasperation and burnout.7

The scientific evidence on the development and consequences of executive functioning in the earliest years of life conveys three important messages. First, executive function skills are crucial building blocks for the early development of both cognitive and social capacities. Second, both normative differences in the nature and pace of individual developmental trajectories and the impacts of significant adversity will affect how the development of executive functioning will unfold for any given child. Third, several interventions focused on supporting the development of specific executive function skills have demonstrated at least short-term effectiveness, with evidence also emerging that they may have impacts on other aspects of learning as well.

Executive functions underlie a broad range of large and small (as well as complicated and straightforward) life skills, competencies, and behaviors. The lifelong importance of these skills and their effect on learning makes it clear that parents, practitioners, and policymakers alike need to be aware of what we now understand about the development of executive function skills, the experiences that foster the healthy emergence of these skills, and the conditions that appear to undermine them.

# What Neuroscience and Developmental Research Tell Us

The building blocks of children's capacities to retain and use new information, focus attention, control impulses, and make plans are acquired during early childhood, but the full range of executive function skills continues to develop into the adolescent years. The rudimentary signs of these capacities emerge toward the end of the first year of life.8,9,10,11,12 By age three, most children can organize themselves to complete tasks that involve following two rules (e.g., "If it's red, put it here, but if it's blue, put it there."), thus showing that they can direct and re-direct their attention to make deliberate choices (mental flexibility), maintain focus in the face of distractions (inhibitory control), and hold rules "on line" mentally as they figure things out (working memory). While we can see clear evidence that these capacities are developing in the three-year-old, they remain relatively limited. The five-year-old mind, by contrast, is remarkably complex. Older preschoolers are capable of conscious problem-solving that involves the ability to shift their attention from one rule to another that is incompatible with the first, and then back again (e.g., "If it's the color game, put the red square here, but if it's the shape game, put the red square there."). They also have the capacity to inhibit responses that are inappropriate even if they are highly desirable (e.g., "I want to eat the candy right now, but I'll wait, because I will get more candy later if I do.") or habitual (e.g., "I've been sorting by color for five minutes, but now I need to shift to the shape rule."), and to execute multi-step, deliberate plans (e.g., "To stack these balls in the right order with just three moves, I need to start here, do this next, and then do that."). A more familiar demonstration of this remarkable development can be seen in the growing proficiency with which young children play "Simon Says" and "Red Light/Green Light." At age 5, these skills are just emerging and still require considerable practice. They are also heavily dependent on the situation and a child's experience with it, and there are large individual differences in children's capacity to deploy these evolving skills. By age 7, some of the capabilities and brain circuits underlying executive function skills are remarkably similar to those found in adults.3 Once these

foundational capacities for directing attention, keeping rules in mind, controlling impulses, and enacting plans are in place, the subsequent developmental tasks of refining them and learning to deploy them more efficiently can proceed into the adolescent and early adult years as tasks grow increasingly complicated and challenging.

Scientists are making major strides in identifying the key brain regions on whose development the healthy emergence of these executive function skills depends. These include circuits and systems that primarily involve the prefrontal cortex, but also include the anterior cingulate, parietal cortex, and hippocampus. The gradual acquisition of executive function skills corresponds closely to the extended development of these prefrontal brain regions from infancy through late adolescence.8,13,14,15 The formative development of these regions occurs during early childhood, as the relevant circuits emerge, mature, and forge critical interconnections. These circuits are then refined and made more efficient during adolescence and into the early adult years. It is also important to note that the brain regions and circuits associated with executive functioning have extensive interconnections with deeper brain structures that control the developing child's responses to threat and stress.16,17,18 This implies that the developing executive functioning system both influences and is affected by the young child's experience and management of threat, stress,10,19 and strong emotions. Thus, extended exposure to threatening situations can compromise the development and deployment of executive function skills, yet well-developed capabilities in these areas can also help children (and adults) manage stress effectively.

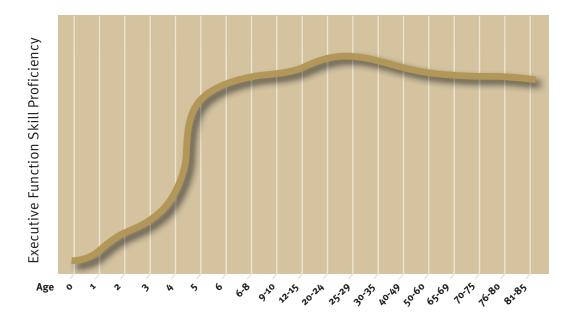
**Executive functioning is distinct from (yet foun-dational to) school readiness and academic success.** Scientists who study executive function skills refer to them as the biological foundation for school readiness.<sup>20,21</sup> They argue that strong working memory, cognitive self-control, and attentional skills provide the basis upon which children's abilities to learn to read, write, and do math can be built. In practice, these skills

support the process (i.e., the how) of learning focusing, remembering, planning—that enables children to effectively and efficiently master the content (i.e., the what) of learning-reading, writing, computation. They enable children to acquire knowledge and to participate in the school experience as actively engaged and competent learners. Children with stronger working memory, inhibition, and attentional skills also have been found to make larger gains on tests of early math, language, and literacy development during the preschool years than their peers with weaker executive function skills.<sup>22,23,24,25,26,27,28,29</sup> Moreover, the working memory and attention control of economically disadvantaged children at the beginning of preschool predicted kindergarten math and reading achievement over and above the contribution of earlier math and reading ability.30 Similarly, children whose behavior was well-regulated and who demonstrated academic engagement at the beginning of the Head Start year scored higher on tests of early reading

and language skills in first grade, compared with children who demonstrated less well-developed executive functioning.31

Children's executive function skills provide the link between early school achievement and social, emotional, and moral development. Executive function skills are considered to be a common denominator for both learning and social interaction. Young children who have problems staying focused and resisting urges to respond impulsively-two core executive function skills—not only have trouble in school but also have trouble following directions generally and are at elevated risk of displaying aggressive and confrontational behavior with adults and other children. 32,33,34 Executive functions like completing tasks, solving problems, organizing information, and making (and revising, if necessary) deliberate plans, are important facilitators of interpersonal interactions and behavior. Indeed, some researchers have hypothesized that

## **Executive Function Skills Build Throughout Childhood and Adolescence**



A range of tests measuring different forms of executive function skills indicates that they begin to develop shortly after birth, with ages 3 to 5 providing a window of opportunity for dramatic growth in these skills. Growth continues throughout adolescence and early adulthood; proficiency begins to decline in later life.

Source: Weintraub et al. (In Press).99

the complexity of human social relationships, rather than the need to do higher math, is why the human prefrontal cortex is so large and our executive function abilities are so advanced.<sup>20,35</sup>

Children's social play is believed to be an important practice ground for the development of executive function skills. Partly, this is because children need to test for themselves the skills that adults have been scaffolding for them. For example, they have to come up with the plan for playing house, communicate with each other about role assignments and then remember that Susie will be the bossy older sister, Ralph will play the dog, and Jackie will be the baby. In this scenario, keeping track of what each actor has done and inserting a new piece of the story that makes sense to everyone requires the effective exercise of emerging executive function skills. The child who cannot demonstrate sufficient executive competence either is told what to do by others, gets pushed out, or causes the play to fall apart. As toddlers, children can barely manage to coordinate play with one other child; by the time they enter first grade, they typically can play cooperatively with several children simultaneously and can work on projects that span hours or even days. Children who lag behind in their emerging executive function capacities relative to their age mates find themselves at a disadvantage, because they cannot keep up with the complexity of the play and, therefore, get frustrated, act out, and may cause other children to not want to play with them.<sup>36</sup> Thus, the skills that help children master many academic tasks are the same as those that help them get along with their peers and be viewed as good classroom citizens.

Large individual differences in executive functioning at kindergarten entry can have important implications for children's adjustment and success in and out of school as well as in their relationships with others. Children enter school with distinct profiles of strengths and weaknesses in executive function skills. 21,37,38 As both teachers and parents know, young children differ widely in how well they are able to adjust their attention, control impulses, follow rules and directions, and adapt to other demands of their environments. Some children have less well-developed executive functioning and are less able to orchestrate their capacities. Children with special needs, such as those

associated with autism, for example, or reading disabilities,<sup>39</sup> may have particular difficulty with executive functioning demands. A child's temperament can also make this orchestration more challenging, as illustrated by individuals who typically react more rapidly and intensely (with either anger and frustration or exuberance) to their experiences. 9,40 Another example of the marked variability in developing skills is the observation that some children can be highly capable in focusing their attention and managing distractions, but have less well-developed working memory capacity. Understanding these individual differences can help adults figure out how much support and structure to provide as children develop and learn. A new generation of educational interventions is available to address this challenge by working with classrooms of children who span the broad spectrum of executive function capacities. (See "What Evaluation Research Tells Us," below.)

A young child's environment of relationships plays an important role in the development of **executive capacities.** Environments that foster executive functioning are characterized by adult-child relationships (both within and outside the home) that guide children from complete dependence on adult support to gradual assumption of the "executive" role for themselves. Such environments neither expect children to have more advanced skills than are reasonable for their age, nor do they treat them as if they had no executive capabilities. Growth-promoting environments provide substantial "scaffolding" to help young children practice emerging skills before they are expected to perform them on their own. Enhancing the development of executive functioning involves sensitive, responsive caregiving and individualized teaching in the context of situations that require making choices, opportunities for children to direct their own activities with decreasing adult supervision over time, effective support of early emotion regulation, promotion of sustained joint attention, and the availability of adults who are not under such pressure that they cannot make time for children to practice their skills. 34,41,42,43,44,45

Children who routinely experience social interactions that provide these kinds of opportunities are more capable of resisting distractions, controlling their behavior and emotions towards others, complying with adult requests

and rules, and engaging in goal-directed behavior by the time they get to school. Experts also hypothesize that more ordered and predictable environments foster the development of executive function skills by offering children ample experiences that involve give-and-take interactions with others. 20,46,47,48

Adverse environments resulting from neglect, abuse, and/or exposure to violence can impair the development of executive function skills as a result of the disruptive effects of toxic stress on the developing architecture of the brain. Chaotic (and thus, from the child's standpoint, unpredictable) environments can also lead to poor self-regulatory behaviors and impulse control.<sup>104</sup> A number of studies have shown that exposure to highly stressful early environments is associated with deficits in the development of children's working memory, attention, and inhibitory control skills. 45,49,50 Damaging fear and toxic stress<sup>51,52,53,54</sup> are likely mechanisms that explain these effects, in part, because they affect the chemistry of brain circuits involved in the development of these capacities, and they impair the specific neuronal architecture that is engaged when we try to keep information in working memory, inhibit a habitual action, or address problems in a flexible manner. All adults have had the experience of encountering a threat, being gripped by fear or anxiety, and having trouble thinking. Under such circumstances, the brain goes into high "fight-or-flight" mode, and we have to calm ourselves down before we can mobilize our executive function skills to plan and execute a well-considered response. In adults and children, acute stress can even cause less-efficient prefrontal cortex activity, leading to a temporary "blip" in executive functioning. 105 Thus, chronic fear and anxiety associated with living in highly threatening, chaotic, or stressful environments can make it very difficult for young children to engage their executive abilities—even in situations (like school) where they may, in fact, be safe.55,56

Mounting evidence is revealing the roles played by community, school, and family contexts, as well as socioeconomic status, in the development of executive function skills. Children from lower (versus higher) socioeconomic backgrounds show poorer performance on tests of working memory, cognitive flexibility, and

inhibition, 57,58,59,60,61 as well as electrophysiological evidence of altered prefrontal functioning between ages 7 to 12.57 One reason that social class may be related to the development of executive function skills is that young children with greater access to resources experience environments that are more likely to contain features that protect and foster the development of these skills (e.g., scaffolding, responsive caregiving, order and predictability, and

Children who experience adversity at an early age are more likely to exhibit deficits in executive functioning, suggesting that these capacities are vulnerable to disruption early in the developmental process.

freedom from sustained threats.) This underscores the importance of efforts to improve children's early environments and experiences as a strategy for increasing the likelihood of positive developmental outcomes later.

Children who experience adversity at an early age are more likely to exhibit deficits in executive functioning, suggesting that these capacities are vulnerable to disruption early in the developmental process. Among the conditions that have been studied and found to affect the development of executive function skills are early abuse and neglect,62 orphanage rearing,63,64,65 prematurity and/or perinatal complications,66,67,68 and prenatal alcohol exposure.69 For example, children with a history of exposure to alcohol before birth have been found to display high levels of impulsivity and disorganization, along with evidence of impaired development of the prefrontal cortex.70 Evidence is also accumulating that childhood maltreatment disrupts the attention systems that affect how children notice, interpret, and respond to social interactions.71,72 Despite subsequent adoption, maltreated children who experienced unstable foster care placements have been found to perform poorly on tests of executive functioning,73 as well as to display oppositional behavior towards their adoptive parents. Furthermore, larger numbers of unique foster care placements have been found to predict lower scores on a wide range of neuropsychological executive functioning tests,74,75

suggesting that frequent changes in a child's primary caregiver may disrupt the development of these important skills. In light of this evidence, it is not surprising that children with a history of early social deprivation from being raised in an orphanage have also been found to perform significantly more poorly than their non-institutionalized peers on neuropsychological

tests of executive function skills.<sup>63,64,76</sup> While all young children benefit from experiences that scaffold executive functioning, those who exhibit problems in self-regulation need particularly sensitive adult support. When children have experienced serious trauma or abuse, special concern is always warranted.

### What Evaluation Research Tells Us

The healthy development of executive function skills can be supported with specialized practice and training. The same neuroplasticity that leaves executive functioning skills vulnerable to genetic and environmental disruption also presents the possibility of actively promoting the successful development of these skills. Thus, scientists and clinicians have begun to design and assess specific training programs aimed at helping young children who face difficulties with several aspects of executive functioning, particularly with attention and working memory. One laboratory-based approach that relies on computerized programs strengthens the neural circuits that control specific executive function skills through "staircase" training that adjusts task difficulty as a child's performance improves. 10,777,78,79,80,81,82 In addition to improving targeted skills (such as inhibitory control and attention set shifting), emerging evidence indicates that the benefits of these programs also extend to enhanced performance on tests of general intelligence. Selected measures of brain activity further suggest that these interventions have direct, beneficial effects on the ways in which the prefrontal cortex is developing; however, these effects have not yet been tested on preschool children.

Focused preschool interventions can also protect and enhance executive functioning. Recent evaluations of a range of preschool interventions designed to strengthen children's capacities to use these executive function skills in the classroom (in contrast to programs focused primarily on cognitive training) are also demonstrating that these skills are open to improvement during the early childhood years. These interventions tend to adopt one of three strategies: (1) programs aimed explicitly at fostering emerging executive

function skills (e.g., the capacities to retain and use information, focus and resist distractions, and plan actions and revise plans as needed);83,84 (2) programs that train and support teachers in effective classroom management strategies (e.g., rewarding positive student behavior, redirecting negative behavior), supplemented with the assistance of a mental health consultant who helps with both overall classroom challenges and the needs of particular children;85,86,87 and (3) programs that train teachers to model and coach children as their social-emotional skills are developing, with the focus on children's prosocial behavior, social problem-solving skills, ability to understand and express emotions constructively, and ability to control impulsive behavior and organize themselves to accomplish goals.48,88,89 What these approaches all have in common is a focus on supporting self-control and effective, goal-oriented approaches to learning and social encounters.

The clearest example of the first strategy, known as Tools of the Mind, has been shown to improve the inhibitory skills of children from low-income families above and beyond what has been accomplished from standard classroom practices. 84,90,91 Evidence from randomized trials of the second and third approaches have documented significant effects on young children's engagement in academic tasks, attention skills, and control of impulsive behavior. 22,92,93 These interventions also had significant impacts on the quality of the teaching experienced by the children, including improved literacy environments, use of preventive behavior management, and overall more positive emotional climate. 83,94

A review of existing evaluation data on interventions focused on executive functioning reveals no evidence that one approach is superior to the others. Moreover, little is known about



# **Building the Foundations** of an "Air Traffic Control" **System in the Brain**

Executive function skills do not just appear in adulthood. They are built over time, starting as early as the first year of life, with more complex skills building on the simpler skills that came before. Executive function skills are also highly interrelated. Just as an air traffic control system requires the interaction of multiple people—pilots, navigators, controllers, weather forecasters—our human executive functioning system requires that each type of skill utilize elements of the others. For example, it takes working memory to hold two rules in mind and inhibitory control to ignore one of the rules in order to flexibly switch between rules as they change. This table presents examples of how these interrelated executive function skills develop when children have the proper scaffolding by adult caregivers.

### **WORKING MEMORY**

**ADULT** Can remember multiple tasks, rules, and strategies that may vary by situation

**5-16 YEARS** Develops ability to search varying locations, remember where something was found, then explore other locations (e.g., a game of Concentration or hiding a penny under one of three cups)

**4-5 YEARS** Comprehends that appearance does not always equal reality (e.g., when given a sponge that looks like a rock)

**3 YEARS** Can hold in mind two rules (e.g., red goes here, blue goes there) and act on the basis of the rules

**9-10 MONTHS** Can execute simple means-to-ends tasks and two-step plans; also able to integrate looking one place and acting (e.g., reaching) at another place

**7-9 MONTHS** Develops ability to remember that unseen objects are still there (toy hidden under a cloth); learns to put two actions together in a sequence (remove cloth, grasp toy)

### INHIBITORY **CONTROL**

**ADULT** Consistent self-control; situationally appropriate responses (e.g., resists saying something socially inappropriate, resists "tit for tat" response)

10-18 YEARS Continues to develop self-control, such as flexibly switching between a central focus (such as riding a bike or driving) and peripheral stimuli that may or may not need attention (road signs and pedestrians vs. billboards and passing houses)

**7 YEARS** Children perform at adult levels on learning to ignore irrelevant, peripheral stimuli (such as a dot on the side of a screen) and focus on the central stimulus (such as a picture in the middle of the screen)

4-5 YEARS Reductions in perseveration (persisting with following a rule even when knowing that the rule has changed). Can delay eating a treat; also can begin to hold an arbitrary rule in mind and follow it to produce a response that differs from their natural instinct (sort colored cards by shape rather than color)

**9-11 MONTHS** Able to inhibit reaching straight for a visible but inaccessible reward, such as a toy on the other side of a window, and instead delay a moment to recognize the barrier and detour around it

**8-10 MONTHS** Begins to maintain focus despite distractions during brief delays in a task

**6 MONTHS** Rudimentary response inhibition (able to not touch something instructed not to touch)

### COGNITIVE **FLEXIBILITY**

**ADULT** Able to revise actions and plans in response to changing circumstances

13-18 YEARS Continued improvement in accuracy when switching focus and adapting to changing rules

10-12 YEARS Successfully adapts to changing rules, even along multiple dimensions (okay to shout on playground, not okay in school, okay sometimes in theater rehearsal)

2-5 YEARS Succeeds at shifting actions according to changing rules (e.g., takes shoes off at home, leaves on at school, puts on boots for rain)

**9-11 MONTHS** Develops ability to seek alternate methods to retrieve objects beyond directly reaching for what's in view

Sources: Best & Miller (2010)100; Diamond (1991a, 1991b, 2002, 2006).101,102,8,103

how these programs produce the benefits that they do. Interventions that include an explicit focus on executive function skills do not need to be implemented separately from those focused on instruction in early literacy and math abilities. Indeed, the complex interactions that occur among executive functioning, social competence, and academic skills in preschool classrooms underscore the likely value of blending interventions designed to strengthen working memory, inhibition, and attention control with curricula focused on early literacy and math skills.<sup>30</sup>

Improvements in executive functioning extend to young children's performance on measures of social skills and academic performance. In three randomized trials, children in classrooms that emphasized the improvement of executive function skills through a range of strategies showed improved performance on other developmental outcomes as well. In the first instance, children assigned to Tools of the Mind classrooms showed significant reductions in teacher-rated problem behavior<sup>83</sup> compared

with preschool children in classrooms that focused on literacy instruction without explicit attention to executive functioning. In the second instance, preschoolers who received instruction that included a focus on executive function skills (i.e., attention and impulsivity) showed significant improvements in these capacities compared with their peers who were enrolled in "usual practice" classrooms, which researchers attribute to the concurrent effects on their early literacy and math skills.92 Children who experienced a curriculum that combined support for executive function skills and an interactive reading program also performed better than their "usual practice" peers on tests of early literacy abilities,22 as well as on measures of emotional understanding and social problem-solving.48 While we cannot be certain that demonstrated improvements in children's abilities to direct their attention, control impulsive behavior, and stay focused on their schoolwork contributed to their academic gains, evidence is increasingly supporting this interpretation. 22,30,48,92,95

# **Correcting Popular Misconceptions of Science**

THE FACT THAT YOUNG CHILDREN HAVE A difficult time with self-control, planning, ignoring distractions, and adjusting to new demands is hardly news to the adults who care for them. It is not widely recognized, however, that these capacities do not automatically develop with maturity over time. Furthermore, it is even less well known that the developing brain circuitry related to these kinds of skills follows an extended timetable that begins in early childhood and continues past adolescence and that it provides the common foundation on which early learning and social skills are built. Based on this new understanding, the following common misconceptions about the development of executive function skills can be laid to rest.

Contrary to popular belief, learning to control impulses, pay attention, and retain information actively in one's memory does not happen automatically as children mature, and young children who have problems with these skills will not necessarily outgrow them. The evidence is clear

that, by 12 months of age, a child's experiences are helping to lay the foundation for the ongoing development of executive function skills. These early abilities to focus attention, control impulses, and hold information "on-line" in working memory appear to be easily disrupted by highly adverse early experiences or biological disruptions. Evidence also shows that early interventions aimed at improving these capacities before a child enters school can have beneficial impacts across a broad array of important outcomes.

Contrary to popular belief, young children who do not stay on task, lose control of their emotions, or are easily distracted are not "bad kids" who are being intentionally uncooperative and belligerent. Young children with compromised or delayed executive function skills can display very challenging behaviors for which they are often blamed. In most circumstances, however, it is the protracted development of the prefrontal cortex that is to "blame." Efforts to help affected children develop better executive function skills

and adjustments of the demands placed upon them to avoid overtaxing their capabilities are much more helpful than punishment for difficult behavior. Particularly when adverse experiences or environments elicit a toxic stress response,96 it can be very difficult for even the most competent children to enlist whatever executive function skills they have. In these circumstances, the provision of a safe and predictable environment offers the sense of security needed for successful behavior change to occur.

Contrary to the theory that guides some early education programs that focus solely on teaching letters and numbers, explicit efforts to foster executive functioning have positive influences on instilling early literacy and numeracy **skills.** Early evidence from randomized trials of interventions designed to foster the cluster of executive function skills (working memory, attention, inhibitory control, etc.) indicates benefits in early literacy and math skills compared with children who experience "regular" classroom activities. 48,92 Indeed, there is also evidence that emerging executive function skills contribute to early reading and math achievement during the pre-kindergarten years and into kindergarten.<sup>28,30</sup> This is not surprising insofar as the acquisition of traditional academic skills depends on a child's capacity to follow and remember classroom rules, control emotions,

focus attention, sit still, and learn on demand through listening and watching. Neuroscientists are also beginning to relate specific aspects of executive functioning, notably attentional skills, to specific steps involved in learning to read and to work with numbers.9

It is important to emphasize that this research

Early education policies that emphasize literacy instruction alone are missing an important opportunity to increase their effectiveness by including attention to the development of executive functioning skills.

is in its infancy, and much remains to be learned. Not only do we need to understand the effectiveness factors that account for the emerging impacts on school readiness from interventions designed to focus on executive function skills, but we also need to examine whether effective early education programs that focus directly on social, numeracy, and language skills also have positive impacts on executive functioning. Thus, the highly interrelated nature of these capacities makes it difficult to label any single intervention as focused explicitly (or not) on the critical domains of executive functioning.

# The Science-Policy Gap

AS ADULTS, MOST OF US DEPLOY EXECUTIVE functioning automatically (and virtually unconsciously) in our everyday lives, as we strive to accomplish both short-term tasks and longer-term goals. Yet, a growing body of evidence shows that acquiring this bundle of skills and putting those skills to work in a variety of roles, such as friend, family member, and student, is highly challenging for many young children—and may explain many disparities in later school achievement. Despite this mounting evidence, little attention has been paid to the development and implementation of strategies to identify children who are at risk for poor executive functioning and to provide supports for them, their families, and the other adults

who care for them. This gap between what we know and what we do is illustrated by the following three examples.

Early education policies that emphasize literacy instruction alone are missing an important opportunity to increase their effectiveness by including attention to the development of executive function skills. Emerging evidence from early intervention programs explicitly aimed at fostering these skills indicates that beneficial effects on components of executive function (e.g., attention, working memory) also have positive secondary impacts, such as improving the quality of teaching that children receive (including improved literacy environments) and the promotion of other facets of early learning, including task engagement and reading skills. Indeed, the most effective early education programs of the future are likely to teach preschool curriculum content (e.g., early literacy, math, social skills) in a way that optimizes the scaffolding and practice of executive function skills.

The expulsion of young children from prekindergarten programs<sup>97,98</sup> because of unmanageable behavior illustrates the need for greater availability of expertise and resources to improve the executive function skills of vulnerable young **children.** Research shows that young children who have problems staying focused and controlling impulsive, reactive behavior—two core executive function skills—are at elevated risk of experiencing behavior problems. Evidence from kindergarten teachers who rank self-control and sustained attention as more critical for school readiness than content knowledge6 further highlights the importance of supporting the early development of executive function skills as a critical prerequisite to a successful transition into school. The extent to which the combination of serious self-regulation problems in young children and excessive stress experienced by early childhood teachers leads to a greater likelihood of expulsion from preschool

programs underscores the need for greater attention to innovative interventions that promote more adaptive behavior.

The lack of services that directly address sources of toxic stress during the earliest years of life indicates a disconnect between policies and the known vulnerability of many aspects of brain development (including executive function skills) to the effects of early adversity and the need for preventive policies to reduce such lost opportunities. It is widely understood that biologically based sources of vulnerability, (e.g., prematurity and other medical complications at birth) and disruptions in brain architecture related to difficulties in early rearing conditions (e.g., child abuse and neglect) place children at tremendous risk for developmental problems ranging from attachment disorders to learning disabilities. Emerging evidence also shows that these adverse conditions, as well as low socioeconomic status, place children at a disadvantage with regard to the development of working memory, cognitive flexibility, and behavioral inhibition. This connection between toxic stress and executive functioning suggests new and important opportunities for interventions focused on these skills to improve the likelihood of success in school and later life for children facing adversity.

# Implications for Policy and Programs

THE STATE OF SCIENTIFIC KNOWLEDGE ABOUT the development of executive function skills is sufficiently mature to support a number of evidence-based implications for those who develop and implement policies and programs that affect the health and well-being of young children. The following observations warrant particularly careful consideration by policymakers.

Given the importance of young children's executive function skills and emerging evidence that these capacities can be improved through focused early intervention programs, efforts to support the development of these skills deserve much greater attention in the design of early care and education programs. In recent years, a growing body of sophisticated intervention and evaluation research has documented positive

short-term impacts for programs aimed explicitly at strengthening young children's executive function skills, including working memory and attentional capacities. Concurrently, evidence from neuroscience is beginning to demonstrate specific changes in the brain that accompany improvements in these skills. Successful interventions have been implemented in a variety of programs that serve low-income children, including Head Start. Although additional replications and evaluations will add important information, the current evidence base is strong enough to warrant systematic, scaled-up initiatives to teach executive function skills in early care and education programs that focus on vulnerable populations in conjunction with evidence-based curricula that promote early literacy and numeracy skills.

Early care and education professionals—as well as kindergarten and early elementary teacherswould be better equipped to understand and address behavioral and learning challenges in their classrooms if they had professional training in (and easy-to-use tools for) the development of executive function skills. Teacher training programs, including degree programs in schools of education, currently devote little or no time to instruction about the development of executive function skills. Yet, teachers of young children are often the first to recognize serious problems with controlling impulses, focusing attention, staying organized, and following instructions that require well-developed working memory. The consequences of mislabeling these problems as "bad behavior" can be severe, leading, in some instances, to expulsion or inappropriate use of medication and, in others, to a highly disrupted classroom. In simple terms, many young children who need assistance are not getting it. Teachers equipped with knowledge and curriculum tools to support the development of executive function skills would be in a better position to calibrate their expectations to the developing capabilities of young children and better prepared to address these classroom challenges appropriately. They would also be in a better position to call upon specialized mental health consultation, when needed.<sup>52</sup>

Parents would benefit from greater access to tools and approaches that provide useful knowledge and ways of supporting the early development of executive function skills. Lessons learned from interventions that have proven successful in fostering executive functioning in young children hold considerable promise for incorporation into parentfocused interventions, such as home visiting, parenting education, and family support programs. The translation of effective management

Adding assessments of executive function skills to the repertoire of evaluation tools used in early childhood programs would not only provide important data for program planning but would also encourage attention to this critical domain of skill development.

strategies for use in these programs should be a high priority.

Adding assessments of executive function skills to the repertoire of evaluation tools used in early childhood programs would not only provide important data for program planning but would also encourage attention to this critical domain of skill development. Policymakers and practitioners measure what they value. The growing availability of valid and practical tools for assessing early executive functioning now makes it feasible for program evaluators to measure impacts on these foundational skills. As attention to executive functioning increases in the early childhood arena, this critical domain of development will receive more prominent attention in the public debate about how best to promote the emerging competencies of young children.

### References

- 1. Diamond, A., & Taylor, C. (1996). Development of an aspect of executive control: Development of the abilities to remember what I said and to "Do as I say, not as I do." Developmental Psychobiology, 29(4), 315-334.
- 2. Greenberg, M.T., Riggs, N. R. & Blair, C. (2007). The role of preventive interventions in enhancing neurocognitive functioning and promoting competence in adolescence. In D. Romer & E. F. Walker (Eds.), Adolescent psychopathology and the developing brain: Integrating brain and prevention science (pp. 441-461). New York: Oxford University Press.
- 3. Rothbart, M.K., Posner, M.I., & Kieras, J. (2006). Temperament, attention and the development of self-regulation. In K. McCartney & D. Phillips (Eds.), The Blackwell handbook of early child development (pp. 328-357). Malden, MA: Blackwell Press.
- 4. Goldberg, E. (2001). The executive brain: Frontal lobes and the civilized mind. New York: Oxford University Press.
- 5. Lewitt, E. M. & Baker, L. S. (1995). School readiness. The Future of Children, 5(2), 128-139.

# NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD NATIONAL FORUM ON EARLY CHILDHOOD POLICY AND PROGRAMS

- Rimm-Kaufman, S. E., Pianta, R. C., & Cox, M. J. (2000). Teachers' judgments of problems in the transition to kindergarten. *Early Childhood Research Quarterly*, 15(2), 147-166
- Brouwers, A., & Tomic, W. (2000). A longitudinal study of teacher burnout and perceived self-efficacy in classroom management. *Teaching and Teacher Education*, 16(2), 239-253
- 8. Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry. In D.T. Stuss & R.T. Knight (Eds.), *Principles of frontal lobe function* (pp. 466-503). New York: Oxford University Press.
- Posner, M.I. & Rothbart, M.K. (2006). Educating the human brain (1st Ed.). Washington, DC: American Psychological Association.
- Rueda, M.R., Posner, M.I., & Rothbart, M.K. (2005). The development of executive attention: Contributions to the emergence of self-regulation. *Developmental Neuropsy*chology, 28(2), 573-594.
- Zelazo, P.D. (2004). The development of conscious control in childhood. *Trends in Cognitive Sciences*, 8(1), 12-17.
- Zelazo, P.D. Carlson, S.M., & Kesek, A. (2008). The development of executive function in childhood. In C.A. Nelson & M. Luciana (Eds.), *Handbook of developmental* cognitive neuroscience (2<sup>nd</sup> Ed.). (pp. 553-574). Cambridge, MA: The MIT Press.
- Diamond, A. (1988). Abilities and neural mechanisms underlying AB performance. Child Development, 59(2), 523-527.
- 14. Goldman-Rakic, P.S. (1987). Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In F. Plum (Ed.), Handbooks of physiology: A spectrum of physiological knowledge and concepts: Section 1: Nervous system: Vol. V, 2 parts: Higher functions of the brain (pp. 373-417). Bethesda, MD: American Physiological Society.
- 15. Rothbart, M.K. & Posner, M.I. (2005). Genes and experience in the development of executive attention and effortful control. In L.A. Jensen & R.W. Larson (Eds.), New horizons in developmental theory and research (pp. 101-108). San Francisco: Jossey-Bass.
- Bush, B., Luu, P., & Posner, M.I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends* in Cognitive Sciences, 4(6), 215-222.
- Drevets, W. C., & Raichle, M. E. (1998). Reciprocal suppression of regional cerebral blood flow during emotional versus higher cognitive processes: Implications for interactions between emotion and cognition. *Cognition and Emotion*, 12(3), 353-385.
- Kuhl, J., & Kazén, M. (1999). Volitional facilitation of difficult intentions: Joint activation of intention memory and positive affect removes Stroop interference. *Journal of Experimental Psychology: General*, 128(3), 382-399.
- Blair, C., Zelazo, P.D. & Greenberg, M.T. (2005). The measurement of executive function in early childhood. *Developmental Neuropsychology*, 28(2), 561-571.
- Barkley, R.A. (2001). The executive functions and selfregulation: An evolutionary neuropsychological perspective. Neuropsychology Review, 11(1), 1-29.
- Blair, C. (2002). School readiness: Integrating cognition and emotion in a neurobiological conceptualization of children's functioning at school entry. *American Psycholo*gist, 57(2), 111-127.

- Bierman, K.L., Nix, R.L., Greenberg, M.T., Blair, C. & Domitrovich, C.E. (2008). Executive functions and school readiness intervention: Impact, moderation, and mediation in the Head Start REDI program. *Development and Psychopathology*, 20(3), 821-843.
- Blair, C., & Razza, R.P. (2007). Relating effortful control, executive function, and false belief understanding to emerging math and literacy ability in kindergarten. *Child Development*, 78(2), 647-663.
- Espey, K., McDiarmid, M., Kwik, M., Stalets, M., Hamby, A., & Senn, T. (2004). The contribution of executive functions to emergent mathematic skills in preschool children. *Developmental Neuropsychology*, 26(1), 465-486.
- Fuchs, L.S., Compton, D.S., Fuchs, D., Paulsen, K., Bryant, J.D. & Hamlett, C.L. (2005). The prevention, identification, and cognitive determinants of math difficulty. *Journal of Educational Psychology*, 97(3), 493-513.
- Gathercole, S.E., Tiffany, C., Briscoe, J., Thorn, A. & the ALSPAC Team. (2005). Developmental consequences of poor phonological short-term memory function in childhood: A longitudinal study. *Journal of Child Psychology* and Psychiatry, 46(6), 598-611.
- Howse, R.B., Calkins, S.D., Anastopoulos, A.D., Keane, S.P., & Shelton, T.L. (2003). Regulatory contributors to children's kindergarten achievement. *Early Education and Development*, 14(1)101-119.
- McClelland, M.M., Cameron, C.E., Connor, C.M., Farris, C.L., Jewkes, A.M., & Morrison, F.J. (2007). Links between behavioral regulation and preschoolers' literacy, vocabulary, and math skills. *Developmental Psychology*, 43(4), 947-959.
- Sektnan, M., McClelland, M.M., Acock, A., & Morrison, F. (In Press). Relations between early family risk, children's behavioral regulation, and academic achievement. *Early Childhood Research Quarterly*.
- Welsh, J.A., Nix, R.L., Blair, C., Bierman, K.L. & Nelson, K.E. (2010). The development of cognitive skills and gains in academic school readiness for children from low-income families. *Journal of Educational Psychology*, 102(1), 43-53.
- Bulotsky-Shearer, R.J., Fantuzzo, J.W., Dominguez, X. & McDermott, P.A. (2009, April). Unique contribution of social-emotional classroom behavior to school readiness for low-income urban preschool children. Paper presented at the biennial meetings of the Society for Research in Child Development, Denver, Colo.
- Eisenberg, N., Fabes, R.A., Nyman, M., Bernzweig, J., & Pinuelas, A. (1994). The relations of emotionality and regulation to children's anger-related reactions. *Child Development*, 65(1), 109-128.
- Hill, A.L. Degnan, K.A. Calkins, S.D. & Keane, S.P. (2006).
   Profiles of externalizing behavior problems for boys and girls across preschool: The roles of emotion regulation and inattention. *Developmental Psychology*, 42(5), 913-928
- Kochanska, G., Murray, K., & Coy, K. C. (1997). Inhibitory control as a contributor to conscience in childhood: From toddler to early school age. *Child Development*, 68(2), 263-277.
- 35. Dunbar, R.I.M, & Shultz, S. (2007). Evolution in the social brain. *Science*, *317*(5843), 1344-1347.
- Diamantopoulou, S., Rydell, A.M., Thorell, L.B., & Bohlin, G. (2007). Impact of executive functioning and symptoms of attention deficit hyperactivity disorder on children's peer relations and school performance. *Developmental Neuropsychology*, 32(1), 521-542.

- 37. Fantuzzo, J., Bulotsky-Shearer, R., McDermott, P.A., Mc-Wayne, C., Frye, D., & Perlman, S. (2007). Investigation of dimensions of social-emotional classroom behavior and school readiness for low-income urban preschool children. School Psychology Review, 36(1), 44-62.
- 38. Raver, C.C., Garner, P., & Smith-Donald R. (2007). The roles of emotion regulation and emotion knowledge for children's academic readiness: Are the links causal? In R.C. Pianta, M.J. Cox, & K.L. Snow (Eds.), School readiness and the transition to kindergarten in the era of accountability (pp. 121-147). Baltimore, MD: Paul H. Brookes Publishing Co.
- 39. Swanson, H.L., & Jerman, O. (2007). The influence of working memory on reading growth in subgroups of children with reading disabilities. Journal of Experimental Child Psychology, 96(4), 249-283.
- 40. Rothbart, M. K., & Rueda, M. R. (2005). The development of effortful control. In U. Mayr, E. Awh, & S. Keele (Eds.), Developing individuality in the human brain: A tribute to Michael I. Posner (pp. 167-188). Washington, DC: American Psychological Association.
- 41. Blair, C. & Diamond, A. (2008). Biological processes in prevention and intervention: The promotion of self-regulation as a means of preventing school failure. Development and Psychopathology, 20(3), 899-911.
- 42. Goldsmith, D. F., & Rogoff, B. (1997). Mothers' and toddlers' coordinated joint focus of attention: Variations with maternal dysphoric symptoms. Developmental Psychology, 33(1), 113-119.
- 43. Kochanska, G., Murray, K., Jacques, T.Y., & Vandegeest, K. (1996). Inhibitory control of young children and its role in emerging internalization. Child Development, 67(2), 490-
- 44. Kochanska, G., & Knaack, A. (2003). Effortful control as a personality characteristic of young children: Antecedents, correlates, and consequences. Journal of Personality, 71(6), 1087-1112.
- 45. Lengua, L.J., Honorado, E. & Bush, N.R. (2007). Contextual risk and parenting as predictors of effortful control and social competence in preschool children. Journal of Applied Developmental Psychology, 28(1), 40-55.
- 46. Bodrova, E., & Leong, D. J. (2007). Play and early literacy: A vygotskian approach. In K. A. Roskos, & J. F. Christie (Eds.), Play and literacy in early childhood: Research from multiple perspectives (2nd ed.). (pp. 185-200). Mahwah, NJ: Lawrence Erlbaum Associates.
- 47. Bodrova, E. & Leong, D. (2005). Promoting student selfregulation in learning. Education Digest, 71(2), 54-57.
- 48. Bierman, K.L., Domitrovich, C.E., Nix, R.L., Gest, S.D. Welsh, J.A., Greenberg, M.T., Blair, C., Nelson, K.E, & Gill, S. (2008). Promoting academic and social-emotional school readiness: The Head Start REDI Program. Child Development, 79(6), 1802-1817.
- 49. Maughan, A. & Cicchetti, D. (2002). Impact of child maltreatment and interadult violence on children's emotion regulation abilities and socioemotional adjustment. Child Development, 73(5), 1525-42.
- 50. O'Connor, T.G., Rutter, M., Beckett, C., Keaveney, L., Kreppner, J.M. (2000). The effects of global severe privation on cognitive competence: Extension and longitudinal follow-up. Child Development, 71(2), 376-90.
- 51. National Scientific Council on the Developing Child. (2005). Excessive stress disrupts the architecture of the developing brain: Working paper no. 3. http://www.developingchild.net

- 52. National Scientific Council on the Developing Child. (2008). Mental health problems in early childhood can impair learning and behavior for life: Working paper no. 6. http://www.developingchild.net
- 53. Pollak, S.D., Cicchetti, D., and Klorman, R. (1998). Stress, memory, and emotion: Developmental considerations from the study of child maltreatment. Development and Psychopathology, 10(4), 811-828.
- 54. Sanchez, M.M., Ladd, C.O. & Plotsky, P.M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. Development and Psychopathology, 3(3), 419-49.
- 55. Liston, C., McEwen, B.S. & Casey, B.J. (2009). Psychosocial stress reversibly disrupts prefrontal processing and attentional control. Proceedings of the National Academy of Sciences, 106(3), 912-917.
- 56. Liston, C., Miller, M.M., Goldwater, D.S., Radley, J.J., Rocher, A.B., Hof, P.R., Morrison, J.H., & McEwen, B. (2006). Stress-induced alterations in prefrontal cortical dendritic morphology predict selective impairments in perceptual attentional set-shifting. The Journal of Neuroscience, 26(30), 7870-7874.
- 57. Kishiyama, M.M., Boyce, W.T., Jimenez, A.M, Perry, L.M., & Knight, R.T. (2009). Socioeconomic disparities affect prefrontal function in children. Journal of Cognitive Neuroscience, 21(6), 1106-1115.
- 58. Li-Grining, C.P. (2007). Effortful control among lowincome preschoolers in three cities: Stability, change, and individual differences. Developmental Psychology, 43(1),
- 59. Noble, K.G., McCandliss, B.D. & Farah, M.J. (2007). Socioeconomic gradients predict individual differences in neurocognitive abilities. Developmental Science, 10(4),
- 60. Noble, K.G., Norman, M.F. & Farah, M.J. (2005). Neurocognitive correlates of socioeconomic status in kindergarten children. Developmental Science, 8(1), 74-87.
- 61. Mezzacappa, E. (2004). Alerting, orienting, and executive attention: Developmental properties and sociodemographic correlates in an epidemiological sample of young, urban children. Child Development, 75(5), 1373-1386.
- 62. Sanchez, M.M., & Pollak, S.D. (2009). Socio-emotional development following early abuse and neglect: Challenges and insights from translational research. In M. de Haan & M.R. Gunnar (Eds.), Handbook of developmental social neuroscience. (pp. 497-520) New York: Guilford Press.
- 63. Bos, K., Fox, N., Zeanah, C.H., & Nelson, C.A. (2009). Effects of early psychosocial deprivation on the development of memory and executive function. Frontiers in Behavioral Neuroscience, 3(16). doi: 10.3389/neuro.08.016.2009
- 64. Colvert, E., Rutter, M. Kreppner, J., Beckett, C., Castle, J., Groothues, C. et al. (2008). Do theory of mind and executive function deficits underlie the adverse outcomes associated with profound early deprivation?: Findings from the English and Romanian Adoptees study. Journal of Abnormal Child Psychology, 36(7), 1057-1068.
- 65. Gunnar, M.R. (2000). Early adversity and the development of stress reactivity and regulation. In C.A. Nelson (Ed.), The effects of early adversity on neurobehavioral development: The Minnesota Symposia on Child Psychology: Volume 31 (pp. 163-200). Mahwah, NJ: Lawrence Erlbaum
- 66. Curtis, W.J., Lindeke, L.L, Georgieff, M.K., & Nelson, C.A. (2002). Neurobehavioral functioning in neonatal intensive care unit graduates in late childhood and early adolescence. Brain, 125, 1646-1659.

# NATIONAL SCIENTIFIC COUNCIL ON THE DEVELOPING CHILD NATIONAL FORUM ON EARLY CHILDHOOD POLICY AND PROGRAMS

- Feldman, R. (2009). The development of regulatory functions from birth to 5 years: Insights from premature infants. *Child Development*, 80(2), 544-561.
- Luciana, M., Lindeke, L., Georgieff, M., Mills, M., & Nelson, C. (1999). Neurobehavioral evidence for working-memory deficits in school-aged children with histories of prematurity. *Developmental Medicine and Child Neurology*, 41(8), 521-533.
- 69. Jacobson, S.W., & Jacobson, J.L. (2000). Teratogenic insult and neurobehavioral function in infancy and childhood. In C. A. Nelson (Ed.), *The effects of early adversity on neu*robehavioral development (pp. 61-113). Mahwah, NJ: Lawrence Erlbaum Associates.
- Olson, H.C., Streissguth, A.P., Sampson, P.O., Barr, H.M., Bookstein, F.L., and Theide, K. (1997). Association of prenatal alcohol exposure with behavioral and learning problems in early adolescence. *Journal of the American Acad*emy of Child and Adolescent Psychiatry, 36(9), 1187-94.
- Dodge, K.A., Pettit, G.S., Bates, J.E., & Valente, E. (1995). Social information-processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of Abnormal Psychology*, 104(4), 632-643.
- Pollak, S.D. and Tolley-Schell, S.A.(2003). Selective attention to facial emotion in physically abused children. *Journal of Abnormal Psychology*, 112(3), 323-338.
- Lewis, E. E., Dozier, M., Ackerman, J., & Sepulveda-Kozakowski, S. (2007). The effect of placement instability on adopted children's inhibitory control abilities and oppositional behavior. *Developmental Psychology*, 43(6), 1415–1427.
- Korkman, M., Kirk, U., & Kemp, S.L. (1998). NEPSY: A developmental neuropsychological assessment. San Antonio, TX: The Psychological Corporation.
- Pears, K., Bruce, J., Fisher, P., & Kim, H. (2010). Indiscriminate friendliness in maltreated foster children. *Child Maltreatment*, 15(1), 64-75.
- Pollak, S.D., Nelson, C.A., Schlaak, M.F., Roeber, B.J., Wewerka, S.S., Wiik, K.L., Frenn, K.A., Loman, M.M., Gunnar, M.R. (2010). Neurodevelopmental effects of early deprivation in postinstitutionalized children. *Child Devel*opment, 81(1), 224-236.
- 77. Klingberg, T., Fernell, E., Olesen, P.J., Johnson, M., Gustafsson, P., Dahlström, K., Gillberg, C.,G., Forssberg, H., & Westerberg, H. (2005). Computerized training of working memory in children with ADHD: A randomized, controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44(2), 177-186.
- Klingberg, T., Forssberg, H., & Westerberg, H. (2002).
   Training of working memory in children with ADHD.
   Journal of Clinical and Experimental Neuropsychology, 24(6), 781-791.
- Olesen, P.J., Westerberg, H., & Klingberg, T. (2004). Increased prefrontal and parietal activity after training of working memory. *Nature Neuroscience*, 7(1), 75-79.
- Rabiner, D.L., Murray, D.W., Skinner, A.T. & Malone, P.S. (2010). A randomized trial of two promising computerbased interventions for students with attention difficulties. *Journal of Abnormal Child Psychology*, 38(1), 131-142.
- 81. Rueda, M.R., Rothbart, M.K., McCandliss, B.D., Saccomanno, L., & Posner, M.I. (2005). Training, maturation and genetic influences on the development of executive attention. *Proceedings of the National Academy of Sciences of the United States of America*, 102(41), 14931-14936.

- Stevens, C., Fanning, J. Coch, D. Sanders, L., & Neville, H. (2008). Neural mechanisms of selective auditory attention are enhanced by computerized training: Electrophysiological evidence from language-impaired and typically developing children. *Brain Research*, 1205, 55-69.
- Barnett, W. S., Jung, K., Yarosz, D. J., Thomas, J., Hornbeck, A., Stechuk, R., Burns, M. S. (2008). Educational effects of the Tools of the Mind Curriculum: A randomized trial. *Early Childhood Research Quarterly*, 23(3), 299-313.
- Diamond, A., Barnett, W. S., Thomas, J., & Munro, S. (2007). Preschool program improves cognitive control. *Science*, 318(5855), 1387-8.
- 85. Raver, C.C., Jones, S.M., Li-Grining, C.P., Metzger, M., Champion, K.M., & Sardin, L. (2008). Improving preschool classroom processes: Preliminary findings from a randomized trial implemented in Head Start settings. *Early Childhood Research Quarterly*, 23(1), 10-26.
- Raver, C. C., Jones, S. M., Li-Grining, C. P., Zhai, F., Metzger, M. W., & Solomon, B. (2009). Targeting children's behavior problems in preschool classrooms: A cluster-randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 77(2), 302-316.
- Webster-Stratton, C., Jamila Reid, M., & Stoolmiller. M. (2008). Preventing conduct problems and improving school readiness: Evaluation of the Incredible Years Teacher and Child Training Programs in high-risk schools. *Journal of Child Psychology and Psychiatry*, 49(5), 471-488.
- Domitrovich, C.E., Cortes, R., & Greenberg, M.T. (2007).
   Improving young children's social and emotional competence: A randomized trial of the preschool PATHS curriculum. *Journal of Primary Prevention*, 28(2), 67-91.
- Domitrovich, C. E., Greenberg, M. T., Kusche, C., & Cortes, R. (1999). Manual for the Preschool PATHS Curriculum. South Deerfield, MA: Channing-Bete Company.
- Bodrova, E., & Leong, D.J. (1996). Tools of the mind: The vygotskian approach to early childhood education. Englewood Cliffs, NJ: Prentice Hall.
- 91. National Scientific Council on the Developing Child. (2008). Focus and planning skills can be improved before a child enters school. http://www.developingchild.net
- Raver, C. C., Jones, S.M., Li-Grining, C., Zhai, F., Bub, K, & Pressler, E. (In Press). CSRP's impact on low-income preschoolers' pre-academic skills: Self-regulation as a mediating mechanism. *Child Development*.
- 93. Riggs, N. R., Greenberg, M. T., Kusché, C. A., & Pentz, M. A. (2006). The mediational role of neurocognition in the behavioral outcomes of a social-emotional prevention program in elementary school students: Effects of the PATHS curriculum. *Prevention Science*, 7(1), 91-102.
- 94. Domitrovich, C.E., Gest, S.D., Gill, S., Bierman, K.L., Welsh, J.A., & Jones, D. (2009). Fostering high-quality teaching with an enriched curriculum and professional development support: The Head Start REDI program. *American Educational Research Journal*, 46(2), 567-597.
- Duncan, G.J., Dowsett, C.J., Claessens, A, Magnuson, K., Huston, A.C., Klebanov, P., Pagani, L.S.., Feinstein, L., Engel, M., Brooks-Gunn, J., Sexton, H., Duckworth, K., & Japel, C. (2007). School readiness and later achievement. Developmental Psychology, 43(6), 1428-1446.
- Shonkoff, J.P. Boyce, W.T., McEwen, B.S. (2009). Neuroscience, molecular biology and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA*, 301(21), 2252-2259.

- 97. Gilliam, W.S. (2005). Prekindergarteners left behind: Explusion rates in state prekindergarten programs. FCD Policy Brief Series, No. 3. New York: Foundation for Child Development.
- 98. Grannan, M., Carlier, C., & Cole, C.E. (1999). Early childhood care and education explusion prevention project. Southgate, MI: Downriver Guidance Clinic, Department of Early Childhood Programs.
- 99. Weintraub S., Dikmen, S.S., Heaton, R.K., Tulsky, D.S., Zelazo, P.D., Bauer, P.J., Carlozzi, N.E., Slotkin, J., Blitz, D., Wallner-Allen, K., Fox, N.A., Beaumont, J.L., Mungas, D., Richler, J., Deocampo, J.A., Anderson, J.E., Manly, J.J., Borosh, B., Havlik, R. & Gershon, R. (In Press). NIH Toolbox for the Assessment of Behavioral and Neurological Function: Cognition domain instruments. Neurology.
- 100. Best, J.R. & Miller, P.H. (2010). A developmental perspective on executive function. Child Development, 81(6), 1641-1660.
- 101. Diamond, A. (1991a). Frontal lobe involvement in cognitive changes during the first year of life. In K.R. Gibson & A.C. Petersen (Eds.), Brain maturation and cognitive development: Comparative and cross-cultural perspectives (pp. 127-180). New York: Aldine de Gruyter.
- 102. Diamond, A. (1991b). Neuropsychological insights into the meaning of object concept development. In S. Carey & R. Gelman (Eds.), The epigenesis of mind: Essays on biology and cognition (pp. 67-110). Hillsdale, NJ: Lawrence Erlbaum Associates.
- 103. Diamond, A. (2006). The early development of executive functions. In E. Bialystok & F. Craik (Eds.), Lifespan cognition: Mechanisms of change (pp. 70-95). New York: Oxford University Press.
- 104. Evans, G.W., & Wachs, T.D., eds. (2010). Chaos and its influence on children's development: An ecological perspective. Washington, DC: American Psychological Associa-
- 105. Arnsten, A. (1998). The biology of being frazzled. Science, 280(5370), 1711-1712.

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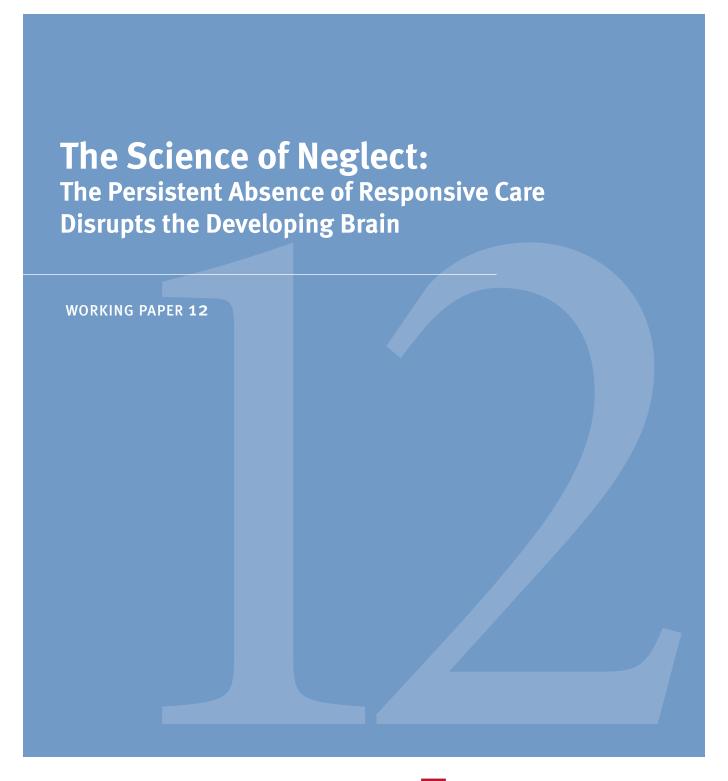
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The National Scientific Council on the Developing Child is a multidisciplinary, multi-university collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children. For more information, go to www.developingchild.net.

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### The Issue

THE BUILDING BLOCKS OF THRIVING COMMUNITIES AND WELL-FUNCTIONING SOCIETIES REST ON the health and development of their people. Beginning immediately after birth, a strong foundation for human well-being requires responsive environments and supportive relationships to build sturdy brain circuits, facilitate emerging capabilities, and strengthen the roots of physical and mental health.<sup>1,2,3</sup> Through mutually rewarding, "serve and return" interactions with the adults who care for them (see sidebar below), young children are both initiators and respondents in this ongoing process. These reciprocal and dynamic interactions are essential for healthy development and literally shape the architecture of the developing brain.<sup>4,5</sup>

Because responsive relationships are developmentally expected and biologically essential, their absence signals a serious threat to child wellbeing, particularly during the earliest years, and this absence activates the body's stress response systems. When decreased responsiveness persists, the lost opportunities associated with diminished interaction can be compounded by the adverse impacts of excessive stress activation, the physiological effects of which can have lifelong consequences. This multidimensional assault on the developing brain underscores why significant deprivation is so harmful in the earliest years of life and why effective interventions are likely to pay significant dividends in better longterm outcomes in learning, health, and parenting of the next generation.6

Extensive biological and developmental research over the last 30 years has generated substantial evidence that young children

# Serve and Return Interaction Between Children and Caregivers

The architecture of the brain is composed of highly integrated sets of neural circuits (i.e., connections among brain cells) that are "wired" under the continuous and mutual influences of both genetics and the environment of experiences, relationships,



and physical conditions in which children live. Experiences "authorize" genetic instructions to be carried out and shape the formation of the circuits as they are being constructed. This developmental progression depends on appropriate sensory input and stable, responsive relationships to build healthy brain architecture.

Abundant scientific evidence demonstrates that a major ingredient in this process is the "serve and return" relationship between children and their parents or other caregivers in the family or community. Young children naturally reach out for interaction through babbling, facial expressions, gestures, and words, and adults respond with the same kind of vocalizing and gesturing back at them. This "serve and return" behavior continues back and forth like a game of tennis or volleyball. If the responses are unreliable, inappropriate, or simply absent,





the developing architecture of the brain may be disrupted, and later learning, behavior, and health may be impaired.

A breakdown in these reciprocal, serve and return interactions between adult caregivers and young children can be the result of a multitude of predisposing factors. These may include significant stresses associated with high levels of economic hardship, social isolation, and/or chronic disease, as well as a wide range of adult mental health impairments, including depression, anxiety, post-traumatic stress disorder, serious personality disorders, or substance abuse involving alcohol or illicit drugs. Caregivers who are at highest risk for providing inadequate care often experience several of these problems simultaneously. Neglectful acts or patterns occur in every culture, at all income levels, and within all racial, ethnic, and religious groups.

who experience severe deprivation or significant neglect—defined broadly as the ongoing disruption or significant absence of caregiver responsiveness—bear the burdens of a range of adverse consequences. Indeed, deprivation or neglect can cause more harm to a young child's development than overt physical abuse, including subsequent cognitive delays, impairments in executive functioning, and

### **Defining Neglect**

Understanding the biological effects of inadequate responsiveness to the needs of young children has important implications for policy decisions—but it is important to also acknowledge that the term "neglect" carries special significance because of its association with the child welfare system and its implications for case documentation and adjudication. The federal Child Abuse Prevention and Treatment Act (CAPTA), as amended by the Keeping Children and Families Safe Act, defines child abuse and neglect as "at a minimum, any recent act or failure to act on the part of a parent or caretaker, which results in death, serious physical or emotional harm, sexual abuse or exploitation, or an act or failure to act which presents an imminent risk of serious harm."<sup>7,8</sup>

This latter dimension—"failure to act which presents an imminent risk of serious harm"—lies at the core of most legal definitions of neglect, but fails to sufficiently acknowledge the less immediately visible but highly threatening, long-term consequences of excessive deprivation that can lead to lifelong problems in learning, behavior, and health. Indeed, science tells us that many young children who are identified by the child welfare system as meeting the criteria for reportable neglect may not exhibit evidence of physical harm, yet they may have already sustained disruptions of their developing brain circuitry (or other developing organs and metabolic systems) that could have serious lifelong consequences.

To researchers, neglect—also sometimes referred to as deprivation—refers to the absence of sufficient attention, responsiveness, and protection that are appropriate to the age and needs of a child. The potential seriousness of such a circumstance is acknowledged broadly, yet its specific parameters can vary widely by type, duration, and cultural differences in child-rearing beliefs and practices. This paper is intended to help policymakers and practitioners distinguish among various forms of neglect—and potential responses to them—by focusing not on state or federal definitions but on biological responses to the diverse characteristics of unresponsive care that can undermine healthy development. While understanding that the terms "neglect" and "serious neglect" indicate important distinctions to policymakers through definitions that have been codified in various legislative or administrative decisions, in this paper they are employed to reflect the descriptive terminology used by neuroscientists and developmental scholars.

disruptions of the body's stress response.<sup>9,10,11</sup> When chronic deprivation leads to persistent activation of stress response systems in a young child, it can actually disrupt and weaken developing brain architecture. Over time, the wear and tear of this excessive stress response and the chemicals it releases can lead to academic struggles, difficulties in social adjustment, mental health problems, and even chronic physical disease.

The early roots of this science are reflected in pioneering behavioral studies of children living in institutions,12 as well as in family settings with compromised caregiving capacities. 10,13 More recently, this knowledge base has been deepened by extensive developmental and neurobiological evidence from studies of young children who experienced extreme deprivation in state-run institutions in Romania, China, and other contexts outside North America. 14,15,16,17,18 Regardless of the differences in settings (i.e., home versus institution) or causes of neglect, however, understanding the fundamental connection between early deprivation and subsequent impairment lies in the realization that healthy development can be threatened not only by bad things that may happen to children (e.g., as a result of physical or sexual abuse), but also by the absence of sufficient amounts of essential experiences that are required for their positive well-being.

Despite these compelling findings, child neglect receives far less public attention than physical abuse and sexual exploitation19,20,21 and a lower proportion of mental health services is dedicated to children who have been neglected in comparison to the availability of treatment programs for victims of physical trauma.<sup>22</sup> Yet neglect is by far the most prevalent form of child maltreatment. In 2010, more than half a million documented cases that met state or federal definitions of neglect were reported in the United States, which accounted for 78% of all maltreatment cases nationwide. This rate far exceeded all other forms of child maltreatment (some of which included both overt abuse and neglect), including physical abuse (17.6%), sexual abuse (9.2%), and psychological abuse (8.1%).<sup>23</sup>

Explicit criteria for determining the threshold for government intervention in cases of suspected neglect are within the purview of each state's child welfare system, and the definitions vary considerably across jurisdictions. Within

this context, most circumstances that are selected for investigation fall within one of the following categories: (1) physical or supervisory neglect (i.e., failure to provide adequate food, shelter, hygiene, and/or appropriate oversight to ensure a child's safety); (2) psychological neglect (i.e., failure to attend to a child's emotional and/or social needs); (3) medical neglect (i.e., failure to secure adequate treatment for an identified health problem); and (4) educational neglect (i.e., failure to meet a child's formal learning needs). Notwithstanding their distinctive characteristics, these four forms of neglect have often been found to co-occur. 24,25,26 And while these distinctions are time-tested and valid, they do not help with the challenge of judging relative severity or determining when to intervene. Here the science of development and neurobiology of stress can help.

The aim of this working paper is to synthesize a wealth of evidence from neuroscience, molecular biology, epigenetics, and a range of behavioral and social sciences to promote greater public understanding of this widespread threat to child well-being. Using science as our guide, we have delineated four types of diminished responsiveness and their consequences in order to provide a useful

framework for developing more effective strategies to protect vulnerable children from this complex challenge.

Occasional Inattention. There is considerable variation in the circumstances and contexts in which parents and other caregivers do or do not respond in a timely fashion to the everyday needs and overtures of young children. If diminished attention occurs on an intermittent basis in an otherwise loving and responsive environment, there is no need for concern. Indeed, some developmental scientists suggest that variations in adult responsiveness present growth-promoting challenges that may help young children recognize the distinction between "self" and "other," which is a necessary prerequisite for moving toward greater independence and increasing capacity for self-care and problem-solving.27

Chronic Understimulation. If caregivers exhibit an ongoing, diminished level of child-focused attention that fails to support a young child's need for cognitive, language, social, and emotional engagement, intervention can be helpful. Common examples of such under-stimulation include few daily interactions that provide opportunities for young children to engage in

	Science Helps to Differentiate Four Types of Unresponsive Care				
	OCCASIONAL INATTENTION	CHRONIC UNDER-STIMULATION	SEVERE NEGLECT IN A FAMILY CONTEXT	PROFOUND DEPRIVATION IN AN INSTITUTION	
Features	Intermittent, diminished attention in an otherwise responsive environment	Ongoing, diminished level of child-focused responsiveness and developmental enrichment	Significant, ongoing absence of serve and return interaction, often associated with failure to provide for basic needs	"Warehouse-like" conditions with many children, few caregivers, and no individualized adult-child relationships that are reliably responsive	
Effects	Can be growth- promoting under caring conditions	Often leads to developmental delays and may be caused by a variety of factors	Wide range of adverse impacts, from significant developmental impairments to immediate threat to health or survival	Basic survival needs may be met, but lack of individualized adult responsiveness can lead to severe impairments in cognitive, physical, and psychosocial development	
Action	No intervention needed	Interventions that address the needs of caregivers combined with access to high-quality early care and education for children can be effective	Intervention to assure caregiver responsiveness and address the developmental needs of the child required as soon as possible	Intervention and removal to a stable, caring, and socially responsive environment required as soon as possible	

active conversation with adult caregivers or frequent periods in which infants or toddlers are left in front of a television for hours at a time. In some cases, this lack of serve and return engagement is the result of limited understanding of the developmental needs of young children. In other circumstances, it may be caused by a range of risk factors such as caregiver depression, social or geographic isolation, the stresses of poverty or discrimination, or a distracting family illness. Understanding the precipitating factors and employing appropriate strategies to

# The significant absence of basic, serve and return interaction can produce serious physiological disruptions that lead to lifelong problems in learning, behavior, and health.

address identified needs (e.g., from simple parenting education to the provision of enriched learning experiences through high-quality child care or early education programs) can produce strong returns on relatively simple, voluntary interventions.

Severe Neglect in a Family Context. The ongoing disruption or significant absence of the kind of basic, serve and return interaction necessary for healthy child development can produce serious physiological disruptions that lead to lifelong problems in learning, behavior, and health. This magnitude of neglect may also be associated with the failure to provide for a child's basic nutritional, medical, and educational needs. Children who experience this level of deprivation typically have no stable, adult source of reliable care and protection, and therefore meet the criteria for public intervention under the jurisdiction of the child welfare system. In the most severe cases (e.g., a baby or toddler who is typically left alone and ignored for many hours at a time), a child's very survival is threatened and immediate intervention is mandatory.

### Profound Deprivation in an Institution.

Of equal concern, yet presenting a very different context, institutions that "warehouse" large numbers of infants and young children serve as extraordinary examples of extreme deprivation. Such conditions typically include staff with little or no training in the care of children, highly regimented "assembly-line" caregiving with minimal one-on-one interaction, youngsters who are ignored and unstimulated for virtually all of their awake hours, and no adult-child relationships that are reliably responsive to a child's individual needs. Young children who live in such settings experience little more than transient serve and return interactions. Frequent staff rotations mean that infants are cared for by many different people, making it extremely difficult to develop meaningful relationships with any single caregiver. In such circumstances, although basic needs for food, warmth, shelter, and medical care may be met (thereby avoiding most legal definitions of neglect), the setting itself may still be a precipitant of severe psychosocial deprivation for the youngest inhabitants. While most of the research relating to profound neglect in such settings has been focused on extreme situations, such as those in Eastern Europe and other locations around the world, a wide range of quality of care in institutional settings exists within the United States as well. Indeed, there is growing evidence that some residential care facilities for infants and toddlers in the United States are harmful to development and therefore not an acceptable alternative to high-quality foster care or adoptive parents.<sup>28</sup>

IN SOME CONTEXTS, THE DETERMINATION OF whether a child is being neglected falls within the relatively low-stakes realm of individual judgment. In other contexts, it falls within the exceedingly high-stakes jurisdiction of child welfare agencies and courts of law, where highly consequential decisions are made about custodial responsibility, parental rights, criminal culpability, and the best interests of the child. The four types of deprivation described above are intended to provide an organizing framework for assessing current policy and program options for young children who are deemed to be experiencing a level of inadequate caregiving that warrants public attention and identifying those whose situations do not. In the final analysis, the magnitude of the decision-making challenge requires a balanced blend of scientific knowledge, cultural values, and shared public responsibility.

# What Science Tells Us About the Impact of Deprivation on the Development of Young Children

THE CLEAREST FINDINGS ON THE EFFECTS OF deprivation on development come from studies of children who have experienced profound neglect while being raised in institutions. Research on these children has provided an opportunity for investigators to examine the distinctive consequences of extreme psychosocial deprivation apart from the impacts of other forms of maltreatment. Additional knowledge comes from studies involving institutionalized children whose life circumstances have been transformed through foster care placements or permanent adoption. Although neurobiological information on children who experience significant neglect in home settings is less available, research currently in progress is likely to generate new insights in the near future.

There is extensive evidence that profound deprivation in institutional settings is associated with abnormalities in the structure and functioning of the developing brain. Children who experience extreme levels of social neglect early in life show diminished electrical activity in the brain, as measured through electroencephalography (EEG).<sup>15,18</sup> These EEG disturbances are similar to those observed in non-neglected children who have difficulties with attention and learning.<sup>29</sup> Institutionally reared children also show differences in the neural reactions that occur as an individual is processing information, such as looking at faces to identify different emotions. 16,17 These findings indicate impairments in the way the brain interprets such input and are consistent with behavioral observations that neglected children struggle to correctly recognize different emotions in others.11,30 Children who experience profound deprivation in institutional settings also exhibit decreased brain metabolism and poorer connections among different areas of the brain that are important for integrating complex information, including cognitive, social, and emotional competencies.14,31

Significant neglect or deprivation in the early childhood years influences the development of a variety of brain regions that are important for thinking, learning, focusing attention, controlling emotions, and managing stress. One particularly sensitive area is the prefrontal cortex (PFC), which serves as the brain's "air traffic control system" by supporting the development of a wide range of executive functions, such as planning, monitoring, working memory, problem-solving, and behavioral self-regulation.32 In neuroimaging studies, adults and adolescents who report histories of severe neglect during childhood show smaller PFC volumes when compared with non-neglected individuals.33,34 Serious deprivation is also associated with abnormal activity in areas of the brain involved in emotion and stress regulation (i.e., the amygdala and hippocampus)33,34,35,36,37,38 as well as attention and self-control (e.g., the anterior cingulate cortex).39 Collectively, these findings indicate that significant deprivation disrupts the way in which children's brains develop and process information, thereby increasing the risk for attentional, emotional, cognitive, and behavioral disorders later in life.

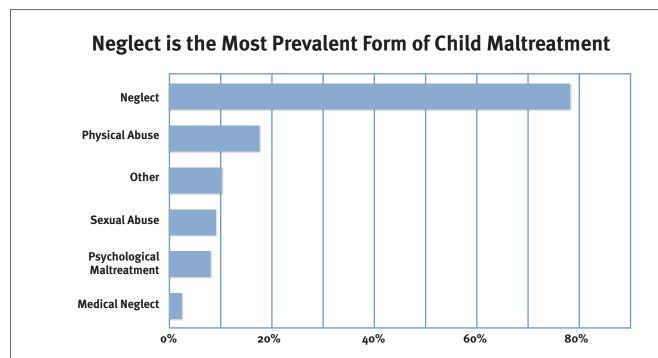
Chronic deprivation or neglect can alter the development of biological stress response systems in a way that compromises children's ability to **cope with adversity.** Extensive research indicates that the two primary stress response systems in humans—the sympathetic-adrenal-medullary (SAM) system, which produces adrenaline and affects heart and respiration rates, and the hypothalamic-pituitary-adrenal (HPA) axis, which elevates cortisol, a key stress hormone are both disrupted by significant deprivation. For example, years after adoption, children who experienced extreme neglect in institutional settings show abnormal patterns of adrenaline activity in their heart rhythms, which can indicate increased biological "wear and tear" that leads to greater risk for anxiety, depression, and cardiovascular problems later in life.40

Cortisol regulation, an important marker of stress response activation in the HPA axis, is altered in children who experience both severe neglect in families and profound deprivation in institutional settings. The normal rhythms of cortisol secretion observed among typically developing children begin with a sharp increase in the morning to get the body going for the day, followed by a steady decline in levels that bottom out in late afternoon or evening as the body prepares for sleep. In contrast, children who experience significant neglect, whether in institutional or family settings, show lower levels of cortisol in the morning and an atypically flat pattern of secretion across the day.<sup>9,41,42</sup> Although these abnormal cortisol responses appear to normalize when children are placed in nurturing home environments, there is evidence that the brain architecture that developed at the time of these atypical patterns may be structurally weakened, which could have enduring, adverse effects throughout life.43

Children who have experienced serious deprivation are at risk for abnormal physical development and impairment of the immune **system.** Severe neglect is associated with significantly delayed growth in head circumference (which is directly related to brain growth) during infancy and into the toddler years.44

More extreme conditions of deprivation, such as those experienced in institutional settings that "warehouse" young children, are associated with even more pervasive growth problems, including smaller body size, as well as impairments in gross motor skills and coordination. 45,46,47 Profound deprivation has also been found to compromise physical health, as children who are raised in institutional settings have more infections and are at greater risk of premature death than children who live in supportive homes.<sup>19</sup> One possible explanation for these findings is that chronically disrupted cortisol levels suppress immunologic reactivity and physical growth, thereby leading to a greater risk for infection and chronic, stress-related disease throughout life.48

Significant neglect in family settings and severe institutional deprivation are both associated with greater risk for emotional, behavioral, and interpersonal relationship difficulties later in life. Children reared in families who



Each state defines the types of child abuse and neglect in its own statute and policy, guided by federal standards, and establishes the level of evidence needed to substantiate a report of maltreatment. The data above, from the National Child Abuse and Neglect Data System (NCANDS), reflects the total number of victims (defined as a child for whom the state determined at least one report of maltreatment was found to be substantiated or indicated) as reported by all 50 states, the District of Columbia, and Puerto Rico, between Oct. 1, 2009, and Sept. 30, 2010. "Other" includes abandonment, threats of harm, and drug addiction.

Source: U.S. Department of Health and Human Services (2010b)<sup>23</sup>

experience chronic neglect show higher rates of insecure or disorganized attachment behaviors with their primary caregivers, and these relationship difficulties extend to interactions with others as they grow older. 49,50,51,52,53,54 Preschoolers with histories of severe neglect in a family setting are more likely to become overly dependent on their teachers for support and nurturance, when compared with non-neglected children.<sup>10</sup> Youngsters who have experienced chronic under-stimulation or serious neglect in family settings engage in fewer social interactions with their peers during preschool when compared with children who experienced other forms of maltreatment.55,56,57 This deficiency in social skills and peer relationships often persists throughout the school-age years 13,56,58,59,60 and can extend into adolescence.61

Children who have been severely neglected also have higher rates of emotional and behavioral problems in comparison to non-neglected children, even when compared to those who have been physically or sexually abused.49 Infants and toddlers exposed to severe neglect within a family context, or to the profound deprivation of an institutional setting, show increased negative emotions, poorer impulse control, and reduced enthusiasm, confidence, and assertiveness when completing problemsolving tasks. 10,49,62,63 Serious deprivation in institutional settings also has been linked to difficulties in children's emerging ability to discriminate emotions. 11,30,64 As they grow older, children reared in neglectful conditions in family settings are at increased risk for a variety of emotional difficulties, such as low self-esteem, poor self-confidence, and diminished assertiveness.<sup>10</sup> Significant neglect is also associated with an increased risk for personality disorders, anxiety, and depression when compared with other forms of maltreatment. 65,66,67,68

Beyond the short-term consequences of neglect, there is also evidence that these emotional difficulties can persist. Adults who report childhood neglect and emotional abuse experience greater anxiety, depression, and post-traumatic stress symptoms when compared to adults without such histories. 69,70 Although the majority of adults who experienced neglect as children do not engage in delinquent, criminal, or violent behavior,71 the odds are significantly greater that they will be arrested for violent crimes and have diagnoses of

antisocial personality disorder compared with adults who were not maltreated as children. 72,73

Children who have experienced deprivation or neglect are more likely to have cognitive problems, academic delays, deficits in executive function skills, and difficulties with attention regulation. Extreme deprivation in institutional settings has been associated with particularly severe cognitive impairments74,75,76 and academic delays,77 with documented effects persisting into adolescence.78 Infants who experience significant neglect in family environments demonstrate poorer performance on later measures of cognition and language development than young children who have experienced other forms of maltreatment. 52,79,80,81 Throughout the elementary school years, children who were previously neglected in family settings show

Chronic deprivation or neglect can alter the development of biological stress response systems in a way that compromises children's ability to cope with adversity.

more academic problems and special education referrals than non-neglected children. 13,56,82,83,84 While research on the long-term effects of deprivation into adulthood is more limited, adults with histories of childhood neglect (and/or abuse) exhibit lower IQ scores, are less likely to graduate from high school, and have poorer reading skills when compared with adults who were not neglected as children.85

Significant deprivation or neglect can have particularly devastating effects on the development of executive function skills, which are critical to the ability to operate effectively and independently throughout life. Consistent with observed alterations in patterns of brain activity, children who have experienced serious levels of deprivation—whether in homes, foster care, or institutions-tend to struggle with the demands of regulating attention.86 They are more frequently rated as inattentive and hyperactive by both their parents and teachers, 87 as well as described as unfocused and inattentive during the school years,13 with longer or more severe deprivation associated with higher levels of dysfunction.<sup>87</sup> Children who experienced serious deprivation in the first few years of life display greater problems in executive function skills during middle childhood, with particular difficulties in visual memory;<sup>88</sup> continuing problems in attention and learning;<sup>89</sup> and atypical neural activity related to attention and executive functioning that persists throughout the school years.<sup>26,75,90</sup>

The impact of deprivation or neglect can be manifested in different ways across different periods of development. At younger ages, maltreated children show impairments in their ability to discriminate different emotions, yet these difficulties are not observed at older ages. 11,35,91 Conversely, antisocial behavior may be more salient among adults or older adolescents with early childhood histories of neglect. 72,73 Given the fact that interpersonal relationships and life challenges (e.g., dealing with peers, becoming involved in romantic relationships, entering parenthood, achieving financial stability) change across the lifespan,

it is essential that the adverse consequences of significant deprivation are addressed in a developmentally appropriate manner.

The negative consequences of deprivation and neglect can be reduced or reversed through appropriate and timely interventions. The capacity for recovery in children who are removed from neglectful conditions and placed in nurturing environments in a timely fashion has been welldocumented.92,93,94,95,96 However, improvement often requires more than simply the cessation of neglectful caregiving. Rather, systematic, empirically supported, and often long-term (six to nine months or longer) interventions are needed to promote effective healing. Successful treatments of this nature have been shown to reduce behavioral difficulties and attachment problems in previously neglected young children who have been placed in foster homes 92,93,95 as well as to promote secure attachments in young children who continue to live with their families while being monitored by child welfare agencies because

#### Neglect Can Be a Greater Threat to Development than Abuse Creativity **Confidence and Assertiveness** 2.5 4.0 2.0 3.0 1.5 2.0 1.0 1.0 0.5 0.0 0.0 No Verbal **Physical** Verbal Physical Neglect Neglect Maltreatment **Abuse Abuse** Maltreatment Abuse Abuse

At age 3 ½, children who had experienced different types of maltreatment in the first year of life and a group that had not experienced any maltreatment were compared in a "barrier box" situation designed to evaluate children's responses to frustrating situations. In the scenario, toys were placed inside a box that could not be opened by a young child. Researchers observed the children's efforts to solve the challenge and rated them on a 3- or 7-point scale and then adjusted scores for time spent on task (median scores for each group are shown above). Results showed that neglected children had the greatest difficulty and lacked the creativity, confidence, and assertiveness to cope with the challenges they faced.

Source: Egeland, et al. (1983)<sup>10</sup>

of previous allegations of neglect.97 On a biological level, systematic interventions targeting the social-emotional needs of young children living in foster care settings (the majority of whom were victims of neglect rather than physical abuse) have shown evidence of improved stress-regulatory capabilities with patterns of cortisol production that are indistinguishable from those of non-neglected, healthy children.<sup>26,93,94,96,98,99</sup> With appropriate intervention, previously institutionalized children have also demonstrated improvements in brain activity as measured by EEG. 100,101

Children's recovery rates are influenced by the severity, duration, and timing of the deprivation as well as by the timing and type of the intervention that is provided. Children who experience more severe deprivation or neglect, especially during the early childhood years, are more likely to withdraw when stressed and show more anxiety and difficulties regulating their mood than children whose experiences of deprivation are less severe.60 Longer periods of deprivation have also been associated with greater deficits in attention and cognitive control,102 academic achievement,78,103 brain activity, 104 and dysregulation of the HPA axis. 105 Previously institutionalized children who experienced the most extreme levels of deprivation often continue to struggle with problems in

attention and behavioral regulation even after intervention has been provided. 106,107,108,109,110

The timing of intervention is a critically important predictor of outcomes. If appropriate intervention occurs very early—in various studies the benchmark age for removal from extreme deprivation has been identified as 6, 12, or 24 months—substantially improved functioning in cognition, attention, memory, and executive functioning can be achieved.74,88,101,111 For example, young children who were removed from Romanian institutions and placed in high-quality foster

If appropriate intervention occurs very early, substantially improved functioning in cognition, attention, and memory can be achieved.

care homes prior to 24 months of age (in comparison to those who were removed after age two years), showed remarkable gains (after an initial period of adjustment) in a range of cognitive abilities74 and neuropsychological functioning, especially in the area of visual memory.88 Generally speaking, it appears that the more profound and pervasive the deprivation, the earlier the child needs to be removed in order to facilitate the greatest recovery.

# **Addressing Common Misconceptions**

Contrary to popular belief, significant deprivation or neglect appears to be at least as great a threat to health and development as physical abusepossibly even greater. When compared with children who have been victimized by overt physical maltreatment, young children who experienced prolonged periods of neglect exhibit more severe cognitive impairments, language deficits, academic problems, withdrawn behavior, and problems with peer interaction. 52,60 This suggests that sustained disruption of serve and return interactions in early relationships may be more damaging to the developing architecture of the brain than physical trauma. 52,66

Contrary to popular belief, the mere removal of a young child from an environment of significant neglect or deprivation is not a guarantee of posi**tive outcomes.** Children who experience severe deprivation typically need therapeutic, supportive care to facilitate their recovery. In the absence of appropriate intervention services, neglected children remain at increased risk for a host of problems (as described above) that have been found to continue through adolescence and into the adult years.71,72,78,85 Evidence-based intervention programs designed to help caregivers respond to the distinctive needs of neglected children are currently available (see sidebar on page 12).94,97 The imperative of appropriate and timely referrals cannot be ignored.

# The Science-Policy Gap

Public concern about the problem of child maltreatment is focused disproportionately on the dangers of physical and sexual abuse, while significant neglect receives less attention. Given the fact that child neglect is the most common reason for engaging protective services, it is particularly striking that there is still no broadbased agreement on clear and objective criteria for defining this form of maltreatment and for authorizing state intervention. Moreover, despite important scientific advances in elucidating the wide range of adverse developmental and neurobiological impacts of early deprivation, there has been relatively little change in the way in which services are provided for this proportionally large and highly vulnerable population within the child welfare system.

Despite considerable advances in scientific knowledge about the short- and long-term consequences

of significant deprivation and the importance of prompt intervention, most child welfare agencies have relatively limited capacity to address the developmental needs of young children who have experienced reportable neglect. The disruptions in neural pathways and stress response systems brought on by significant neglect can be lessened by the provision of skilled, supportive caregiving. 98,100,104,112,113 Growing evidence for this assertion underscores the imperative for increased investment in the development and implementation of effective, scalable, evidence-informed programs in community-based settings for children who are experiencing significant deprivation. Recognizing the potentially greater returns on prevention compared with rehabilitation, there is a particularly compelling need for more effective outreach to families facing considerable adversity that puts their young children at risk for significant neglect and its consequences.

# **Implications for Policy and Programs**

SCIENCE TELLS US THAT REPEATED AND PERsistent periods of prolonged unresponsiveness from primary caregivers will lead to excessive activation of a young child's psychological and physiological stress response systems. This, in turn, can lead to toxic stress and its consequences—a lifetime of impairments in learning, behavior, and both physical and mental health. Conversely, extensive research also points toward the healing power of nurturing, responsive, and reliable relationships for young children who have experienced significant neglect, with or without associated trauma.

Notwithstanding the dangers of significant deprivation, common sense tells us that young children do not need constant attention every minute of every day—and most are able to tolerate reasonable delays in responsiveness from the adults who care for them without any evidence of harm. Indeed, extensive research demonstrates that manageable levels of normative stress provide opportunities for young children to develop their own capacities to cope with adversity, particularly when adults provide the supportive "scaffolding" necessary to help build

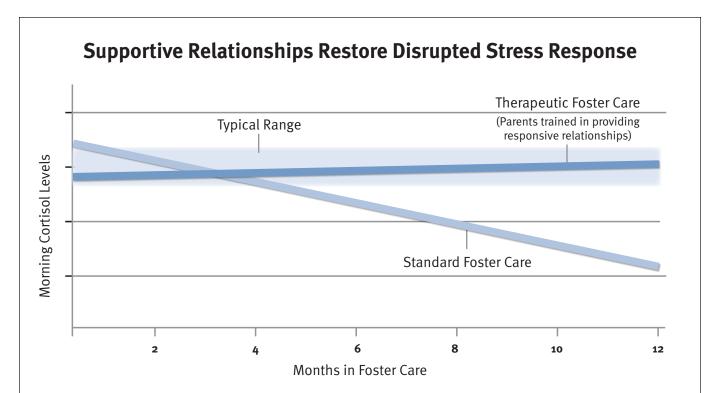
those adaptive skills over time. It is also important to recognize that normative child-rearing beliefs and practices vary considerably across and within cultures with respect to what might be considered a desirable or ideal amount of "serve and return" interaction between young children and adults.

Given the dangers of both over- and underidentification of "inadequate" caregiving, the time has come to leverage advances in science to inform a fundamental re-examination of our approaches to the identification, prevention, reduction, and mitigation of neglect and its consequences, particularly in the early years of life. The principal question facing policymakers and practitioners is clear: How can the decisions we make ensure that all young children receive the benefits of the caring and responsive relationships they need to develop in a healthy way?

There is a compelling need to re-assess the allocation of resources to and within the child welfare system, and to invest more in the development and implementation of evidence-based programs specifically designed to address the distinctive needs of children who are experiencing significant neglect. Given the limited public attention focused on problems that result from deprivation, it is not surprising that so few financial and programmatic resources are directed toward addressing this costly societal problem. That said, the immediate circumstances and long-term prospects of neglected children could be enhanced significantly by several critical actions. These include (1) dissemination of new scientific findings to child welfare professionals; (2) collaboration between child development researchers and service providers to develop more effective prevention and intervention strategies; (3) coordination across policy and service sectors to identify vulnerable children and families as early as possible; and (4) cooperation among policymakers, family court judges, and practitioners to improve access to non-stigmatizing, community-based services. Greater societal benefits would also be realized if we moved beyond

a narrow focus on children whose parents are struggling with significant social and economic hardship and directed more attention to the threat of neglect in families across the socioeconomic spectrum, such as in circumstances where parents are overwhelmed by chronic psychological or medical conditions. To this end, the types of deprivation described in this paper help to identify potentially neglecting environments that are less likely to be brought to the attention of child welfare professionals.

The long-term, neurobiological and developmental consequences of the most serious conditions of deprivation underscore the need for prevention programs as early as possible. Powerful and robust findings from developmental science suggest that the sooner neglected children receive appropriate intervention, the less likely they are to demonstrate long-term, adverse effects. It is therefore critical that key personnel



Children in the child welfare system, many suffering from serious neglect, can see dramatic improvements in stress response with the provision of supportive relationships. Without such relationships, children in this study who received standard foster care showed suppressed levels of the stress hormone cortisol, which worsened the longer they were in foster care. Foster parents trained to provide responsive relationships through the Multidimensional Treatment Foster Care for Preschoolers intervention (see sidebar on page 12) were able to restore foster children's stress hormones to typical levels, as measured in a control group of children from the same community who were not in foster care.

Source: Fisher, et al. (2007)96

# **Promising Intervention Models**

Three promising intervention models (among others) for children who have experienced significant neglect and other forms of maltreatment have demonstrated effectiveness on a variety of measures.

### **Attachment and Biobehavioral Catch-Up** (ABC) Intervention

This short-term intervention, developed by Mary Dozier at the Infant Caregiver Lab at the University of Delaware, is designed to improve attachment regulation and biobehavioral regulation in children who have experienced abuse and neglect.

### **TARGET POPULATION**

Infants and toddlers placed in foster care, relative care, or living with their birth parents.

### **PROGRAM GOALS AND INTERVENTION STRATEGIES**

Strengthen parents' or caregivers' sensitivity and responsiveness to an infant's cues and help them provide an environment in which they are able to foster a young child's regulatory abilities.

### **EVIDENCE FOR EFFECTIVENESS**

Young children who received the ABC intervention developed more secure attachments to their caregivers more frequently,92 showed more normative patterns of cortisol production (indicative of improved stress regulation),93,98 and demonstrated better behavioral regulation<sup>93</sup> than children who received a control intervention.

### **Child-Parent Psychotherapy (CPP)**

This treatment model, developed by Alicia Lieberman and Patricia Van Horn at the University of California San Francisco (UCSF) Child Trauma Research Program, is designed to improve social-emotional, behavioral, and cognitive functioning in children exposed to interpersonal violence and other traumatic events.

### **TARGET POPULATION**

Children aged o-5 who experience mental health, attachment, and/or behavioral problems as a result of traumatic events.

### PROGRAM GOALS AND INTERVENTION STRATEGIES

Repair the rupture of trust in the parent-child relationship following trauma by enhancing the parent's capacity to protect the child and helping the child to regain a sense of safety in the relationship with the parent. Treatment also focuses on contextual factors that may affect the parent-child relationship, such as cultural norms and socioeconomic and immigration-related stressors.

### **EVIDENCE FOR EFFECTIVENESS**

CPP is listed as an evidence-based treatment in the SAMHSA National Register of Evidence-Based Practices and Programs, with five randomized studies documenting CPP efficacy. After CPP treatment, relative to control groups, anxiously attached toddlers of recent immigrant Latina mothers showed improvements in attachment and their mothers showed increased responsiveness;114 children who witnessed domestic violence showed greater reductions in behavior problems and traumatic stress symptoms; 115,116,117 the rate of secure attachment in maltreated infants improved significantly; 118 maltreated preschoolers showed better self-esteem and attitude towards the mother; 119 and toddlers of depressed mothers showed more secure attachment and improved cognitive functioning. 120,121,122

### Multidimensional **Treatment Foster Care** for Preschoolers

This early intervention service model, developed by Phil Fisher at the Oregon Social Learning Center, is designed to promote healthy selfregulation, positive relationships with caregivers and peers, and enhanced school readiness in vulnerable young children.

### **TARGET POPULATION**

Three- to six-year-old children in foster care, many of whom have histories of neglect.

### **PROGRAM GOALS AND INTERVENTION STRATEGIES**

Help caregivers provide and maintain a positive, responsive, and consistent environment for young children through the use of concrete encouragement to reinforce positive behaviors and effective limit-setting to reduce problematic behaviors. Children also receive additional support through behavioral therapy in a preschool setting and participate in a weekly playgroup that promotes positive socialization.

### **EVIDENCE FOR EFFECTIVENESS**

Preschoolers in the MTFC-P program showed improvements in attachment-related behaviors,95 fewer behavior problems, and improved cortisol rhythms,96,99 when compared with children who received a control intervention.

in the medical, child welfare, mental health, and legal systems are educated about this new science and organized to work collaboratively to assure the earliest possible identification of families that require preventive assistance as well as children who need therapeutic intervention. State responses to the federal Title IV-E waiver guidelines issued in 2012, focusing on child well-being and trauma-informed practice, offer a promising opportunity to address this need. Moreover, because child neglect often co-occurs with other family problems (particularly parental mental health disorders, such as depression and addictions to alcohol and drugs), effective intervention will require specialized services to address a variety of adult medical, economic, and social needs. Programs that serve adults facing serious challenges such as these—even those that don't specifically focus on children—can have a significant impact on child outcomes by building caregiver capacities and family resources that

prevent deprivation from occurring in the first

Measuring the economic benefits of interventions that improve life outcomes for young children who experience significant neglect or chronic understimulation would provide important data to justify and guide enhanced resource allocation. Much of the public discourse about economic returns on investment in the early childhood years is focused on the benefits of early care and education programs that generate long-term savings by reducing the societal costs of special education and incarceration, while increasing economic productivity. A rich and growing evidence base on the adverse impacts of serious deprivation on lifelong learning, behavior, and both physical and mental health suggests that the economic benefits of greater attention to this under-addressed problem in the early years could be substantial and thus justify targeted investments in both prevention and early intervention.

# References

- 1. Field, T. (1994). The effects of mother's physical and emotional unavailability on emotion regulation. Monographs of the Society for Research in Child Development, 59(2-3), 208-227.
- 2. Hofer, M. A. (1994). Hidden regulators in attachment, separation, and loss. The development of emotion regulation: Biological and behavioral considerations. Monographs of the Society for Research in Child Development, 59(2-3), 192-207.
- Shonkoff, J. & Phillips, D. (Eds.). (2000). From Neurons to Neighborhoods: The Science of Early Childhood Development. Committee on Integrating the Science of Early Childhood Development, National Research Council and Institute of Medicine. Washington, DC: National Academy Press.
- 4. National Scientific Council on the Developing Child. (2004). Young Children Develop in an Environment of Relationships: Working Paper 1. http://www. developingchild.harvard.edu
- National Scientific Council on the Developing Child. (2007). The Timing and Quality of Early Experiences Combine to Shape Brain Architecture: Working Paper 5. http://www.developingchild.harvard.edu
- Center on the Developing Child at Harvard University. (2010). The Foundations of Lifelong Health Are Built in Early Childhood. http://www.developingchild.harvard. edu
- U.S. Department of Health and Human Services, Administration for Children and Families, & Child Welfare Information Gateway. (2008). What is child abuse and neglect? http://www.childwelfare.gov/pubs/ factsheets/whatiscan.pdf

- 8. CAPTA Reauthorization Act of 2010 (P. L. 111-320). (2010). http://www.gpo.gov/fdsys/pkg/BILLS-111s3817e nr/pdf/BILLS-111s3817enr.pdf
- Bruce, J., Fisher, P. A., Pears, K. C., & Levine, S. (2009). Morning cortisol levels in preschool-aged foster children: Differential effects of maltreatment type. Developmental Psychobiology, 51(1), 14-23.
- 10. Egeland, B., Sroufe, A., & Erickson, M. (1983). The developmental consequence of different patterns of maltreatment. Child Abuse & Neglect, 7(4), 459-469.
- 11. Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. Developmental Psychology, 36(5), 679-688.
- 12. Provence, S & Lipton, R. (1962). Infants in Institutions. New York: International Universities Press.
- 13. Erickson, M., & Egeland, B. (1996). Child neglect. In J. Briere, L. Berliner, J. A. Bulkley, C. Jenny, T. Reid, (Eds.), The APSAC handbook on child maltreatment (pp. 4-20). Thousand Oaks, CA: Sage Publications, Inc.
- 14. Eluvathingal, T. J., Chugani, H. T., Behen, M. E., Juhasz, C., Muzik, O., Magbool, M., ... & Makki, M. (2006). Abnormal brain connectivity in children after early severe socioemotional deprivation: A diffusion tensor imaging study. Pediatrics, 117(6), 2093-2100.
- 15. Marshall, P. J., Fox, N. A. & the BEIP Core Group. (2004). A comparison of the electroencephalogram between institutionalized and community children in Romania. Journal of Cognitive Neuroscience, 16(8), 1327-1338.
- 16. Parker, S. W., Nelson, C. A., & the BEIP Core Group. (2005a). An event-related potential study of the impact

- of institutional rearing on face recognitions. *Development and Psychopathology*, 17(3), 621-639.
- Parker, S. W., Nelson, C. A. & the BEIP Core Group. (2005b). The impact of early institutional rearing on the ability to discriminate facial expressions of emotion: An event-related potential study. *Child Development*, 76(1), 54-72.
- Tarullo, A., Garvin, M. C., & Gunnar, M. (2011). Atypical EEG power correlates with indiscriminately friendly behavior in internationally adopted children. *Developmental Psychology*, 47(2), 417-431.
- De Bellis, M. D. (2005). The psychobiology of neglect. Child Maltreatment, 10(2), 150-172.
- 20. Minty, B., & Pattinson, G. (1994). The nature of child neglect. *British Journal of Social Work*, 24(6), 733-747.
- Wolock, I., & Horowitz, B. (1984). Child maltreatment as a social problem: The neglect of neglect. *American Journal of Orthopsychiatry*, 54(4), 530-543.
- Erickson, M. F., & Egeland, B. (2002). Child neglect. In J. E. B. Myers, L. Berliner, J. Briere, C. T. Hendrix, C. Jenny, & T. A. Reid (Eds.), *The APSAC handbook on child maltreatment* (2nd ed., pp. 3-20). Thousand Oaks, CA: Sage Publications, Inc.
- U.S. Department of Health and Human Services, Administration for Children and Families, Administration on Children, Youth and Families, & Children's Bureau. (2011). Child Maltreatment 2010. http://archive.acf.hhs. gov/programs/cb/pubs/cm10/cm10.pdf
- Kaufman, J., Jones, B., Stieglitz, E., Vitulano, L. & Mannarino, A. (1994). The use of multiple informants to assess children's maltreatment experiences. *Journal of Family Violence*, 9(3), 227-248.
- Levy, H. B., Markovic, J., Chaudry, U., Ahart, S., & Torres, H. (1995). Re-abuse rates in a sample of children followed for 5 years after discharge from a child abuse inpatient assessment program. *Child Abuse & Neglect*, 19(11), 1363-1377.
- Pears, K. C., Kim, H. K., & Fisher, P. A. (2008).
   Psychosocial and cognitive functioning of children with specific profiles of maltreatment. *Child Abuse & Neglect*, 32(10), 958-971.
- Tronick, E. Z., & Gianino, A. (1986). Interactive mismatch and repair: Challenges to the coping infant. *Zero To Three*, 6(3), 1-6.
- Jones-Harden, B. (2002). Congregate care for infants and toddlers: Shedding new light on an old question. *Infant Mental Health Journal*, 23(5), 476-495.
- Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003).
   A review of electrophysiology in attention-deficit/ hyperactivity disorder: I. Qualitative and quantitative electroencephalography. Clinical Neurophysiology, 114(2), 171-183.
- Wismer-Fries, A. B., & Pollak, S. D. (2004). Emotion understanding in post institutionalized Eastern European children. *Developmental Psychopathology*, 16(2), 355–369.
- Sheridan, M. S., Fox, N. A., Zeanah, C. H., McLaughlin, K., and Nelson, C.A. (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. Proceedings of the National Academy of Sciences of the United States of America, 109(32), 12927-12932.
- Center on the Developing Child at Harvard University.
   (2011). Building the Brain's "Air Traffic Control" System: How Early Experiences Shape the Development of Executive Function: Working Paper 11. http://www.developingchild. harvard.edu

- Edmiston, E., Wang, F., Mazure, C., Guiney, J., Sinha, R., Mayes, L., & Blumberg, H. (2011). Corticostriatallimbic gray matter morphology in adolescents with selfreported exposure to childhood maltreatment. Archives of Pediatric and Adolescent Medicine, 165(12), 1069-1077.
- Frodl, T., Reinhold, E., Koutsouleris, N., Reiser, M., & Meisenzahl, E. M. (2010). Interaction of childhood stress with hippocampus and prefrontal cortex volume reduction in major depression. *Journal of Psychiatric Research*, 44(13), 799-807.
- Maheu, F. S., Dozier, M., Guyer, A. E., Mandell, D., Peloso, E., Poeth, K., ... & Ernst, M. (2010). A preliminary study of medial temporal lobe function in youths with a history of caregiver deprivation and emotional neglect. *Cognitive Affective and Behavioral Neuroscience*, 10(1), 34-49.
- Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R., ... & Sonuga-Barke, E. J. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: the English and Romanian adoptees study pilot. *Journal of Child Psychology and Psychiatry*, 50(8), 943–951.
- Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., & ... Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and emotion regulation difficulties. *Developmental Science*, 13(1), 46–61.
- Tottenham, N., Hare, T., Millner, A., Gilhooly, T., Zevin, J. D., & Casey, B.J. (2011). Elevated amygdala response to faces following early deprivation. *Developmental Science*, 14(2), 190–204.
- Mueller, S., Maheu, F., Dozier, M., Peloso, E., Mandell, D., Leibenluft, E., ... & Ernsta, M. (2010). Early-life stress is associated with impairment in cognitive control in adolescence: an fMRI study. *Neuropsychologia*, 48(10), 3037–3044.
- Gunnar, M. R., Frenn, K., Wewerka, S. S., & Van Ryzin, M. J. (2009). Moderate versus severe early life stress: Associations with stress reactivity and regulation in 10-12-year-old children. *Psychoneuroendocrinology*, 34(1), 62-75.
- Dozier, M., Manni, M., Gordon, M. K., Peloso, E., Gunnar, M. R., Stovall-McClough, K., ... & Levine, S. (2006). Foster children's diurnal production of cortisol: An exploratory study. *Child Maltreatment*, 11(2), 189–197.
- 42. Carlson, M., & Earls, F. (1997). Psychological and neuroendocrinological sequelae of early social deprivation in institutionalized children in Romania. In C. Carter, I. Lederhendler, & B. Kirkpatrick (Eds.), *The Integrative Neurobiology of Affiliation* (pp. 419-428). New York: New York Academy of Sciences.
- Brunson, K. L., Grigoriadis, D. E., Lorang, M. T., & Baram,
   T. Z. (2002) Corticotropin-releasing hormone (CRH) downregulates the function of its receptor (CRF1) and induces CRF1 expression in hippocampal and cortical regions of the immature rat brain. Experimental Neurology, 176(1), 75-86.
- Strathearn, L., Gray, P. H., O'Callaghan, F., & Wood, D. O. (2001). Childhood neglect and cognitive development in extremely low birth weight infants: A prospective study. *Pediatrics*, 108(1), 142-151.
- Johnson, D. E., & Gunnar, M. R. (2011), IV. Growth failure in institutionalized children. Monographs of the Society for Research in Child Development, 76(4), 92–126.
- Macovei, O. (1986). The Medical and Social Problems of the Handicapped in Children's Institutions in Iasi Bucharest, Romania. Bucharest, Romania: Institutl de Igiena si Sanatate Publica.

- 47. Miller, L. C., Kiernan, M. T., Mathers, M. I., & Klein-Gitelman, M. (1995). Developmental and nutritional status of internationally adopted children. Archives of Pediatrics & Adolescent Medicine, 149(1), 40-44.
- 48. McEwen, B. S., Biron, C. A., Brunson, K. W., Bulloch, K., Chambers, W.H., Dhabhar, F. S., ...& Weiss, J. M. (1997). Neural-endocrine-immune interactions: the role of adrenocorticoids as modulators of immune function in health and disease. Brain Research Review, 23(1-2), 79-133
- 49. Bakermans-Kranenburg, M.J. Steele, H., Zeanah, C. H., Muhamedrahimov, R. J., Vorria, P., Dobrova-Krol, N. A., Steele, M., van IJzendoorn, M. H., Juffer, F., & Gunnar, M. R. (2011). III. Attachment and emotional development in institutional care: Characteristics and catch-up. Monographs of the Society for Research in Child Development, 76(4), 62-91.
- 50. Carlson, V., Cicchetti, D., Barnett, D., & Braunwald, K. (1989). Disorganized/disoriented attachment relationships in maltreated infants. Developmental Psychology, 25(4), 525-531.
- 51. Crittenden, P. M., & Ainsworth, M. D. S. (1989). Child maltreatment and attachment theory. In D. Cicchetti & V. Carlson (Eds.), Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect (pp. 432-464). New York: Cambridge University Press.
- 52. Egeland, B., & Sroufe, L. (1981). Attachment and early maltreatment. Child Development, 52(1), 44-52.
- 53. Hesse, E., & Main, M. (2000). Disorganized infant, child, and adult attachment: Collapse in behavioral and attentional strategies. Journal of the American Psychoanalytic Association, 48(4), 1097-1127.
- 54. Lyons-Ruth, K., Connell, D. B., Grunebaum, H. U., & Botein, S. (1990). Infants at social risk: Maternal depression and family support services as mediators of infant development and security of attachment. Child Development, 61(1), 85-98.
- 55. Camras, L. A., & Rappaport, S. (1993). Conflict behaviors of maltreated and nonmaltreated children. Child Abuse & Neglect, 17(4), 455-464.
- 56. Erickson, M., Egeland, B., & Pianta, R. (1989). The effects of maltreatment on the development of young children. In D. Cicchetti & V. Carlson (Eds.), Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect (pp. 647-684). New York: Cambridge University Press.
- 57. Hoffman-Plotkin, D., & Twentyman, C. T. (1984). A multimodal assessment of behavioral and cognitive deficits in abused and neglected preschoolers. Child Development, 55(3), 794-802.
- 58. Bolger, K. E., Patterson, C. J., & Kupersmidt, J. B. (1998). Peer relationships and self-esteem among children who have been maltreated. Child Development, 69(4), 1171-1197.
- 59. Kaufman, J., & Cicchetti, D. (1989). Effects of maltreatment on school-age children's socioemotional development: Assessments in a day-camp setting. Developmental Psychology, 25(4), 516-524.
- 60. Manly, J., Kim, J. E., Rogosch, F. A., & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. Development and Psychopathology, 13(4), 759-782.
- 61. Chapple, C. L., Tyler, K. A., & Bersani, B. E. (2005). Child neglect and adolescent violence: Examining the effects of self-control and peer rejection. Violence and Victims, 20, 39-54.

- 62. Crittenden, P. M., & DiLalla, D. L. (1988). Compulsive compliance: The development of an inhibitory coping strategy in infancy. Journal of Abnormal Child Psychology, 16(5), 585-599.
- 63. Koenig, A. L., Cicchetti, D., & Rogosch, F. A. (2000). Child compliance/noncompliance and maternal contributors to internalization in maltreating and non-maltreating dyads. Child Development, 71(4), 1018-1032.
- 64. Vorria, P., Papaligoura, Z., Sarafidou, J., Kopakaki, M., Dunn, J., van Ijzendoorn, M.H., & Kontopoulou, A. (2006). The development of adopted children after institutional care: A follow-up study. Journal of Child Psychology and Psychiatry, 47(12), 1246-1253.
- 65. Johnson, J. J., Smailes, E. M., Cohen, P., Brown, J., & Bernstein, D. P. (2000). Associations between four types of childhood neglect and personality disorder symptoms during adolescence and early adulthood: Findings of a community-based longitudinal study. Journal of Personality Disorders, 14(2), 171-187.
- 66. Toth, S. L., Cicchetti, D., Macfie, J., & Emde, R. N. (1997). Representations of self and other in the narratives of neglected, physically abused, and sexually abused preschoolers. Development and Psychopathology, 9(4),
- 67. Toth, S. L., Cicchetti, D., Macfie, J., Maughan, A., & Vanmeenen, K. (2000). Narrative representations of caregivers and self in maltreated preschoolers. Attachment & Human Development, 2, 271-305.
- 68. Waldinger, R. J., Toth, S. L., & Gerber, A. (2001). Maltreatment and internal representations of relationships: Core relationship themes in the narratives of abused and neglected preschoolers. Social Development, 10, 41-58.
- 69. Ogawa, J. R., Sroufe, L., Weinfield, N. S., Carlson, E. A., & Egeland, B. (1997). Development and the fragmented self: Longitudinal study of dissociative symptomatology in a nonclinical sample. Development and Psychopathology, 9(4), 855-879.
- 70. Spertus, I., Yehuda, R., Wong, C., Halligan, S., & Seremetis, S. (2003). Childhood emotional abuse and neglect as predictors of psychological and physical symptoms in women presenting to a primary care practice. Child Abuse and Neglect, 27(11), 1247-1258.
- 71. Widom, C. S. (1989). The cycle of violence. Science, 244(4901), 160-166.
- 72. Luntz, B. K., & Widom, C. (1994). Antisocial personality disorder in abused and neglected children grown up. The American Journal of Psychiatry, 151(5), 670-674.
- 73. Maxfield, M. G., & Widom, C. S. (1996). The cycle of violence: Revisited six years later. Archives of Pediatrics Adolescent Medicine, 150(4), 390-395.
- 74. Nelson, C., Zeanah, C. H., Fox, N. A., Marshall, P. J., Smyke, A. T., & Guthrie, D. (2007). Cognitive recovery in socially deprived young children: The Bucharest Early Intervention Project. Science, 318, 1937-1940.
- 75. Pears, K. & Fisher, P.A. (2005). Developmental, cognitive, and neuropsychological functioning in preschool-aged foster children: Associations with prior maltreatment and placement history. Journal of Developmental & Behavioral Pediatrics, 26(2), 112-122.
- 76. Smyke, A. T., Koga, S. F., Johnson, D. E., Fox, N. A., Marshall, P. J., Nelson, C. A., ... & Group, B. C. (2007). The caregiving context in institution-reared and familyreared infants and toddlers in Romania. Journal of Child Psychology & Psychiatry, 48(2), 210-218.
- 77. Loman, M., Wiik, K., Frenn, K., Pollak, S., & Gunnar, M. (2009). Postinstitutionalized children's development:

- Growth, cognitive, and language outcomes. Journal of Development and Behavioral Pediatrics, 30(5), 426–434.
- 78. Beckett, C., Maughan, B., Rutter, M., Castle, J., Colvert, E., Groothues, C., ... & Sonuga-Barke, E. (2007). Scholastic attainment following severe early institutional deprivation: A study of children adopted from Romania. Journal of Abnormal Child Psychology, 35(6), 1063-1073.
- 79. Allen, R. E., & Oliver, J. M. (1982). The effects of child maltreatment on language development. Child Abuse & Neglect, 6(3), 299-305.
- 80. Culp, R. E., Watkins, R. V., Lawrence, H., Letts, D., Kelly, D. J., & Rice, M. L. (1991). Maltreated children's language and speech development: abused, neglected, and abused and neglected. First Language, 11(33), 377-389.
- 81. Gowan, J. (1993). Effects of neglect on the early development of children: Final report. Washington, D. C.: National Clearinghouse on Child Abuse and Neglect, Administration for Children & Families.
- 82. Eckenrode, J., Laird, M., & Doris, J. (1993). School performance and disciplinary problems among abused and neglected children. Developmental Psychology, 29, 53-62.
- 83. Egeland, B. (1991). A longitudinal study of high risk families: Issues and findings. In R. Starr & D. A. Wolfe (Eds.), The effects of child abuse and neglect (pp. 33–56). New York: Guilford Press.
- 84. Wodarski, J. S., Kurtz, P. D., Gaudin, J. M., & Howing, P. T. (1990). Maltreatment and the school-aged child: Major academic, socioemotional, and adaptive outcomes. Social Work, 35(6), 506-513.
- 85. Perez, C. M., & Widom, C. (1994). Childhood victimization and long-term intellectual and academic outcomes. Child Abuse & Neglect, 18(8), 617-633.
- 86. Pears, K., Bruce, J., Fisher, P., & Kim, H. (2010). Indiscriminate friendliness in maltreated foster children. Child Maltreatment, 15, 64-75.
- 87. Kreppner, J. M., O'Connor, T. G., Rutter, M., & English and Romanian Adoptees Study Team. (2001). Can inattention/overactivity be an institutional deprivation syndrome? Journal of Abnormal Child Psychology, 29(6), 513-528.
- 88. Bos, K. J., Fox, N., Zeanah, C. H., & Nelson, C. (2009). Effects of early psychosocial deprivation on the development of memory and executive function. Frontiers In Behavioral Neuroscience, 3, 16.
- 89. Pollak, S. D., Nelson, C. A., Schlaak, M. F., Roeber, B. J., Wewerka, S. S., Wiik, K. L., ... & Gunnar, M. R. (2010). Neurodevelopmental effects of early deprivation in post-institutionalized children. Child Development, 81, 224-236.
- 90. McDermott, J., Westerlund, A., Zeanah, C. H., Nelson, C.A., & Fox, N.A. (2012). Early adversity and neural correlates of executive function: Implications for academic adjustment. Developmental Neuroscience, 2, S55-66.
- 91. Pine, D. S., Mogg, K., Bradley, B. P., Montgomery, L., Monk, C. S., McClure, E., ... & Kaufman, J. (2005). Attention bias to threat in maltreated children: Implications for vulnerability to stress-related psychopathology. The American Journal Of Psychiatry, 162(2), 291-296.
- 92. Dozier, M., Lindhiem, O., Lewis, E., Bick, J., Bernard, K., & Peloso, E. (2009). Effects of a foster parent training program on young children's attachment behaviors: Preliminary evidence from a randomized clinical trial. Child & Adolescent Social Work Journal, 26(4), 321-332.

- 93. Dozier, M., Peloso, E., Lindhiem, O., Gordon, M., Manni, M., Sepulveda, S., ... & Levine, S. (2006). Developing evidence-based interventions for foster children: An example of a randomized clinical trial with infants and toddlers. Journal of Social Issues, 62(4), 767-785.
- 94. Fisher, P. A., Gunnar, M. R., Chamberlain, P., & Reid, J. B. (2000). Preventive intervention for maltreated preschool children: Impact on children's behavior, neuroendocrine activity, and foster parent functioning. Journal of the American Academy of Child & Adolescent Psychiatry, 39(11), 1356-1364.
- 95. Fisher, P. A., & Kim, H. K. (2007). Intervention effects on foster preschoolers' attachment-related behaviors from a randomized trial. Prevention Science, 8(2), 161-170.
- 96. Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. Psychoneuroendocrinology, 32(8-10), 892-905.
- 97. Bernard, K., Dozier, M., Bick, J., Lewis, E., Lindhiem, O., & Carlson, E. (2012). Enhancing attachment organization among maltreated children: Results of a randomized clinical trial. Child Development, 83(2), 623-636.
- 98. Dozier, M., Peloso, E., Lewis, E., Laurenceau, J., & Levine, S. (2008). Effects of an attachment-based intervention of the cortisol production of infants and toddlers in foster care. Development and Psychopathology, 20(3), 845-859.
- 99. Fisher, P. A., Gunnar, M., Dozier, M., Bruce, J., & Pears, K. C. (2006). Effects of a therapeutic intervention for foster children on behavior problems, caregiver attachment, and stress regulatory neural systems. Annals of the New York Academy of Sciences, 1094, 215-225.
- 100. Moulson, M. C., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2009). Early adverse experiences and the neurobiology of facial emotion processing. Developmental Psychology, 45(1), 17-30.
- 101. Vanderwert, R. E., Marshall, P. J., Nelson, C., Zeanah, C. H., & Fox, N. A. (2010). Timing of intervention affects brain electrical activity in children exposed to severe psychosocial neglect. PLoS ONE, 5(7), e11415.
- 102. Colvert, E., Rutter, M., Kreppner, J., Beckett, C., Castle, J., Groothues, C., ... & Sonuga-Barke, E. S. (2008). Do theory of mind and executive function deficits underlie the adverse outcomes associated with profound early deprivation?: Findings from the English and Romanian adoptees study. Journal of Abnormal Child Psychology, 36(7), 1057-1068.
- 103. Van IJzendoorn, M.H. & Juffer, F. (2006). The Emanuel Miller Memorial Lecture 2006: Adoption as intervention. Meta-analytic evidence for massive catch-up and plasticity in physical, socio-emotional, and cognitive development. Journal of Child Psychology and Psychiatry, 47(12), 1228-1245.
- 104. Marshall, P. J., Reeb, B. C., Fox, N. A., Nelson, C., & Zeanah, C. H. (2008). Effects of early intervention on EEG power and coherence in previously institutionalized children in Romania. Development and Psychopathology, 20(3), 861-880.
- 105. Gunnar, M., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. Development and Psychopathology, 13(3), 611-628.
- 106. Hodges, J., & Tizard, B. (1989). IQ and behavioural adjustment of ex-institutional adolescents. Journal of Child Psychology and Psychiatry, 30, 53-75.
- 107. Kreppner, J., Kumsta, R., Rutter, M., Beckett, C., Castle, J., Stevens, S. & Sonuga-Barke, E. J. (2010). IV. Developmental course of deprivation-specific

- psychological patterns: Early manifestations, persistence to age 15, and clinical features. Monographs of the Society for Research in Child Development, 75, 79-101.
- 108. Rutter, M. and the English and Romanian Adoptees (ERA) study team. (1998). Developmental catch-up, and deficit, following adoption after severe global early privation. Journal of Child Psychology and Psychiatry, 39(4), 465-476.
- 109. Verhulst, F. C., Althaus, M., & Versluis-den Bieman, H. J. (1990). Problem behavior in international adoptees: II. Age at placement. Journal of the American Academy of Child and Adolescent Psychiatry, 29(1), 104-111.
- 110. Verhulst, F. C., Althaus, M., & Versluis-den Bieman, H. J. (1992). Damaging backgrounds: Later adjustment of international adoptees. Journal of the American Academy of Child and Adolescent Psychiatry, 31(3), 518-524.
- 111. Rutter, M., Beckett, C., Castle, J., Colvert, E., Kreppner, J., Mehta, M., ... & Sonuga-Barke, E. (2007). Effects of profound early institutional deprivation: An overview of findings from a UK longitudinal study of Romanian adoptees. European Journal of Developmental Psychology, 4(3), 332-350.
- 112. Bruce, J., McDermott, J. M., Fisher, P. A., & Fox, N. A. (2009). Using behavioral and electrophysiological measures to assess the effects of a preventive intervention: A preliminary study with preschool-aged foster children. Prevention Science, 10(2), 129-140.
- 113. Fisher, P. A., & Stoolmiller, M. (2008). Intervention effects on foster parent stress: Associations with child cortisol levels. Development and Psychopathology, 20(3), 1003-1021.
- 114. Lieberman, A.F., Weston, D. R., & Pawl, J. H. (1991). Preventive intervention and outcome with anxiously attached dyads. Child Development, 62(1), 199-209.
- 115.Lieberman, A.F., Van Horn, P.J., & Ghosh Ippen, C. (2005). Toward evidence-based treatment: Child-Parent Psychotherapy with preschoolers exposed to marital violence. Journal of the American Academy of Child and Adolescent Psychiatry, 44(12), 1241-1248.
- 116. Lieberman, A.F., Ghosh Ippen, C., Van Horn, P.J. (2006). Child-Parent Psychotherapy: Six month follow-up of a randomized control trial. Journal of the American Academy of Child and Adolescent Psychiatry, 45(8), 913-918.
- 117. Ghosh Ippen, C., Harris, W.W., Van Horn, P., & Lieberman, A.F. (2011). Traumatic and stressful events in early childhood: Can treatment help those at highest risk? Child Abuse and Neglect, 35(7), 504-513.
- 118.Cicchetti, D., Rogosch, F.A., & Toth, S.L. (2006). Fostering secure attachment in infant in maltreating families through preventive interventions. Development and Psychopathology, 18(3), 623-649.
- 119. Toth S.L., Maughan A., Manly J.T., Spagnola M., Cicchetti D. (2002). The relative efficacy of two interventions in altering maltreated preschool children's representational models: Implications for attachment theory. Developmental Psychopathology, 14(4), 877-908.
- 120. Cicchetti D., Toth S.L., Rogosch F.A. (1999). The efficacy of toddler-parent psychotherapy to increase attachment security in offspring of depressed mothers. Attachment and Human Development, 1(1), 34-66.
- 121. Cicchetti, D., Rogosch, F.A., & Toth, S.L. (2000). The efficacy of Toddler-Parent Psychotherapy for fostering cognitive development in offspring. Journal of Abnormal Child Psychology. 28(2), 135-148.

122. Toth, S. L., Rogosch, F. A., & Cicchetti, D. (2006). The efficacy of Toddler-Parent Psychotherapy to reorganize attachment in the young offspring of mothers with major depressive disorder: A randomized preventive trial. Journal of Consulting and Clinical Psychology, 74(6), 1006-1016.

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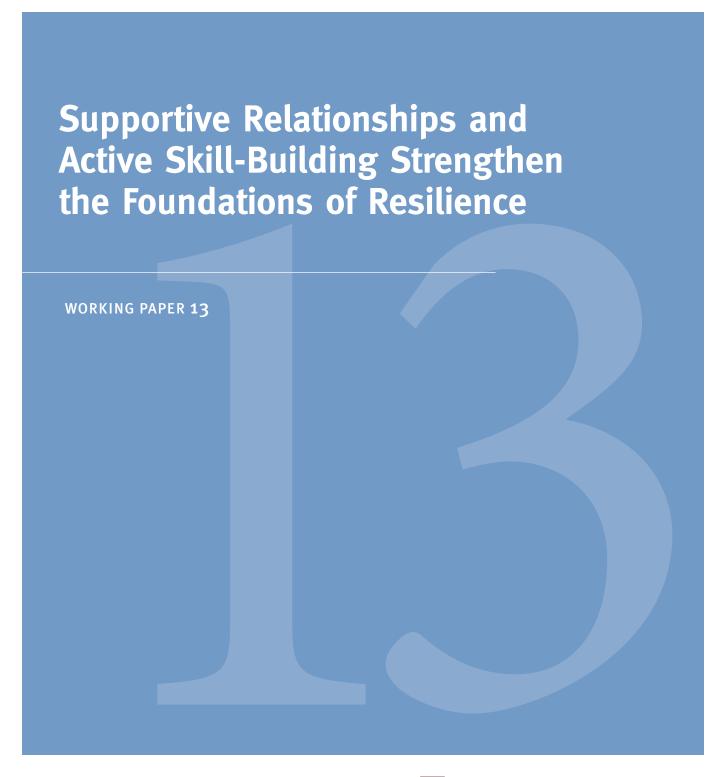
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The National Scientific Council on the Developing Child is a multidisciplinary, multi-university collaboration designed to bring the science of early childhood and early brain development to bear on public decision-making. Established in 2003, the Council is committed to an evidence-based approach to building broad-based public will that transcends political partisanship and recognizes the complementary responsibilities of family, community, workplace, and government to promote the well-being of all young children. For more information, visit www.developingchild.net.

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### The Issue

THE FUTURE PROSPERITY OF ANY SOCIETY DEPENDS ON A CONTINUING INVESTMENT IN THE HEALTHY development of the next generation. The well-documented connection between adverse early experiences and a wide range of costly problems, such as lower school achievement and higher rates of criminal behavior and chronic disease, underscores the extent to which reducing the burdens of significant adversity on families with young children must be a critical part of that investment. That said, not all children exposed to stressful circumstances experience detrimental consequences. A better understanding of why some do well despite serious hardship could inform more effective policies and programs to provide support for families and help more disadvantaged children reach their full potential.

Decades of research in the behavioral and social sciences have produced a rich knowledge base that explains why some people develop the adaptive capacities to overcome significant adversity and others do not. Whether the burdens come from the hardships of poverty, the challenges of parental substance abuse or serious mental illness, the stresses of war, the threats of recurrent violence or chronic neglect, or a combination of factors, the single most common finding is that children who end up doing well have had at least one stable and committed relationship with a supportive parent, caregiver, or

other adult. These relationships provide the personalized responsiveness, scaffolding, and protection that buffer children from developmental disruption. They also build key capacities—such as the ability to plan, monitor and regulate behavior, and adapt to changing circumstances—that enable children to respond to adversity and to thrive. This combination of supportive relationships, adaptive skill-building, and positive experiences constitutes the foundations of what is commonly called *resilience*.

Recent discoveries in molecular biology, genomics, and epigenetics provide remarkable

### What Is Resilience?

In the social, behavioral, and biological sciences, the term resilience is used in a variety of ways and contexts—sometimes as an individual characteristic, sometimes as a process, and sometimes as an outcome. Despite these differences, there is a set of common, defining features of resilience that illustrates how the concept has been used in research and intervention sciences. These features include the following:

- The capacity of a dynamic system to adapt successfully to disturbances that threaten its function, viability, or development.<sup>8</sup>
- **2.** The ability to avoid deleterious behavioral and physiological changes in response to chronic stress. <sup>18</sup>
- **3.** A process to harness resources to sustain well-being.<sup>76</sup>
- **4.** The capacity to resume positive functioning following adversity.<sup>77</sup>
- A measure of the degree of vulnerability to shock or disturbance.<sup>78</sup>

- **6.** A person's ability to adapt successfully to acute stress, trauma, or more chronic forms of adversity.<sup>11</sup>
- 7. The process of adapting well in the face of adversity, trauma, tragedy, threats, or significant sources of stress.<sup>79</sup>

Whether it is considered an outcome, a process, or a capacity, the essence of resilience is a positive, adaptive response in the face of significant adversity. It is neither an immutable trait nor a resource that can be used up. On a biological level, resilience results in healthy development because it protects the developing brain and other organs from the disruptions produced by excessive activation of stress response systems. Stated simply, resilience transforms potentially *toxic* stress into *tolerable* stress. In the final analysis, resilience is rooted in both the physiology of adaptation and the experiences we provide for children that either promote or limit its development.

new insights into the underlying causal mechanisms that explain how supportive relationships build the capacities to deal with adversity. This rapidly advancing research frontier demonstrates that resilience is the result of multiple interactions among protective factors in the social environment *and* highly responsive biological systems. These findings provide an opportunity to examine how current policies and programs could be enhanced to produce more favorable life outcomes for disadvantaged children, both by reducing their exposure to sources of adversity and by designing better ways of building their coping skills and adaptive capacities.

The answer to this challenge begins with extensive scientific evidence that the development of healthy brain architecture is influenced by consistent, "serve and return" interactions between young children and their primary caregivers. When these experiences are unavailable or repeatedly disrupted, the body perceives their absence as a serious threat, and activates its stress response systems. Although the immediate effects of the stress response are protective, its excessive or prolonged activation produces physiological changes that can have a wear and tear effect on the developing brain,

cardiovascular system, immune function, and metabolic regulatory systems—in short, it becomes toxic stress.<sup>2,3</sup> In contrast, when responsive interactions with caring adults are provided or restored, stress response systems return to their normal baselines, the developing brain and other maturing organ systems are protected from disruption, and children are helped to develop the coping skills needed to deal with adversity. The net result of these protective effects is that what could have been a toxic stress experience for a child becomes what we call "tolerable stress."

One way to understand the development of resilience is to visualize how protective experiences and adaptive skills both counterbalance significant adversity and produce positive outcomes. This can be illustrated through the concept of a balance scale or perhaps a seesaw or teeter-totter (see box). In this model, resilience is evident when a child's health and development are tipped in the positive direction, even when a heavy load of negative factors is stacked on the other side. Understanding all of the influences that might tip the scale in the positive direction is critical to devising more effective strategies for promoting healthy development in the face of significant disadvantage.

### What Science Tells Us

OVER THE PAST FEW DECADES, THERE HAVE BEEN numerous longitudinal studies of children's development under conditions of adversity that typically lead to toxic stress responses. The power of this research lies in the compilation of rich datasets from the same individuals over an extended period of time, often beginning at birth or even prenatally and, in some instances, continuing well into adulthood.

Many of these studies have identified a subset of children whose life outcomes were remarkably positive despite their exposure to a variety of adverse experiences that typically produce increased risks for impairments in learning, behavior, and both mental and physical health. Gaining a greater understanding of how and why these unexpected outcomes happen is helping to build a more robust science of resilience. This science can stimulate fresh thinking about how to enhance the life prospects of all chil-

dren—especially those living in environments that can prompt toxic stress responses. The observations and evidence described in the following sections provide a strong first step toward achieving that goal.

**Resilience results from a dynamic interaction between internal predispositions and external experiences.** Children who do well in the face of significant disadvantage typically exhibit both an intrinsic resistance to adversity *and* strong relationships with the important adults in their family and community. Indeed, it is the *interaction* between biology and environment that builds the capacities to cope with adversity and overcome threats to healthy development. Resilience, therefore, is the result of a combination of protective factors—and neither individual characteristics nor social environments alone are likely to generate sufficiently positive out-

# **Tipping the Scale Toward Positive Outcomes**

Child development is like a balance scale with two sides. Experiences that can result in toxic stress, such as repeated or chronic exposure to violence, poverty, or maltreatment, pile on the negative side of the scale. Positive influences that can help make significant stress tolerable, such as supportive relationships, skill-building opportunities, and practice dealing with manageable challenges, tip the scale the other way. Part of the reason for the variability in how individual children develop is that their scales can be loaded and tipped in different ways. Even under highly adverse conditions, development can proceed in a positive direction if parents and other caregivers provide consistent responsiveness, and if communities provide resources and supports that strengthen families' capacities and make a broader environment of protective relationships accessible to all children.

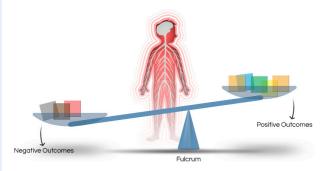
There is another part of the scale that affects how it tips, called the fulcrum. As with any scale or seesaw, if a child's fulcrum is placed closer to one end than the other, it becomes harder to tip the scale in that direction. In this representation, the initial placement of the fulcrum represents individual predispositions, which vary from one child to another. These variations in temperament and innate abilities, which reflect underlying genetic differences, mean that individual children start with their fulcrums in different places along the scale. This placement affects how they respond to the weight of experiences they have—whether minor adversity will tip the child's scale toward poor outcomes, for example, or whether major therapeutic intervention is needed to tip the scale toward positive outcomes.

Although the initial placement of the fulcrum has an early impact on a child's developmental trajectory, advances in science are now showing us that the position of the fulcrum is not fixed. To the contrary, the cumulative impacts of life experiences that tip the scale in either direction can also shift the fulcrum's location over time. Stated differently, the continuing accumulation of positive and negative experiences over time actually influences the child's mental and physical constitution—and thus has the power to slide the fulcrum.

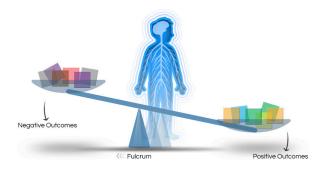
One way to actively move the fulcrum to a position that makes the scale better able to bear the weight of negative experiences is to build the capabilities needed to manage stress. These include the ability to focus attention, solve problems, plan ahead, adjust to new circumstances, regulate behavior, and control impulses. These skills, many of which fall within what is called executive function and selfregulation, constitute important building blocks for dealing



When positive experiences outweigh negative experiences, a child's "scale" tips toward positive outcomes.



The initial placement of the fulcrum affects how easily the scale tips toward positive or negative outcomes.



Over time, the cumulative impact of positive life experiences and coping skills can shift the fulcrum's position, making it easier to achieve positive outcomes.

with adversity, and the mastery of these skills can positively reposition the fulcrum. It is important to note that the fulcrum's position is never completely locked. However, the brain's ability to change decreases with age, making it more difficult to shift the fulcrum's location as children get older.

comes for children who experience prolonged periods of toxic stress.

Resilience is seen in how the brain, the immune system, and genes respond to experiences during development. A deeper understanding of why and how some children have unexpectedly positive outcomes despite adversity is beginning to emerge as new scientific discoveries are illuminating the complex interplay among genetic differences, developing brain circuitry, and immune responsiveness, all of which interact with the caregiving environment and social context.<sup>11</sup>

• Gene expression: Given the extensive evidence that virtually all aspects of development and health are affected by the interaction between genes and experience, 12 scientists have begun to identify specific biological factors (including variations in gene sequence, gene expression, and neural mechanisms) that work together with aspects of the social environment to generate positive outcomes. 13-21 For example, certain genetic variants result in the production of proteins in the brain that control the chronic stress response, either exaggerating or blunting the negative effects of exposure to adversity.<sup>22-25</sup> Biological differences also control the sensitivity of genes to environmental influences,26 which leads to

# Biological differences control the sensitivity of genes to environmental influences, affecting how individuals respond to stressful experiences.

different ways in which individuals respond to stressful experiences.<sup>21</sup> Certain genetic variations have also been shown to enhance the beneficial effects of a protective intervention, making some individuals more likely to thrive in response to supportive environments.<sup>27</sup>

 Brain function: Variation in the activation of brain chemicals, such as oxytocin and vasopressin, is related to the ability to initiate and sustain social behavior, form attachments with others, and manage social anxiety throughout life.<sup>28,29</sup> Functional differences in the brain's fear and reward circuits may also be responsible for capacity-building traits such as optimism or emotion regulation. 30,31 Many of these differences have roots in the way that early experiences affect brain development. For example, threatening situations cause a number of stress hormones to be released. Certain combinations of these hormones enhance brain function after mild to moderate stress, but suppress it after severe, acute stress. Sustained stress can even alter the size and number of neural connections in certain parts of the brain. When the danger passes, a healthy, resilient brain can recover from these changes, but early life adversity can alter that capacity for recovery. Moreover, chronic stress triggered by early adversity can cause long-term changes in brain regions that manage behavioral control and emotional wellness. These changes limit the brain's ability to respond appropriately to challenging or threatening situations, predisposing individuals toward the development of depression, anxiety disorders, substance abuse, and cardiovascular disease in adulthood.32-34

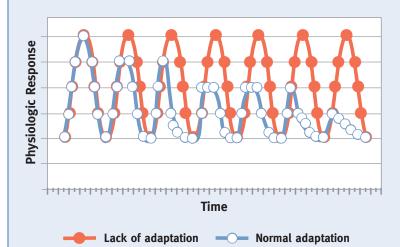
• Immune-related responses: Chemicals activated by the immune system, which are produced and expressed in the brain and other parts of the body, are also regulated by both genetic and environmental factors. 18 Inflammation, which is a physiological mechanism of selfprotection over the short term, is a serious threat if activated chronically. Indeed, chronic inflammation can lead to a variety of illnesses, including diabetes, cardiovascular disease, arthritis, cancer, dementia, and depression. Given the centrality of inflammation to multiple diseases, the fact that early life adversity is associated with elevated inflammatory responses suggests that toxic stress increases the probability of lifelong health impairments. For example, the experience of abuse and neglect increases the production of pro-inflammatory cytokines, which are an early marker of greater risk for heart disease.35 The body's ability to restore balance between pro- and anti-inflammatory cytokines in the aftermath of a stress-inducing experience can therefore be considered one biological indicator of resilience.<sup>18</sup> Scientific evidence is also building that differences in the types and amounts of microbes (bacteria and viruses) to which infants and young children are exposed may affect the responsiveness and adaptability of the immune system to later environmental challenges.36

Multiple lines of research have identified a common set of factors that predispose children to positive outcomes in the face of significant adversity. These factors encompass strengths that derive from the child, the family, peer and adult relationships, and the broader social environments that build and support sturdy brain architecture. When these positive influences are operating effectively, they "stack the scale" with positive weight and optimize resilience.8,37 When these positive factors are absent, disrupted, or undermined, there is little to counterbalance the negative effects of significant adversity, thus creating the conditions for poor outcomes and diminished life prospects. These counterbalancing factors include the following:

- The availability of at least one *stable*, *caring*, and supportive relationship between a child and the important adults in his or her life. These relationships begin in the family, but they can also include neighbors, providers of early care and education, teachers, social workers, or coaches, among many others.
- Helping children build a sense of *mastery* over their life circumstances. Those who believe in their own capacity to overcome hardships and guide their own destiny are far more likely to adapt positively to adversity.
- Children who develop strong executive function and self-regulation skills. These skills enable individuals to manage their own behavior and emotions,<sup>38</sup> and develop and execute adaptive strategies to cope effectively with difficult circumstances.
- The supportive context of affirming faith or cultural traditions. Children who are solidly grounded within such traditions are more likely to respond effectively when challenged by a major stressor or a severely disruptive experience.39-41

Learning to cope with manageable threats to our physical and social well-being is critical for the development of resilience. Not all stress is harmful; all children experience varying degrees of stress in the course of their day-to-day lives. From the impacts of minor infections or abrasions, which trigger immune reactions that activate the body's stress response, to the threat of

## **Experiences and Coping Skills Build Resilience to Adversity**



When we experience something stressful, our body's stress response systems are activated. A healthy physiological stress response is characterized by a sharp increase followed by a rapid decrease in activation. When the system is resilient, it adapts over time (depicted above in blue), leading to less activation each time a similar stressor is experienced. But when the stress response does not activate the way it should, fails to turn off when the stressful experience is over, or fails to recognize and adapt to the same type of stressor over time, we know that it is not working properly. In the latter case (depicted above in red), the same physiological response is triggered over and over with no signs of adaptation. When this happens, it can upset the body's chemical balance and change the architecture of specific regions of the developing brain. A resilient brain adapts to similar types of non-life-threatening stressors by adopting coping skills based on experience. As a result, the stress response system "learns" to activate more moderately.

Source: McEwen (1998). 80

social exclusion, failing a test, or flubbing one's lines in a play, there are numerous opportunities in every child's life to experience manageable stress—and with the help of supportive adults, this "positive stress" can be growthpromoting. Over time, both our bodies and our brains begin to perceive these kinds of threats as increasingly manageable and we become better able to cope with life's obstacles and hardships, both physically and mentally. 42,43

One promising approach to strengthening adaptability is through the development of explicit skills and capabilities that support cognitive flexibility, goal-setting, problem-solving, and the ability to resist impulsive behavior.

Many of these skills fall within the domains of executive function and self-regulation,<sup>38</sup> and have a lengthy developmental trajectory that begins in infancy and does not fully mature until age 25 to 30 years.<sup>44</sup> Just as these skills serve as protective factors for children (see above), they are critically important capabilities for the adults who care for them—and can be strengthened through coaching and practice.<sup>45</sup>

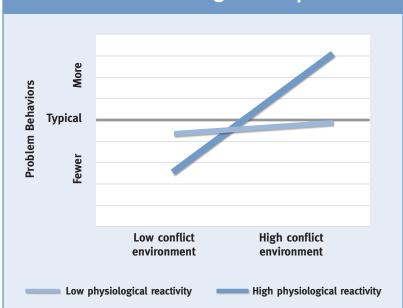
Some children demonstrate greater sensitivity to both negative and positive experiences. A growing body of evidence illustrates that some children experience more extreme biological responses to social contexts of all kinds than other children. These highly sensitive individuals show increased vulnerability in stress-

ful circumstances but respond in exceptionally positive ways under supportive conditions. 46 Biologically sensitive children are more responsive to positive environments that provide parental warmth and supportive interventions, 27, 47 and easily overloaded in stressful caregiving contexts that are burdened by marital conflict, overall family adversity, and parental psychiatric disorders such as depression. 48-50 This heightened susceptibility to the consequences of adversity has also been connected to higher rates of respiratory illnesses, 51,52 as well as to depression and behavior problems. 53

Resilience can be situation-specific. Research shows that differences that protect some children in the face of one form of adversity may have little or no effect in other conditions. <sup>54,55</sup> For example, some children may demonstrate resilience in response to being bullied at school but not to witnessing parental conflict. Others may demonstrate resilience in achieving some kinds of positive outcomes (e.g., academic performance) but not others (e.g., risk of stress-related disease). <sup>56,57</sup> In short, resilience is often situation-specific, rather than a general trait that applies in all contexts.

How individuals respond to stressful experiences varies dramatically, but extreme adversity nearly always generates serious problems that require treatment. Most children do not experience the unpredictable, uncontrollable, chronic stressors that can lead to lifelong negative consequences. However, children who experience circumstances of threat or catastrophe of historic magnitude-such as genocide, famine, or environmental devastation—almost always exhibit short-term and/or long-term impairments in their health and development. 58-60 For example, studies of children who survived the Holocaust during World War II, many of whom showed remarkable resilience in the face of horrific atrocities, reported residual vulnerabilities to psychiatric symptoms well into the adult years.<sup>61</sup> Irrespective of constitutional strengths or the availability of supportive relationships that help build capacities to deal with a wide range of challenges or threats, extreme adversity can rarely be weathered without harm. Under such conditions, intensive therapeutic interventions tailored to individuals and contexts are often needed.

## Some Children Are Affected More by Both Positive *and* Negative Experiences



Each child has a different level of physiological reactivity—the degree to which one's biological systems trigger a response to an external event. In this study, children who have higher reactivity (dark blue line) showed more problem behaviors when they lived in an environment with significant conflict than their less reactive peers (light blue line). However, in a low-conflict environment, highly reactive children responded with far fewer problem behaviors than their counterparts. Behaviors included oppositional defiant behaviors (anger, resentment, arguing), conduct problem behaviors (cheating, vandalizing, threatening), and overt hostility behaviors (fighting, kicking, taunting).

Source: Obradović, et al (2011).53

## Facts About Resilience That Are Often Misunderstood

SCIENTISTS HAVE STUDIED THE PHENOMENON of resilience in a wide range of perilous circumstances, including poverty,10 severe parental psychopathology,<sup>62</sup> conditions of racially motivated threat,63 institutional care,9 exposures to violence and war, 64,65 and the Holocaust during World War II.61 Consistent findings from this extensive knowledge base provide an opportunity to set the record straight about several widely believed but incorrect assumptions about individuals who beat the odds in the face of severe hardship.

Resilience requires relationships, not rugged individualism. There is no "resilience gene" that determines the life course of an individual irrespective of the experiences that shape genetic expression. The capacity to adapt and thrive despite adversity develops through the interaction of supportive relationships, gene expression, and adaptive biological systems. 8,18,66 Despite the widespread belief that individual grit, extraordinary self-reliance, or some in-born, heroic strength of character can triumph over calamity, science now tells us that it is the reliable presence of at least one supportive relationship and multiple opportunities for developing effective coping skills that are essential building blocks for the capacity to do well in the face of significant adversity.

The capabilities that underlie resilience can be strengthened at any age. A growing body of evidence shows that the coping skills that support effective adaptation in the face of adversity are built through a developmental process that occurs over an extended period of time, from infancy through adolescence and into the adult years. Age-appropriate activities that confer widespread health benefits (for the brain as well as for the rest of the body) hold considerable promise for improving the odds that an individual will recover from stress-inducing experiences. For example, increasing evidence suggests that regular physical exercise and stress-reduction practices (such as mindfulness meditation) at all ages can alter brain structure and function, while also reducing the expression of proinflammatory genes. 67-69 Programs that actively build skills for planning, organization, impulse control, cognitive flexibility, and other executive functions can also improve the abilities of adults with limited education and low income to cope with, adapt to, and even prevent adversity in their lives and in the lives of their children. 45

Individuals who demonstrate resilience in response to one form of adversity may not necessarily do so in response to another. Resilience is shaped by the accumulation of experiences-both good and bad-and the continuing development of adaptive coping skills that are attuned to those experiences. The brain and other biological systems are most adaptable early in life, and the development that occurs in the earliest years lays the foundation for a wide range of resilient behaviors. As individuals develop over time, they never completely lose their ability to hone these capabilities, but they often must learn how to adapt to new challenges. Nevertheless, when adverse experiences are extreme or cataclysmic, even the hardiest individual is likely to require therapeutic support at some point. Stated simply, resilience in the face of some hardships does not guarantee resilience in the face of all threatening circumstances.

## The Science-Policy Gap

WHEN OVERCOMING THE ODDS IS ERRONEOUSLY viewed as simply a matter of individual motivation or grit, the failure to succeed is perceived as the fault of the individual, and "blaming the victim" becomes the most frequent response. 8,70 Many economic, education, health, and social policies that address the effects of adversity in

individuals do little to create the conditions that are known to build greater resilience. The following examples illustrate the extent to which many public policies do not yet reflect the scientific understanding of how the capacities that support resilience develop.

When child welfare policies focus solely on removal of a child from an environment that is physically unsafe, they miss the opportunity to restore the relationships and build the capacities that underlie resilience. Removal from harm's way without also strengthening supportive relationships and providing therapeutic services does not provide the healing experiences necessary to counterbalance the negative effects of maltreatment and move toward positive outcomes. The science of resilience demands a critical shift from focusing exclusively on protection from imminent danger to adopting a strength-building approach that promotes the adaptive capacities that facilitate healthy development.

When poverty-reduction policies require parents to work without assuring access to affordable, high-quality child care, they miss the opportunity to promote both adult economic self-sufficiency and developmentally supportive experiences for their children. The failure to invest in state-of-the-art early care and education programs for the children of low-income, working parents reflects a fundamental misunderstanding of how the foundations of resilience are built early in life. Decades of research on child development suggest that programs that facilitate positive and stable adult-child relationships, both in the home and in the non-parental settings in which young children spend significant amounts of

time, are likely to reduce the intergenerational transmission of economic dependence and social disadvantage. Conversely, policies that neglect the basic needs of vulnerable young children miss critical opportunities that can "tip the scale" in a more positive direction and pay a lifetime of dividends for the individual and for society for generations to come.

When programs use "character education" models in contexts for which they were not designed, they miss the power of creating supportive, growth-promoting environments that build skills that generalize across contexts. Socially desirable character traits require a foundation of underlying skills and capabilities that include self-regulation and executive functions, such as inhibitory control, planning, and cognitive flexibility. Thus, programs that have been designed to "build character" in a context where children already have those underlying skills, such as high-achieving schools, are not necessarily going to transfer successfully to the different context of most low-achieving schools. In these latter circumstances, where children have not had the experiences needed to develop the same foundational capabilities, program staff must work on building supportive relationships and adaptive capacities that can be applied in multiple contexts before introducing conventional "character education" curricula.

## **Future Directions for Policy and Programs**

ADVANCES IN THE SCIENCE OF HUMAN DEVELOPment and its underlying biology can be mobilized to inform a new wave of innovative strategies for building the capabilities that help both children and adults thrive in the face of economic and social disadvantage. Promising new approaches include both public and private sector actions that can strengthen the foundations of resilience, beginning in the earliest years and continuing well into adulthood.

Use scientific knowledge to help identify and support children whose needs are not being addressed adequately by existing services. Individual differences in resilience and vulnerability among children facing significant adversity present important unmet challenges for in-

tervention programs that have been developed as a "one size fits all" model for service delivery. Drawing on new insights from 21st-century medicine, molecular biology, and genetics, as well as advances in the social sciences, researchers are beginning to identify interesting patterns of differential impact and new ways of measuring the variable effects of adversity that can strengthen our ability to match specific interventions to the distinctive resources and needs of different subgroups of children and families. These rapidly moving frontiers of scientific investigation could be mobilized to develop, test, and scale new ways of individualizing services for children who are more likely to exhibit resilience in severely challenging situations and those who will need greater assistance.

Enhance "serve and return" interactions between babies living in disadvantaged environments and the adults who care for them in order to strengthen the building blocks of resilience. The ability to respond to life's challenges in a positive, adaptive manner is rooted in the quality of the relationships that children have with their primary caregivers and other important individuals in their lives. The importance of this influence emerges directly from an understanding of how much serve and return responsiveness facilitates cognitive, social, and emotional development. These growing capabilities are then available to help a child cope with hardship and adapt to challenging situations. The knowledge and skills of parents, teachers, and caregivers greatly influence the responsiveness of their interactions with children. Recognizing the critical role of these interactive capabilities provides a strong incentive for developing new intervention strategies that explicitly target adult skillbuilding to improve the quality of adult-child relationships in order to improve life outcomes for vulnerable children.

Target the development of specific skills that are needed for adaptive coping, sound decisionmaking, and effective self-regulation in children and adults. Interventions to help individuals master stressful experiences are likely to be more effective if they target skills that can be used in a variety of circumstances and roles, whether as students, parents, job seekers, or community members. Many of these essential capabilities fall within the domains of executive function and self-regulation, which can be built through programs that focus explicitly on their development, beginning in early childhood, 38,71 and strengthened in adulthood through services that provide appropriate coaching, scaffolding, and practice.<sup>45</sup> A wide range of early care and education, parent training, and employment preparation programs could all benefit from a greater scientific understanding of how these skills develop from early infancy into the adult years.

Develop new frameworks for integrating policies and programs across sectors that collectively reduce adversity and build capacity. Some sources of significant adversity are out of one's control, such as natural disasters, the death of a loved one, and serious illnesses, yet most severe hardship

encountered by young children and their parents is preventable. Common triggers of toxic stress in families and communities include severe neglect, recurrent abuse, malnutrition, chaotic environments, and poor health management. Strategies that build child and adult capacities work best when they are integrated within complementary policies across sectors that collectively lower the burden of stress on families due to the often interrelated threats of poverty, crime, mental illness, substance abuse, discrimination, and

## Promising new approaches can strengthen the foundations of resilience, beginning in the earliest years and continuing into adulthood.

community violence. A fresh approach using a unified, science-based framework could identify the best strategies for coordinated public-private partnerships to implement together. These could include subsidized parental leave policies, access to affordable and high-quality early care and education services, community recreation and support activities, and home-visiting programs that coach new parents on how to interact positively with their children.

The following are some of many examples of how current policies and programs could build the foundations of resilience in children more effectively.

- Work requirements for receiving cash assistance through Temporary Assistance for Needy Families (TANF) could be linked directly to the availability of high-quality child care.
- Child welfare policies could work with families to reduce sources of chronic stress in their lives and provide therapeutic services to strengthen vulnerable relationships before the removal of children becomes necessary.
- Formal school settings can provide a range of opportunities for meaningful participation and belonging, as well as for the development of knowledge, cognitive skills, and self-regulation abilities, all of which augment adaptive systems that underlie the capacity to deal with adversity.8,72-73

Productive innovation is likely to have its greatest impact when family-based programs are designed to complement teacher-student programs with a common goal of assuring supportive and caring relationships as well as child skill-building.<sup>74</sup> School-based programs that focus explicitly on enhancing children's executive function skills and preventive interventions that foster secure attachment in infants where there is risk for maltreatment are promising examples of applying such knowledge.<sup>71,75</sup>

Finally, maximize the ultimate effectiveness of all early childhood policies and programs by focusing collectively on the full range of factors that facilitate resilience. Extensive evidence collected over decades of research points toward the powerful influence of a composite of personal, relational, and contextual factors that are associated with positive outcomes in the face of adversity. Drawing on this powerful knowledge base, all prevention and intervention programs would benefit from focusing on combinations of the following factors: (1) facilitating supportive adult-child relationships; (2) building a sense of self-efficacy and perceived control; (3) providing opportunities to strengthen adaptive skills and self-regulatory capacities; and (4) mobilizing sources of faith, hope, and cultural traditions.

## References

- Center on the Developing Child at Harvard University. (2012). The science of neglect: The persistent absence of responsive care disrupts the developing brain: Working paper no. 12. Retrieved from www.developingchild.harvard.edu.
- National Scientific Council on the Developing Child. (2005/2014). Excessive stress disrupts the architecture of the developing brain: Working paper no. 3 (updated edition). Retrieved from www.developingchild.harvard.edu.
- Shonkoff, J.P., Boyce, W.T., & McEwen, B.S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA*, 301(21), 2252–2259.
- Garmezy, N. (1981). Children under stress: Perspectives on antecedents and correlates of vulnerability and resistance to psychopathology. In A.I. Rabin, J. Aronoff, A.M. Barclay, & R.A. Zucker (Eds.), Further Explorations in Personality (pp. 196–269). New York, NY: Wiley.
- Haggerty, R.J., Sherrod, L.R., Garmezy, N., & Rutter, M. (Eds.) (1994). Stress, Risk, and Resilience in Children and Adolescents: Processes, Mechanisms, and Interventions. Cambridge, UK: Cambridge University Press.
- Luthar, S.S., & Brown, P.J. (2007). Maximizing resilience through diverse levels of inquiry: Prevailing paradigms, possibilities, and priorities for the future. *Development* and Psychopathology, 19(3), 931–955.
- Masten, A.S. (2007). Resilience in developing systems: Progress and promise as the fourth wave rises. Development and Psychopathology, 19(3), 921–930.
- 8. Masten, A.S. (2012). Risk and resilience in development. In P.D. Zelazo (Ed.), *The Oxford Handbook of Developmental Psychology, Vol. 2.* New York, NY: Oxford University Press.
- Rutter, M. (2012). Resilience as a dynamic concept. Development and Psychopathology, 24(2), 335–344.
- Werner, E.E., & Smith, R.S. (1992). Overcoming the Odds: High Risk Children from Birth to Adulthood. Ithaca, NY: Cornell University Press.
- Feder, A., Nestler, E.J., & Charney, D.S. (2009).
   Psychobiology and molecular genetics of resilience.
   Nature Reviews Neuroscience, 10(6), 446–457.

- 12. Boyce, W.T., Sokolowski, M.B., & Robinson, G.E. (2012). Toward a new biology of social adversity. *PNAS*, *109*(Supplement 2), 17143–17148.
- Cicchetti, D., & Rogosch, F.A. (2012). Gene x environment interaction and resilience: Effects of child maltreatment and serotonin, corticotropin releasing hormone, dopamine, and oxytocin genes. *Development and Psychopathology*, 24(2), 411–427.
- Kim-Cohen, J., & Gold, A.L. (2009). Measured geneenvironment interactions and mechanisms promoting resilient development. *Current Directions in Psychological Science*, 18(3), 138–142.
- Lyons, D.M., Parker, K.J., Katz, M., & Schatzberg, A.F. (2009). Developmental cascades linking stress inoculation, arousal regulation, and resilience. Frontiers in Behavioral Neuroscience, 3(32), 1–6.
- Macri, S., Granstrem, O., Shumilina, M., Antunes Gomes dos Santos, F.J., Berry, A., Saso, L., & Laviola, G. (2009). Resilience and vulnerability are dose-dependently related to neonatal stressors in mice. *Hormones and Behavior*, 56(4), 391–398.
- Wu, G., Feder, A., Cohen, H., Kim, J., Calderon, S., Charney, D.S., & Mathé, A.A. (2013). Understanding resilience. Frontiers of Behavioral Neuroscience, 7(10), 1–15.
- Russo, S.J., Murrough, J.W., Han, M.H., Charney, D.S., & Nestler, E.J. (2012). Neurobiology of resilience. *Nature Neuroscience*, 15(11), 1475–1484.
- Golden, S.A., Christoffel, D.J., Heshmati, M., Hodes, G.E. Magida, J., Davis, K., ... Russo, S.J. (2013). Epigenetic regulation of RAC1 induces synaptic remodeling in stress disorders and depression. *Nature Medicine*, 19(3), 337–344.
- Mehta, D., Klengel, T., Conneely, K.N., Smith, A.K., Altmann, A., Pace, T.W., ... Binder, E.B. (2013).
   Childhood maltreatment is associated with distinct genomic and epigenetic profiles in posttraumatic stress disorder. *PNAS*, 110(20), 8302–8307.
- Klengel, T., Mehta, D., Anacker, C., Rex-Haffner, M., Pruessner, J.C., Pariante, C.M., ... Binder, E.B. (2012). Allele-specific FKBP5 DNA demethylation mediates gene-childhood trauma interactions. *Nature Neuroscience*, 16(1), 33–41.

- 22. Bradley, R.G., Binder, E.B., Epstein, M.P., Tang, Y., Nair, H.P., Liu, W., ... Ressler, K.J. (2008). Influence of child abuse on adult depression: Moderation by the corticotropin-releasing hormone receptor gene. Archives of General Psychiatry, 65(2), 190-200.
- 23. Caspi, A., Williams, B., Kim-Cohen, J., Craig, I.W., Milne, B.J., Poulton, R., ... Moffitt, T.E. (2007). Moderation of breastfeeding effects on the IQ by genetic variation in fatty acid metabolism. PNAS, 104(47), 18860-18865.
- 24. Rutter, M., Moffitt, T.E., & Caspi, A. (2006). Geneenvironment interplay and psychopathology: Multiple varieties but real effects. Journal of Child Psychology and Psychiatry, 47(3-4), 226-261.
- 25. Caspi, A., Hariri, A.R., Holmes, A., Uher, R., & Moffitt, T.E. (2010). Genetic sensitivity to the environment: The case for the serotonin transporter gene and its implications for studying complex diseases and traits. American Journal of Psychiatry, 167(5), 509-527.
- 26. National Scientific Council on the Developing Child (2010). Early experiences can alter gene expression and affect long-term development: Working paper no. 10. Retrieved from www.developingchild.harvard.edu.
- 27. Bakermans-Kranenburg, M.J., Van Ijzendoom, M.H., Pijlman, F.T., Mesman, J., & Juffer, F. (2008) Experimental evidence for differential susceptibility: Dopamine D4 receptor polymorphism (DRD4 VNTR) moderates intervention effects on toddlers' externalizing behavior in a randomized controlled trial. Developmental Psychology, 44(1), 293-300.
- 28. Kubzansky, L.D., Mendes, W.B., Appleton, A.A., Black, J., & Adler, G.K. (2012). A heartfelt response: Oxytocin effects on response to social stress in men and women. Biological Psychology, 90(1), 1–9.
- 29. Meyer-Lindenberg, A., Domes, G., Kirsch, P., & Heinrichs, M. (2011). Oxytocin and vasopressin in the human brain: Social neuropeptides for translational medicine. Nature Reviews Neuroscience, 12(9), 524-538.
- 30. Schiller, D., Levy, I., Niv, Y., LeDoux, J.E., & Phelps, E.A. (2008). From fear to safety and back: Reversal of fear in the human brain. Journal of Neuroscience, 28(45), 11517-11525.
- 31. Sharot, T., Riccardi, A.M., Raio, C.M., & Phelps, E.A. (2007). Neural mechanisms mediating optimism bias. Nature, 450(7166), 102-105.
- 32. Arnsten, A.F. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. Nature Reviews Neuroscience, 10(6), 410-422.
- 33. Lupien, S.J., McEwan, B.S., Gunnar, M.R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behavior and cognition. Nature Reviews Neuroscience, 10(6), 434-445.
- 34. McEwen, B.S., & Morrison, J.H. (2013). The brain on stress: Vulnerability and plasticity of the prefrontal cortex over the life course. Neuron, 79(1), 16-29.
- 35. Danese, A., & McEwen, B.S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Physiology and Behavior, 106(1), 29-39.
- 36. Martinez, F.D. (2014) The human microbiome: Early life determinant of health outcomes. Annals of the American Thoracic Society, 11(Supplement 1), S7-S12.
- 37. Masten, A.S., Herbers, J.E., Cutuli, J.J., & Lafavor, T.L. (2008). Promoting competence and resilience in the school context. Professional School Counseling, 12(2), 76 - 84.

- 38. Center on the Developing Child at Harvard University. (2011). Building the brain's "air traffic control" system: How early experiences shape the development of executive function: Working paper no. 11. Retrieved from www.developingchild.harvard.edu.
- 39. Ungar, M. (Ed.) (2012). The Social Ecology of Resilience: A Handbook of Theory and Practice. New York, NY: Springer.
- 40. Masten, A.S. (2014). Global perspectives on resilience in children and youth. Child Development, 85(1), 6-20.
- 41. Kasen S., Wickramaratne P., Gameroff M.J., & Weissman M.M. (2012). Religiosity and resilience in persons at high risk for major depression. Psychological Medicine, 42(3), 509-519.
- 42. Daskalakis, N.P., Bagot, R.C., Parker, K.J., Vinkers, C.H., & de Kloet, E.R. (2013). The three-hit concept of vulnerability and resilience: Toward understanding adaptation to early-life adversity outcome. Psychoneuroendocrinology, 38(9), 1858-1873.
- 43. Ellis, B.J., Essex, M.J., & Boyce, W.T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. Development and Psychopathology, 17(2), 303-328.
- 44. Weintraub, S., Dikmen, S.S., Heaton, R.K., Tulsky, D.S., Zelazo, P.D., Bauer, P.J., ... Gershon, R. (2013). Cognition assessment using the NIH Toolbox. Neurology, 80(Supplement 3), S54-S64.
- 45. Babcock, E. (2014). Using brain science to design new pathways out of poverty. Crittenton Women's Union. Retrieved from http://www.liveworkthrive.org/.
- 46. Boyce, W.T., & Ellis, B.J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. Development and Psychopathology, 17(2), 271-301.
- 47. Ellis, B.J., McFadyen-Ketchum, S., Dodge, K.A., Pettit, G.S., & Bates, J.E. (1999). Quality of early family relationships and individual differences in the timing of pubertal maturation in girls: A longitudinal test of an evolutionary model. Journal of Personality and Social Psychology, 77(2), 387-401.
- 48. El-Sheikh, M., Keller, P.S., & Erath, S.A. (2007). Marital conflict and risk for child maladjustment over time: Skin conductance level reactivity as a vulnerability factor. Journal of Abnormal Child Psychology, 35(5), 715-727.
- 49. Obradović, J., Bush, N.R., Stamperdahl, J., Adler, N.E., & Boyce, W.T. (2010). Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socio-emotional behavior and school readiness. Child Development, 81(1), 270-289.
- 50. Shannon, K.E., Beauchaine, T.P., Brenner, S.L., Neuhaus, E., & Gatzke-Kopp, L. (2007). Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. Development and Psychopathology, 19(3), 701-727.
- 51. Boyce, W.T., Chesney, M., Alkon, A., Tschann, J., Adams, S., Chesterman, B., ... Manuck, S.B. (1995). Psychobiologic reactivity to stress and childhood respiratory illnesses: Results of two prospective studies. Psychosomatic Medicine, 57(5), 411-422.
- 52. Guerra, S., & Martinez, F.D. (2008). Asthma genetics: From linear to multifactorial approaches. Annual Review of Medicine, 59, 327-341.
- 53. Obradović, J., Bush, N.R., & Boyce, W.T. (2011). The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors. Development and Psychopathology, 23(1), 101-114.

- 54. Rutter, M., Kim-Cohen, J., & Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. Journal of Child Psychology and Psychiatry, 47(3), 276-295.
- 55. Rutter, M., Moffitt, T.E., & Caspi, A. (2006). Geneenvironment interplay and psychopathology: Multiple varieties but real effects. Journal of Child Psychology and Psychiatry, 47(3-4), 226-261.
- 56. Brody, G., Tianyi, Y., Chen, E., Miller, G., Kogan, S., & Beach, S. (2013). Is resilience only skin deep? Rural African Americans' socioeconomic status-related risk and competence in preadolescence and psychological adjustment and allostatic load at age 19. Psychological Science, 24(7), 1285-1293.
- 57. Rutter, M. (2006). Implications of resilience concepts for scientific understanding. Annals of the New York Academy of Sciences, 1094, 1-12.
- 58. McFarlane, A.C. (1987). Posttraumatic phenomena in a longitudinal study of children following a natural disaster. Journal of the American Academy of Child and Adolescent Psychiatry, 26(5), 764-769.
- 59. Painter, R., Osmond, C., Gluckman, P., Hanson, M., Phillips, D., & Roseboom, T. (2008). Transgenerational effects of prenatal exposure to the Dutch famine on neonatal adiposity and health in later life. BJOG: An International Journal of Obstetrics & Gynaecology, 115(10), 1243-1249.
- 60. Yehuda, R., Halligan, S.L., & Grossman, R. (2001). Childhood trauma and risk for PTSD: Relationship to intergenerational effects of trauma, parental PTSD, and cortisol excretion. Development and Psychopathology, 13(3), 733-753.
- 61. Barel, E., Van Ijzendoom, M.H., Sagi-Schwartz, A., & Bakermans-Kranenburg, M.J. (2010). Surviving the Holocaust: A meta-analysis of the long-term sequelae of a genocide. Psychological Bulletin, 136(5), 677-698.
- 62. Masten, A.S., Hubbard, J.J., Gest, S.D., Tellegen, A., Garmezy, N., & Ramirez, M. (1999). Competence in the context of adversity: Pathways to resilience and maladaptation from childhood to late adolescence. Development and Psychopathology, 11(1), 143-169.
- 63. Coles, R. (1967). Children of Crisis: A Study of Courage and Fear. Boston, MA: Little, Brown and Company.
- 64. Wathen, C.N., Macgregor, J.C., Hammerton, J., Coben, J.H., Herrman, H., Stewart, D.E., & MacMillan, H.L. (2012). Priorities for research in child maltreatment, intimate partner violence and resilience to violence exposures: Results of an international Delphi consensus development process. BMC Public Health, 12(1), 684-665.
- 65. Werner, E.E. (2012). Children and war: Risk, resilience, and recovery. Development and Psychopathology, 24(2), 553-558.
- 66. Cicchetti, D. (2010). Resilience under conditions of extreme stress: A multilevel perspective. World Psychiatry, 9(3), 145-154.

- 67. Davidson, R.J., & McEwen, B.S. (2012). Social influences on neuroplasticity: Stress and interventions to promote well-being. Nature Neuroscience, 15(5), 689-695.
- 68. Erickson, K.I., Voss, M.W., Prakash, R.S., Basak, C., Szabo, A., Chaddock, L., ... Kramer, A.F. (2011). Exercise training increases size of hippocampus and improves memory. PNAS, 108(7), 3017-3022.
- 69. Kaliman, P., Álvarez-López, M.J., Cosín-Tomás, M., Rosenkranz, M.A., Lutz, A., & Davidson, R.J. (2014). Rapid changes in histone deacetylases and inflammatory gene expression in expert meditators. Psychoneuroendocrinology, 40(1), 96–107.
- 70. Ryan, W. (1976). Blaming the Victim (revised edition). New York, NY: Vintage Books.
- 71. Diamond, A. & Lee, K. (2011). Interventions shown to aid executive function development in children 4 to 12 years old. Science, 333(6045), 959-964.
- 72. Henderson, N., & Milstein, M.M. (2003). Resiliency in schools: Making it happen for students and educators. Thousand Oaks, CA: Corwin Press.
- 73. Masten, A.S., & Obradović, J. (2006). Competence and resilience in development. Annals of the New York Academy of Sciences, 1094(1), 13-27.
- 74. Luthar, S.S. (2006). Resilience in development: A synthesis of research across five decades. In D. Cicchetti & D. J. Cohen (Eds.), Developmental Psychopathology: Risk, Disorder, and Adaptation (2nd ed., pp. 739-795). New York, NY: Wiley.
- 75. Cicchetti, D., Rogosch, F.A., & Toth, S.L. (2006). Fostering secure attachment in infants in maltreating families through preventive interventions. Development and Psychopathology, 18(3), 623-649.
- 76. Panter-Brick, C., & Leckman, J. F. (2013). Editorial commentary: Resilience in child development— Interconnected pathways to wellbeing. The Journal of Child Psychology and Psychiatry, 54(4), 333-336.
- 77. Foster, K.A. (2012). In search of regional resilience. In M. Weir, N. Pindus, H. Wial, & H. Wolman (Eds.) Urban and Regional Policy and Its Effects: Building Resilient Regions, Volume 4 (pp. 24-59). Washington, D.C.: Brookings Institution Press.
- 78. Holling, C.S., & Gunderson, L.H. (2002). Resilience and adaptive cycles. In L.H. Gunderson and C.S. Holling (Eds.), Panarchy: Understanding Transformations in Humans and Natural Systems (pp. 25-62). Washington, D.C.: Island Press.
- 79. American Psychological Association. (2014). The Road to Resilience. Retrieved from http://www.apa.org/helpcenter/road-resilience.aspx.
- 80. McEwen, B. (1998). Protective and Damaging Effects of Stress Mediators. Seminars in Medicine of the Beth Israel Deaconess Medical Center, 338(3), 171-179.

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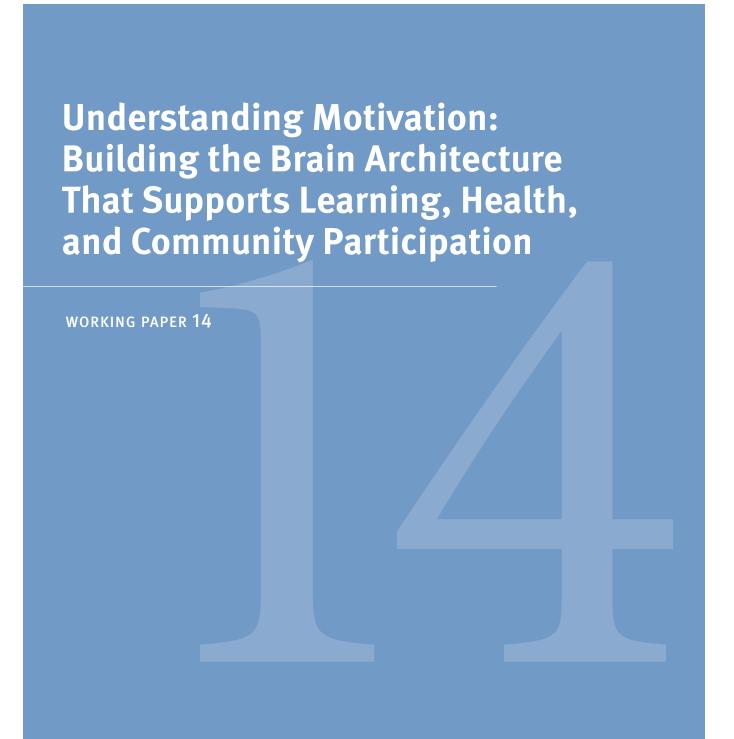
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## The Issue

A HEALTHY, ENGAGED COMMUNITY DEPENDS ON PEOPLE ACHIEVING TO THE BEST OF THEIR potential, contributing actively to the economy and public well-being, and helping the next generation to thrive. A complex set of intertwined social and biological factors influences people's motivation to participate actively and productively in schools, jobs, and communities and to persevere in the face of setbacks. To unlock this puzzle and ensure that all people have the opportunity to develop motivation to learn, improve skills, and make healthy choices, it would be helpful to understand the underlying mechanisms in the brain that develop in childhood and build the foundation for later complex behavior (see page 7 for more information).

The brain circuits underlying motivation are critical for attention, learning, and decision-making. When these circuits have either not developed in a balanced and healthy way or have been chemically hijacked by addictions, challenging life circumstances can overpower the best of intentions. Programs intended to support parents and children facing adversity often find that participation is one of their greatest challenges. Dropping out of school and not participating in family support, job training, or addiction programs—all of these are reflections of motivation systems that have been disrupted by threat or hardship. Substantial scientific knowledge can inform the search for solutions

by helping us understand what leads to these behaviors.

The brain systems that govern motivation are built over time, starting in the earliest years of development. These intricate neural circuits and structures are shaped by interactions between the experiences we have and the genes we are born with, which together influence both how our motivation systems develop and how they function later in life. Providing children with the kinds of early life experiences that support the development of healthy, balanced motivation systems is key to ensuring positive outcomes laterfor school, work, health, and raising the next generation.

## The Science of Motivation

In the brain, motivation is the result of neurons (brain cells) in specific regions sending chemical signals via high-speed neural networks to other regions, creating pathways for future signals to follow. Experiences trigger the release of these chemicals to regions that connect emotions, memory, and the sensation of pleasure or reward. This links the feeling of reward to the emotions we felt and the experience that led to it—and that influences both our expectations of reward and the actions we are motivated to take in order to get it. Given the appeal of anticipating an immediate reward, it takes strong self-regulation to resist these powerful memories and cues in favor of a long-term reward.

The chemicals, which include dopamine, serotonin, norepinephrine, glutamate,1-2 and naturally occurring opioids that are produced in the brain, each serve different purposes. For example, when dopamine is released, it signals to the Memories rest of the brain that something important is about happen—something that we should enjoy **Experiences** or avoid at all costs. Rewards (See page 7 for more Actions information on the brain chemicals and regions involved in motivation.) **Emotions** 

There are two types of motivation: one directed toward expected rewards (known as approach motivation) and another directed away from threat (known as avoidance motivation). In other words, we can be motivated either to seek pleasure or to avoid danger. Both kinds of motivation are necessary for survival, and supportive developmental processes create a healthy balance between the two. When they are out of reward-seeking balance, excessive danger-avoidance can lead to a range of disorders, including attention-deficit/hyperactivity, depression, substance abuse,

Intrinsic

Preferences

Substance

Dependence

Motivation

Life Circumstances

Drivers

anxiety, and post-traumatic stress.3-6 Both types of develop motivation in childhood and are strongly influenced by what's happening in a child's environment.7 For example, the consistent presence of a supportive adult in a child's life can calm an overactivat-Stressful ed amygdala, an area of the brain that is critical for learning fear and responding to threat. The result is a bal-

anced system that assesses and responds to real threats appropriately. On the other hand, children who are raised in abusive, chaotic, or scary environments without supportive adults tend to be *more* likely to perceive experiences as threatening—and respond to them as threats—but *less* likely to expect rewards when they do something positive.<sup>8</sup> Here, the systems become overly attuned to impulsive self-protection and less to long-term goal achievement.

 Approach motivation is key to most forms of learning. Anticipating a reward—which can be any experience that causes pleasure, from the taste of a delicious food to the satisfaction of achieving a goal or the glow of an act of kindness—triggers a dopamine surge. That surge is a signal to expect new experiences that are worth seeking out and learning from. The surge also increases communication between the brain region responsible for dopamine release and the regions responsible for emotion and memory. As a result, our memories of the learning experience become linked with the reward received and the emotions we felt. Strong emotional connections to memories help us retrieve them more readily. The decisions made in order to receive a short-term reward may not always achieve a long-term

benefit, as anyone tempted by a dessert underdelicious stands, but the brain is attuned to experiences that have previously led to a reward, and it learns to pre-Extrinsic dict which ex-Feedback periences likely to trigger the reward again.

Avoidance
 m o t i v a t i o n
directs us away from

threatening or unpleasant experiences. Avoidance motivation-which we associate with the emotions of fear or disgust—often involves activation of the amygdala.<sup>12</sup> When the amygdala is activated in response to threat, norepinephrine and other stress hormones are released, triggering what is often called the "fight or flight" response: increases in heart rate, blood glucose levels, and oxygen intake to the brain, and a temporary shutdown of less mission-critical functions, such as the digestive or metabolic systems. This response may be triggered by an instinctual detection and response to threat or by the conscious awareness of a potential threat (physical or emotional). Disgust protects us from ingesting or touching repulsive and often unhealthy substances. Fear and disgust—both the result of avoidance motivation—develop in the brain

Responsive

Social

Supports

even before language: Babies feel afraid well before they can say that the feeling is fear, and are able to express disgust as early as the first day of life.13 Avoidance motivation can also be learned through experience—when the brain correctly or incorrectly predicts how serious a particular threat may be, it learns whether (and how much) to avoid it in the future. But while important for survival, the avoidance response can actually inhibit higher-level learning by focusing the brain's activity on immediate response rather than planning to attain a long-range goal or resisting an impulsive behavior.

Both approach and avoidance motivation are influenced by intrinsic (internal) drivers and extrinsic (external) feedback.

While all organisms have the intrinsic drive to survive, approach reward, and avoid threat, we may also be motivated by the inherent pleasure and satisfaction derived from an activity. Although neuroscientific understanding of intrinsic motivation is still quite recent, it is believed that intrinsically motivating experiences trigger a dopamine surge, signaling the anticipation of pleasure.14 Once their basic needs are met, young children are motivated intrinsically by exploration, active involvement in play, and achieving mastery or success in a task, whether banging a spoon to make a noise or solving a problem. This kind of motivation is important for learning and development because it leads to intense engagement in a task and mastery is associated with pride and satisfaction.15

Intrinsic drivers are considered to be the strongest and most lasting motivators, especially in early childhood, but positive feedback can support and reinforce the inherent feelings of satisfaction or pleasure. For example, satisfaction from mastery is supported by positive feedback from an authority figure. But while positive feedback is important in boosting a child's selfconfidence, in some cases, external rewards have been shown to undermine intrinsic drivers. That is, children are less likely to engage spontaneously in activities after they

have received a tangible reward for having performed them.<sup>16</sup> When traditional school systems rely heavily on extrinsic feedback, such as grades and awards, this can lead to a shift from the intrinsic drive to learn to a desire for external recognition and accolades or avoidance of failure or punishment. While extrinsic motivation may be effective for some in the short term, it is unlikely to last.

Once their basic needs are met, young children are motivated intrinsically by exploration, active involvement in play, and achieving mastery or success in a task.

The combination of intrinsic drivers supported by positive extrinsic feedback is best for building a healthy motivation system, but extrinsic feedback by itself is not an effective driver of behavior over the long term.<sup>17</sup>

In approach motivation, there is a "wanting" system and a "liking" system, which can separate the desire to have an experience from the reward that is actually experienced. Most people are not directly aware of the underlying processes of wanting or liking—that is, it is possible to want something at a deep, physiological level without being conscious of the pleasure it elicits.

The intense desire for experiencing pleasure, or "wanting," is generated in the brain by the dopamine network, which connects the regions of the brain that trigger automatic, "non-thinking" responses to those that manage memories, emotions, and behaviors.18 The circuits that connect the reward, the action that led to it, and the emotions felt at the time are so strong that even when the reward is withheld or diminished, the brain will still prompt us to repeat the action that initially led to the reward. That can produce "wanting" (the desire for the experience) without "liking" (the actual pleasure or reward that is felt). This separation explains why some people engage in once-rewarding behaviors past the point where they are enjoyable, like eating too much dessert or drinking too much alcohol.

The "liking" system, on the other hand, is highly localized within a small region of the brain. When naturally occurring opioids, endocannabinoids, and serotonin are transmitted, they must be received by this region to activate the actual feeling of pleasure rather than the craving for it. The liking system, therefore, consists of narrower, more fragile circuits than the wanting system, and thus is less easily activated. 19 The fragililty of these circuits may be one reason why intense pleasure is harder to experience than intense desire.<sup>7</sup> For example, the smell of a candy store may trigger "wanting" based on past memories linking sweets to pleasure. But the actual experience of eating the candyor of eating too much—may not be nearly as pleasurable as the memory.

Typically, the pleasure received during *liking* triggers *wanting*, but people dealing with addiction often *want* substances or experiences intensely even when pleasure is no longer obtained from them. That's because repeated exposure to many addictive drugs causes the *wanting* systems to activate more easily and strongly. At the same time, overstimulation of the *liking* system (through, for example, artificial opioids and opiates) can lead to the need for increasing amounts of the drug to achieve the same effect. Therefore, *wanting* the drug increases even while the *liking* for a given dose may fade. <sup>20-21</sup> (See page 7 for more information.)

Wanting System	Liking System
Widespread, robust network across multiple regions of the brain	Highly localized within a small region of the brain
Activated through connections involving the reward, the action that led to it, and the emotions felt at the time	Activated when dopamine, serotonin, and naturally occurring opioids are received in this specific brain region
Even when a reward is diminished or absent, the brain will still prompt "wanting"	The dopamine system triggers less neural activity over time, leading to reduced pleasure from the same experiences

## **How Motivation Systems Develop**

THE BRAIN'S MOTIVATION SYSTEMS ARE particularly sensitive during certain periods of early childhood development, when infants and young children are learning approach, avoidance, and attachment behaviors. While genes provide the basic blueprint for the motivation circuits, disruptions in developmentally appropriate experiences during these sensitive periods can affect how the circuits develop and the behaviors they shape. For example, a child can become more highly attuned to avoidance than approach, which would reduce motivation to try new activities. Because the timing of the development of motivation systems is so important, different kinds of experiences may have different impacts at different stages—the influence of peers, for example,

is much stronger in adolescence than in early childhood.

Especially in the early years of life, adults caring for a child strongly influence the development of the brain and motivation system. Infants learn best through interactions with parents and other important adult caregivers who establish responsive, supportive relationships with them. The brain is biologically prepared to form strong, lasting emotional connections to these caregivers, a bond known as attachment.

Research shows that there are different sensitive periods in which attachment figures affect motivation early in life. In the earliest period, babies learn simple preferences among pleasant and unpleasant experiences. Then they begin to distinguish between threats that truly need to be avoided and those that are less dangerous-here, the supportive presence of a trusted adult can have a buffering effect.<sup>22</sup> For example, a loud noise may startle a child, but if children are in the presence of someone they are attached to and feel secure with, and who responds supportively, they will be less likely to experience high levels of stress. Without these relationships—or in situations where these primary relationships are themselves a frequent source of fear-an imbalance may develop between approach and avoidance responses.

## During adolescence, the motivation system is increasingly influenced by peers, exploration, and performance feedback.

Adolescence represents a period of adjustment to increased independence; it is also a time of social reorientation from the influence of parents to peers. During this time, relationships with peers become more complex and intimate. They require greater social understanding and become more rewarding. Adolescence is also a time of neural and behavioral flexibility and change. Because different regions of the brain mature at different rates, during adolescence, the neural circuits involved in cognitive, emotional, and social information processing are at different stages of development and reorganization, and have not yet achieved their adult balance. Typically, adolescents show stronger neural responses to social acceptance and rejection than adults. This may explain why youth seem especially sensitive to negative social feedback, and why positive social feedback and acceptance are so rewarding.

Social interaction induces an increase in natural opioids and activates the region of the brain responsible for releasing dopamine and serotonin.23-24 The pleasure of social acceptance is present early in life and remains important in adulthood, but is especially powerful in adolescence when the brain is particularly tuned into these rewards. The anterior cingulate cortex (ACC), a portion of the brain that monitors and appraises social acceptance and exclusion, plays a key role in reward-based decision-making and learning.

## Because the timing of the development of motivation systems is so important, different kinds of experiences may have different impacts at different stages.

Researchers have found that children who were chronically rejected, or had been emotionally abused or neglected, had a more active ACC and showed heightened sensitivity to the neurotransmitters released as a result of social exclusion.25 So, while all adolescents are strongly motivated by social feedback, those who have experienced emotional abuse or rejection are even more motivated bv it.26

The cognitive and emotional development and increased cognitive flexibility that occurs during adolescence creates both opportunities and challenges. The increased sensitivity to social rewards can lead to an inclination toward risk-taking and self-oriented acts, but also powers exploratory learning and the ability to adapt to different social contexts and cultures. Adolescents learn both by personal exploration and external feedback on their performance. For example, positive feedback can increase motivation by signaling to an adolescent that a goal is of high value and attainable. If the youth has low commitment to the goal, positive feedback can reinforce the feeling that he or she is on the right track. When someone is strongly committed to the goal already, critical feedback may be effective because it points out the discrepancy between where the youth currently is and where he or she wants to be.27 For example, an athlete who is intrinsically driven to participate in a particular sport may be motivated to improve on weaknesses by a coach's constructive criticism, but a student who is less drawn to the sport may be motivated more by encouragement—and might disengage as a result of criticism.

## **How Motivation and Reward Systems Can Be Disrupted**

Excessive stress and a lack of positive relationships can derail the development of well-balanced motivation systems. Children who experience a safe, supportive, predictable environment healthy motivation systems that are driven by a balance of approach and avoidance, and of wanting and liking. Children whose environment is chaotic and stressful, however, may develop motivation systems that are driven by avoidance and focused on fear. Environmental factors can actually change the mapping of the nucleus accumbens, a key region of the brain that receives dopamine. In animal studies, stressful environments resulted in this region expanding its feargenerating zone, while shrinking the zone that generates desire. Conversely, calm, quiet environments expanded desire-generation and reduced fear-generation.28

# Children whose environment is chaotic and stressful may develop motivation systems that are driven by avoidance and focused on fear.

Research has also shown that, when the stress response is activated repeatedly, the brain adapts by identifying threats more frequently and reacting to them more strongly—even if the sources of stress do not increase in severity. Over time, that increasingly stronger stress reaction may reshape circuits in the hippocampus, which controls memory, or the amygdala, which is involved in emotional reactions.

While a responsive, consistent relationship with an adult can suppress the release of stress hormones in an infant's brain, the reverse is also true. Studies find that children can over-learn fear from the adults around them, which affects the amygdala and may have long-term consequences for a child's health, learning, and social relationships.<sup>23</sup> Expressing fear is not always a bad thing: for example, an urgent warning from a mother when her child is moving a finger toward an

electrical socket will help the child learn to avoid the socket. But excessive or misdirected fear by a primary caregiver can lead a child to lose interest in healthy exploration when the motivation to avoid threat overpowers the motivation to approach new experiences.

## Feeling helpless and believing that abilities are fixed and unchangeable can disrupt the brain's motivational systems.

The motivation to act also requires some expectation of success.<sup>29</sup> Indeed, successfully making things happen is rewarding in and of itself. We can observe this even in very young infants. If a ribbon is tied to an infant's leg and the other end attached to a mobile, when the baby notices that when she kicks the mobile moves, she will kick more vigorously.30 She may also smile and gurgle until the ribbon is untied from the mobile, and then kick vigorously and cry angrily.31 When a child does not see any effects from her actions, it produces what is termed "learned helplessness." People who learn that they are helpless often give up even in situations in which they can be successful.<sup>32</sup>

Researchers are also finding that how children and adults think about skills and talents makes a big difference in whether they are motivated to keep trying. If skills and talents are thought of as fixed-something people either have or don't have—an initial failure is likely to be attributed to a lack of natural ability and, in turn, it may decrease motivation. This is called a fixed mindset. On the other hand, if skills and talents are seen as capacities that can be developed through practice—a growth mindset—then a failure signals the need to develop the skill or talent through continued effort and practice. Evidence shows that mindset interventions with adolescents can improve academic performance, particularly among low-achieving teens.33-34

Notably, adults sometimes inadvertently lead children to develop a fixed mindset when we praise them for being smart, or for their talent in a particular domain, as if the ability

## Key Brain Regions and Chemicals for Motivation and Reward

## **Regions**

## **Amygdala**

"Emotion Trigger"—rapidly assesses incoming information from the environment and activates either approach or avoidance behaviors. This structure is critical for threat detection and learned fear.

## **Hippocampus**

"Memory Center"—lays down detailed memories of events and triggers retrieval of these memories when presented with a relevant cue. This structure also is involved in regulating the duration of stress responses to environmental stimuli.

## **Anterior Cingulate Cortex (ACC)**

"Behavior Tracker"—monitors the environment as well as one's own behavior and others' (such as social exclusion). This region sounds the alarm when behavior needs to be modified, mobilizing regions in the prefrontal cortex involved in self-regulation and decision-making.

## **Prefrontal Cortex**

"Air Traffic Control"—manages executive functions, selfregulation, behavioral control, planning, and complex decision-making.

## **Nucleus Accumbens**

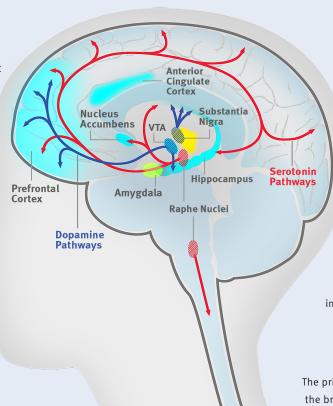
"Reward Anticipator"—evaluates stimuli that produce wanting or liking responses. This structure also plays an important role in learning from feedback and in reward-based decision-making.

## Substantia Nigra/Ventral Tegmental Area (VTA)

"Dopamine Distributors"—produce the brain chemical dopamine and deliver it to other regions of the brain that are involved in motor function and in motivating and rewarding behaviors.

## Raphe Nuclei

"Serotonin Distributors"—produce the neurotransmitter serotonin and deliver it to a wide network of circuits across the brain, including structures related to motivation, reward, and threat detection.



## Chemicals

## Dopamine

A key factor in "wanting," dopamine modulates neural activity when a rewarding event has occurred. Increases in dopamine reinforce the behaviors that elicited the reward and lead individuals to seek out and learn from new experiences in anticipation of a positive outcome.

## Serotonin

A key factor in "liking," serotonin combines with other neurochemicals to convey euphoria and has the widest distribution in the brain. Serotonin modulates a wide array of behaviors, including a major influence on emotional states, sleep cycles, eating, and other rewarding behaviors.

## Glutamate

The primary, fast chemical neurotransmitter in the brain that excites and communicates with neurons across synapses.

## Norepinephrine

Triggers "fight or flight" response, which increases heart rate, glucose, and oxygen intake to the brain, and temporarily shuts down less mission-critical functions. Norepinephrine works in part by activating attention systems to the most important stimuli in the environment at that moment.

## **Endocannabinoids**

Regulate neuronal activity caused by dopamine and related behaviors and work with opioids and serotonin to produce euphoria.

## **Opioids**

A class of naturally occurring chemicals, such as endorphins, that reduce pain and can produce euphoria. Opioids are released by the body during exercise and pleasurable activities. Use of artificial opioids, such as heroin and a number of prescription painkillers, can desensitize the reward circuitry of the brain and result in addiction that increases drug-seeking.

Illustration by Betsy Hayes

is something that they possess naturally, and not something they can develop over time. Modeling is also important and can influence a sense of self-efficacy. A study in which infants witnessed one adult working hard to achieve a specific goal and another succeeding at a goal effortlessly found that infants persisted at a novel task more after observing the high-effort example. 35-36

# Instrinsic motivation can either be encouraged or suppressed by the experiences adults provide for children.

Thus, even babies are sensitive to what they witness regarding the relationship between effort and outcome.

Addictive drugs and behaviors can hijack the brain's motivation and reward systems. The overwhelming compulsion to seek and take drugs or alcohol, or engage in a range of pleasure-stimulating activities such as gambling or casual sex, involves both positive and negative reinforcement. By flooding the nucleus accumbens with dopamine, these activities provide a shortcut to pleasure, bypassing the time and effort required to trigger similiar positive feelings of reward that are generated by achieving a goal or mastering a skill. Other parts of the brain create memories of this rapid

experience of pleasure and connect them to the wanting and liking systems. By contrast, these activities may also temporarily suppress the intensity of negative emotions, leading to their use as self-medication. Stated simply, these experiences can quickly ramp up good feelings *and* tamp down bad feelings—but only temporarily. This can create a spiral of dysregulation, in which the addictive experience triggers initial pleasure, followed by negative emotions and physical craving, which can only be suppressed by the addictive behavior.<sup>37</sup>

Addictive drugs can often release more dopamine than natural rewards. This overstimulation of cells that receive dopamine can, over time, change the neural pathways and chemistry in the motivation systems. As a result, the brain becomes less and less affected by dopamine, at least as long as the drug continues to be taken.<sup>38</sup> However, the wanting systems can become permanently hyper-reactive to drug cues, even after ending drug use. In other words, the repeated use of a short-cut to pleasure creates circuitry that is so strongly associated with memories of pleasure that the wanting system produces powerful urges to follow it, even when the pleasure itself fades. Ultimately, the mere memory of the behavior-and even the associations of people or places with the behavior—can lead to the impulsive actions that characterize addiction.<sup>39</sup>

## Implications for Parents, Caregivers, and Teachers

THE INTRINSIC MOTIVATION TO LEARN ABOUT the world around us begins in infancy. This type of motivation can either be encouraged or suppressed by the experiences adults provide for children. Psychological research points to a set of promising approaches that parents and practitioners can use to promote positive motivation and learning during development.

 Follow babies' lead. Babies naturally orient toward novel objects and events. They look away from objects that are overly familiar, but also from new ones that are too complex. This is sometimes called the "Goldilocks effect:" things are interesting when they are novel, but not too novel.<sup>40</sup> When interacting with infants, notice what they pay attention to, and engage with them around their interests.

• Elicit curiosity. Infants seek to explore objects—especially those that behave in surprising ways. When they drop something on the floor or throw it, they're trying to see what will happen next.<sup>41</sup> Provide infants with opportunities to interact with

- new objects-and let them lead and learn!
- Encourage children's playful explo**ration**. When given the opportunity, children of all ages spontaneously engage in play. The ingredients of play are precisely the ones that fuel learning: play is intrinsically motivating, it presents an opportunity for novel experiences and for learning from others, it requires active engagement, and it can strengthen social bonds and reduce stress. When life is busy or chaotic, it can be hard to find the time and space to encourage children's play, but this is an important aspect of development.42
- Prioritize social interaction during **learning**. In the digital age, there are many educational, computer-based applications designed for children, even as young as 6 months.<sup>43-44</sup> However, even the best-designed and most effective apps cannot replace real-life social interactions with adults and peers. In one study, babies learned elements of language more effectively when face-to-face with a teacher or caregiver than when watching her on video.45 Recent research shows that young children can learn from digital media, such as touch-screen tablets, but social interaction during this learning experience appears to be essential.46

## Five Facts About Motivation That Are Often Misunderstood

- Motivation comes from a set of neurochemical networks that develop over time, as a result of the experiences we have. Despite the common misperception that some people just naturally have or lack motivation, science shows that the nature of caregiving relationships and opportunities for safe exploration that we provide young children affect the development of these systems—for better or for worse.
- The best way to sustain motivation is to support internal drivers with the right kind of external feedback. Carrots (rewards) and sticks (punishments) are not the only ways to motivate people. Systems focused solely on external rewards and punishments are unlikely to achieve sustained, productive motivation; those that balance intrinsically motivating activities—such as creative problem-solving and playful learning-with positive feedback are more likely to support healthy motivation over the long run.
- Addictions divert motivation systems and require more than willpower to overcome. Addictions chemically hijack the basic biological systems that have evolved for optimal survival. Addiction does not reflect a simple lack of conscious effort or a "failure of character;" managing addictions requires blocking these chemical diversions below the conscious level.
  - Motivation is complicated and has many influences. Behavior is affected by the experiences and conditions that shape a mindset that goal achievement is possible—and, critically, by having the resources, time, skills, and supports that make successful action feasible. It is incorrect to say that if anyone wants something badly enough, he or she will find a way to do it.
- Providing a predictable reward is not enough to sustain motivation over time. Experiences that are exactly as expected every time lose their novelty, and eventually elicit less neural activity in the dopamine system. When we experience a reward that is better than predicted, the brain will prefer that experience in the future; if the reward is less than expected, the brain will ultimately exhibit less motivation toward that experience. So "keeping things fresh," whether through new activities, different locations, or a different reward, is good advice for sustaining motivation.<sup>14</sup>

- Challenge children just enough. Kids are motivated to work toward achievable goals. From infancy onward, effort is required to sustain motivation, but success must be possible. They lose motivation when a task is too easy, but also when it is so difficult as to be insurmountable. Video games harness this basic principle of learning effectively, constantly increasing the level of challenge based on an individual child's performance. Try to adapt a challenge according to a child's current capabilities, and provide prompt feedback on his or her performance.
- **Give children agency**. Children are more motivated when they have some degree of self-determination, and can elect to pursue tasks that are personally meaningful. When they have a choice of projects, or at least a little wiggle room as to how a task gets done, children are more likely to stay engaged.<sup>15</sup>
- Provide incentives only when necessary. When children are suddenly rewarded for something they enjoy and do freely, they may begin to do it only when they know they will be compensated afterwards.<sup>47</sup> Wherever possible, harness children's natural curiosity and inclination to work toward an achievable goal, rather than promising a reward.
- Praise the process rather than the outcome.<sup>47</sup> When we praise children for their intellect or skill level—or the grade or gold medal they received—it can lead to a performance orientation. They may be motivated to achieve

- more rewards, but they may also learn to shy away from challenging activities that they might not excel at, for fear of negative evaluation. Performance pressure increases as children move up in school, and it is associated with depression and anxiety in addition to diminished joy of learning. When we praise children for their effort and help them see falling short as an opportunity to learn and improve (rather than simply focus on the outcome), they will be more motivated to work hard and more likely to believe that they can achieve what they put their mind to.
- Maintain a close connection with **adolescents.** Adolescence is a period when many young people take risks and push boundaries. This trend reflects, in large part, a natural inclination toward novel and exciting experiences that maximize learning opportunities and are important in making the transition to independence. As teens become more motivated by the approval of their peers, it can be socially rewarding to follow risk-taking leaders or stand out by breaking boundaries. However, teens with close family relationships are less prone to risk-taking.48 High parental support and open dialogue are associated with fewer problem behaviors, including less substance abuse and delinquency. Be empathetic and supportive, knowing that youth are going through changes in their brains, bodies, and social relations that can make risky behavior appealing to them. Keep the lines of communication open—and keep close tabs on teens.

## Implications for Policy and Public Systems

- Support the development of motivation in early childhood programs. Knowing that the brain systems underlying motivation begin to develop in infancy, we can help children develop the balanced systems they will need later in life by starting in the earliest years. High teacher-to-child ratios, training in effective strategies to facilitate playful exploration and build selfefficacy, reducing stress in families' lives, and skill-building for parents and providers of early care and education are all contributors to ensuring that the foundations of healthy motivation systems are built in early childhood.
- Shift schools toward a balance of positive feedback that supports intrinsic drivers. To improve student motivation, school systems should reduce emphasis on extrinsic rewards (like grades, tests, and performancebased recognition programs) and increase emphasis on constructive feedback and coaching to improve performance. Support and reward exploration, praise effort, and use successes in one area to inspire effort in another, while avoiding punishmentbased approaches.
- Focus response to addiction on treatment rather than punishment. Policies and programs relating to addictions can be improved by understanding that addictive drugs rewire and redirect motivation and reward systems. Knowing that craving or wanting addictive substances happens below the conscious level means that solutions must also occur on the physiological or biological level, rather

- than expecting awareness of potential punishments to change behavior.
- Include motivation-building supports in programs for adults who care for young children. Foster a growth mindset by praising effort, looking at mistakes as learning opportunities, and monitoring progress toward goals. Incorporate and test promising methods for goal-setting and keeping people on track to achieve self-defined goals. These approaches can not only improve retention in programs, but can also enable parents and caregivers to model and support these skills and mindsets in children.
- Replace punitive approaches to program retention with methods that reduce stress, provide positive feedback and social/peer support, and demonstrate quick successes. Many programs designed to help families with young children struggle to engage parents to participate actively and stay with the program long enough to truly benefit. Many factors contribute to program engagement and retention, ranging from inconvenience of timing, location, and transportation, to cost, child care needs, and competition with other activities or the needs of other family members and friends. Along with addressing those factors, programs can increase motivation to participate by making it easier to rejoin after a lapse (reducing stress), helping participants achieve small successes quickly (building self-efficacy), and making participation more rewarding by praising effort (strengthening a growth mindset).

## References

- Powledge, T.M. (1999). Addiction and the brain: The dopamine pathway is helping researchers find their way through the addiction maze. BioScience, 49(7), 513-19.
- Tzschentke, T.M. & Schmidt, W.J. (2003). Glutamatergic mechanisms in addiction. *Molecular Psychiatry*, 8(4), 73-82.
- Meyer, B., Johnson, S.L., & Carver, C.S. (1999). Exploring behavioral activation and inhibition sensitivities among college students at risk for bipolar spectrum symptomatology. *Journal of Psychopathology and Behavioral* Assessment, 21(4), 275-92.
- Muris, P., Merckelbach, H., Schmidt, H., & et al. (2001).
   Anxiety and depression as correlates of self-reported behavioural inhibition in normal adolescents. *Behaviour Research Therapy*, 39(9), 1051-61.
- Kasch, K.L., Rottenberg, J., Arnow, B.A., & et al. (2002). Behavioral activation and inhibition systems and the severity and course of depression. *Journal of Abnormal Psychology*, 111(4), 589-97.
- Nelson-Gray, R.O., Keane, S.P., Hurst, R.M., & et al. (2006).
   A modified DBT skills training program for oppositional defiant adolescents: Promising preliminary findings.
   Behaviour Research and Therapy, 44(12), 1811-20.
- Berridge, K.C. & Kringelbach, M.L. (2013). Neuroscience of affect: Brain mechanisms of pleasure and displeasure. Current Opinion in Neurobiology, 23(3), 294-303.
- Berridge, K.C. & O'Doherty, J.P. (2014). From experienced utility to decision utility. In Glimcher P.W., & Fehr, E. (Eds.) Neuroeconomics: Decision making and the brain. (San Diego: Academic Press), 335-51.
- Adcock, R.A., Thangavel, A., Whitfield-Gabrieli, S., & et al. (2006). Reward-motivated learning: Mesolimbic activation precedes memory formation. *Neuron*, 50(3), 507-17.
- Murty, V.P., Ballard I.C., Macduffie K.E., & et al. (2013).
   Hippocampal networks habituate as novelty accumulates.
   Learning & Memory, 20(4), 229-35.
- Murty, V.P., Labar, K.S., & Adcock, R.A. (2012). Threat of punishment motivates memory encoding via amygdala, not midbrain, interactions with the medial temporal lobe. *Journal of Neuroscience*, 32(26), 8969-76.
- Shabel, S.J., Schairer, W., Donahue R.J., & et al. (2011).
   Similar neural activity during fear and disgust in the rat basolateral amygdala. PLoS One, 6(2), 317-25.
- Graillon, A., Barr, R.G., Young, S.N., & et al. (1997).
   Differential response to intraoral sucrose, quinine and corn oil in crying human newborns. *Physiol Behav.*, 62(2), 317-25.
- Di Domenico, S.I. & Ryan, R.M. (2017). The emerging neuroscience of intrinsic motivation: A new frontier in selfdetermination research. Frontiers in Human Neuroscience, 11, 145.
- Stipek, D. (2011). Classroom practices and children's motivation to learn. In Ziegler, E., Gilliam, W.S., & Barnett, W.S. (Eds.) The pre-K debates: Current controversies and issues. (Baltimore, MD: Brookes Publishing), 98-103.
- Deci, E.L., Koestner, R., & Ryan, R.M. (1999). A metaanalytic review of experiments examining the effects of extrinsic rewards on intrinsic motivation. *Psychological Bulletin*, 125(6), 627-68.
- Stipek, D. (2011). Education is not a race. Science, 332(6037), 1481.

- Berridge, K.C., Robinson, T.E., & Aldridge, J.W. (2009).
   Dissecting components of reward: 'Liking,' 'wanting,' and learning. Current Opinion in Pharmacology, 9(1), 65-73.
- Peciña, S. (2008). Opioid reward 'liking' and 'wanting' in the nucleus accumbens. Physiology & Behavior, 94(5), 675-80.
- Berridge, K.C. & Robinson T.E. (1995). The mind of an addicted brain: Neural sensitization of wanting verus liking. Current Directions in Psychological Science, 4(3), 71-5.
- Galanter, M., Kleber, H.D., & Brady, K.T. (Eds.). (2015). Neurobiology of Opiates and Opioids. In *The American Psychiatric Publishing Textbook of Substance Abuse Treatment, Fifth Edition*. (Arlington, VA: American Psychiatric Publishing), 277-95.
- Moriceau, S. & Sullivan, R.M. (2006). Maternal presence serves as a switch between learning fear and attraction in infancy. *Nature Neuroscience*, 9(8), 1004-6.
- Lahvis, G.P. (2016). Social reward and empathy as proximal contributions to altruism: The camaraderie effect. In Wöhr M., Krach S. (Eds.) Social Behavior from Rodents to Humans. Neural Foundations and Clinical Implications. (Cham, Switzerland: Springer International Publishing Group), 127-57.
- Krach, S., Paulus, F.M., Bodden, M., & et al. (2010). The rewarding nature of social interactions. Frontiers in Behavioral Neuroscience, 4, 22.
- van Harmelen, A.L., Hauber, K., Gunther Moor, B., & et al. (2014). Childhood emotional maltreatment severity is associated with dorsal medial prefrontal cortex responsitivity to social exclusion in young adults. PLoS One, 9(1), e85107.
- Crone, E.A. & Dahl, R.E. (2012). Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nature Reviews Neuroscience*, 13(9), 636-50.
- Fishbach, A., Eyal, T., & Finkelstein S.R. (2010). How positive and negative feedback motivate goal pursuit. Social and Personality Psychology Compass, 4(8), 517-30.
- Braver, T.S., Krug, M.K., Chiew K.S., & et al. (2014). Mechanisms of motivation-cognition interaction: Challenges and opportunities. Cognitive, Affective, & Behavioral Neuroscience, 14(2), 443-72.
- Duckworth, A. & Gross, J.J. (2014). Self-control and grit: Related but separable determinants of success. Current Directions in Psychological Science, 23(5), 319-25.
- Rovee-Collier, C.K. & Gekoski, M.J. (1979). The economics of infancy: A review of conjugate reinforcement. Advances in Child Development and Behavior, 13, 195-255.
- Lewis, M. & Ramsay, D. (2005). Infant emotional and cortisol responses to goal blockage. *Child Development*, 76(2), 518-30.
- Seligman, M.E. (1972). Learned helplessness. Annual Review of Medicine, 23, 407-12.
- Gunderson, E.A., Gripshover, S.J., Romero, C., & et al. (2013). Parent praise to 1- to 3-year-olds predicts children's motivational frameworks 5 years later. *Child Development*, 84(5), 1526-41.
- Mueller, C.M. & Dweck, C.S. (1998) Praise for intelligence can undermind children's motivation and performance. *Journal of Personality and Social Psychology*, 75(1), 33-52.
- Leonard, J.A., Lee, Y., & Schulz, L.E. (2017). Infants make more attempts to achieve a goal when they see adults persist. *Science*, 357(6357), 1290-4.
- 36. Cook, D.A. & Artino, A.R. (2016). Motivation to learn: An

- overview of contemporary theroies. *Medical Education*, 50(10), 997-1014.
- Edwards, S. & Koob, G.F. (2010). Neurobiology of dysregulated motivational systems in drug addiction. *Future Neurobiology*, 5(3), 393-401.
- Harvard Health Publishing (2011). How addiction hijacks the brain. Harvard Mental Health Letter. Retrieved from: https://www.health.harvard.edu/newsletter\_article/how-addiction-hijacks-the-brain.
- Volkow, N.D., Wang, G.J., Fowler, J.S., & et al. (2010).
   Addiction: Decreased reward sensitivity and increased expectation sensitivity conspire to overwhelm the brain's control circuit. *BioEssays*, 32(9), 748-55.
- Kidd, C. & Hayden, B.Y. (2015). The psychology and neuroscience of curiosity. *Neuron*, 88(3), 449-60.
- Stahl, A.E. & Feigenson, L. (2015). Observing the unexpected enhances infants' learning and exploration. *Science*, 348(6230), 91-4.
- Milteer, R.M., Ginsburg, K.R., & et al. (2012). The importance of play in promoting healthy child development and maintaining strong parent-child bond: Focus on children in poverty. *Pediatrics*, 129(1), e204-13.
- Sifferlin, A. (2015). 6-month-old babies are now using tablets and smartphones. *Time*. Retrieved from: http://time. com/3834978/babies-use-devices/.
- Russo-Johnson, C., Troseth G., Duncan, C., & et al. (2017).
   All tapped out: Touchscreen interactivity and young children's word learning. Frontiers in Psychology, 8, 578.
- Kuhl, P.K. (2004). Early language acquisition: Cracking the speech code. Nature Reviews Neuroscience, 5(11), 831-43.
- Lovato, S.B. & Waxman, S.R. (2016). Young children learning from touch screens: Taking a wider view. Frontiers in Psychology, 7, 1078.
- Grant H. & Dweck, C.S. (2003). Clarifying achievement goals and their impact. *Journal of Personality and Social Psychology*, 85(3), 541-53.
- Qu, Y., Fuligni, A.J., Galván, A., & et al. (2015) Buffering effect of positive parent–child relationships on adolescent risk taking: A longitudinal neuroimaging investigation. Developmental Cognitive Neuroscience, 15, 26-34.

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# Connecting the Brain to the Rest of the Body: Early Childhood Development and Lifelong Health Are Deeply Intertwined

**WORKING PAPER 15** 

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This paper is dedicated to Bruce S. McEwen, Ph.D. (1938-2020)

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## The Issue: Health and Learning Are Interrelated in the Body but Separated in Policy

A GROWING UNDERSTANDING OF HOW RESPONSIVE RELATIONSHIPS AND LANGUAGE-RICH experiences for young children help build a strong foundation for later success in school has driven increased investment and sparked innovation in early learning around the world. The rapidly advancing frontiers of 21st-century biological sciences now provide compelling evidence that the foundations of lifelong *health* are also built early, with increasing evidence of the importance of the prenatal period and first few years after birth. The science is clear on two points:

- 1. What happens during this period can have substantial effects on both short- and long-term outcomes in learning, behavior, and both physical and mental health.
- 2. All of these domains are remarkably interdependent and the potential for learning is inexorably linked to the quality of physical and mental health.

A child who is living in an environment with supportive relationships and consistent routines is more likely to develop wellfunctioning biological systems, including brain circuits, that promote positive development and lifelong health. Children who feel threatened or unsafe may develop physiological responses and coping behaviors that are attuned to the harsh conditions they are experiencing at the time,<sup>2</sup> at the long-term expense of physical and mental well-being, self-regulation, and effective learning.<sup>3</sup> Policymakers, leaders of human services systems, intervention developers, and practitioners can all use this knowledge to create innovative solutions to reduce disparities in preventable diseases and premature deaths and lower the high costs of health care for chronic illnesses that have their origins in early childhood adversity.<sup>4,5</sup> Moreover, these costs are likely to grow unless society's investment in promoting health and preventing disease moves "upstream" to address the sources of these problems in early childhood.

Nearly all aspects of early development and later health are affected by interactions among experiences, genes, age, and the environments in which young children live. These interactions influence every biological system in the body, with especially powerful effects in the earliest years.<sup>6,7</sup> Systems relating to brain development, heart and

lung function, digestion, energy production, fighting infection, and physical growth are all interconnected and influence each other's development and function. Each system "reads" the environment, prepares to respond, and shares that information with the others. Each system then "signals back" to the others through feedback loops that are already functioning at birth.<sup>8</sup> As an example,

The environments we create and the experiences we provide for young children and their families affect not just the developing brain, but also many other physiological systems.

higher rates of infection in early childhood can increase the level of anxiety at later ages<sup>9</sup>, which can then compromise school performance. Children living in conditions of threat and deprivation may emerge as adults with a greater risk for multiple forms of cardiometabolic disease. In short, the environments we create and the experiences we provide for young children and their families affect not just the developing brain, but also many other physiological systems, from cardiovascular function and immune responsiveness to metabolic regulation. All of these systems are responsible for our lifelong

health and well-being.

The brain and all other organs and systems in the body are like a team of highly skilled athletes, each with a specialized capability that complements the others and all of whom are dedicated to a common goal. The members of a well-functioning team read each other's actions, adjust their own actions according to what happens around them, and continuously learn from each other. Over time, biological systems in the body mature into a finely tuned unit and respond as one to a multitude of challenges. As their shared experiences or environments change, these systems must adjust, just as players in each position must respond. Each performance builds on what came before and, while adjustments are always possible, it is more difficult—and more costly to change strategies, patterns, and habits later than to build a well-functioning and efficient team from the beginning. And just as every team is different in how the players respond and adjust to their environment, so is every child. The core concepts of development

apply to every individual, but how these systems adapt and interact can vary, and these differences are essential for developing effective prevention and intervention strategies based on 21st-century science.

The policy and practice implications of this knowledge are striking: Strategic investments in young children and the adults who care for them affect long-term physical and mental health as much as they affect early learning. When access to essential resources and supportive relationships is secure, the building blocks of both resilience (e.g., selfregulation and adaptive skills) and wellness (e.g., well-regulated stress response systems) are strengthened. 10 When hardships or threats are extreme or persistent, particularly in the context of intergenerational poverty and/or systemic racism<sup>11</sup>, multiple biological systems can be disrupted. The "downstream" results of these disruptions are poor educational achievement, lower economic productivity, higher rates of crime, and increased heath care costs, 12,13,14,15

## What 21st-Century Science Is Teaching Us

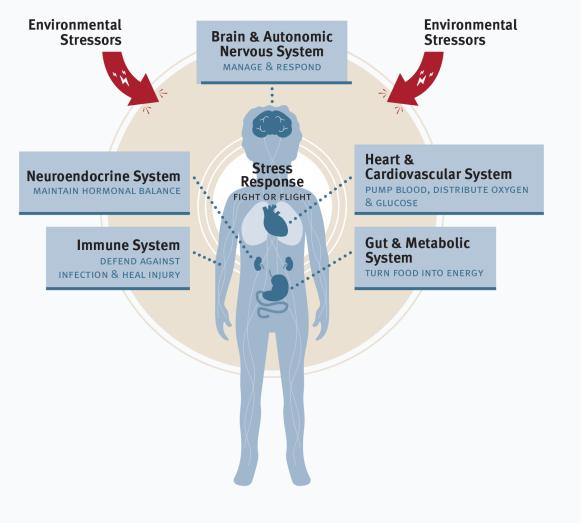
All biological systems in the body interact with each other and adapt to the contexts in which a child is developing—for better or for worse-and adaptations in one system can influence adaptations in others. Think about how all the systems in a young child's body must function in a highly coordinated way to respond to challenging conditions. The initial biological response is the same whether the experience is a short-lived, normative experience, like the first day in a child care center, or the ongoing trauma of recurrent physical abuse—it's the duration, severity, and timing of the experience (along with the availability of supportive relationships) that determine whether the response is ultimately harmful or growth-promoting.<sup>16</sup> In both situations, the body's stress systems respond by coordinating multiple interactive components: (1) the autonomic nervous system increases heart rate and breathing so the cardiovascular

system can pump more oxygen-rich blood to the brain and muscles to drive the "fight or flight" response; (2) the immune system is activated to fight against the possibility of open wounds and infection; (3) metabolic systems are tuned up to generate more energy to fuel the body's cells, tissues, and organs; (4) the neuroendocrine system maintains the delicate balance of hormones that regulate many dimensions of the body's adaptation to what it senses in the environment.

This integrated response to threat is a vivid example of team players working toward a common purpose: All of these systems are robustly interconnected and together help the body adapt to the environment around it. The brain receives signals from each system, which influence how it works (and can even alter its chemistry and architecture), and then sends signals *back* to other organs. For example, multiple studies show that physical exercise

## **Biological Systems Interact With** Each Other and the Environment

When external threats trigger the body's stress response, multiple systems spring into to action like a team of highly skilled athletes, each with a specialized capability that complements the others. Systems relating to brain activity, heart and lung function, digestion, energy production, and fighting infection are all interconnected and influence each other's development.



promotes cardiovascular health and also stimulates the processes that lead to new neural connections and increased blood flow in the brain that improve memory and mood. 17,18,19,20,21 Diabetes is associated with problems in sugar metabolism that can affect tiny blood vessels in the eyes and kidneys that may lead to impaired vision and kidney malfunction. These same

metabolic disruptions can also produce changes in brain architecture that may lead to impaired mood and memory, as well as increased risk for later dementia. 22,23 These are just a few of many examples that illustrate the connections between the brain and the rest of the body.

Our bodies are designed to maintain a healthy physiological balance and to restore it when it's disrupted. The continuous interactions and responsive feedback among multiple systems are designed to seek and sustain that balance within a relatively narrow range of operation, a process that scientists call *homeostasis*. Normal body temperature, for example, is programmed to remain at around 98 degrees Fahrenheit, and too much variation on either side triggers multiple physiological responses to restore a normal range (e.g., sweating decreases body temperature and shivering increases it).

# early in life can overload biological systems and lead to long-term consequences. Mobilizing the body's responses to threat diverts energy away from growth and healthy development. For a child experiencing "time out" for a temper tantrum, the challenge will pass.

**Excessive and persistent adversity** 

from growth and healthy development. For a child experiencing "time out" for a temper tantrum, the challenge will pass, balance is restored, and biology can return to the business of building a healthy brain and body. Not so for a child experiencing the persistent threat of maltreatment, as continuing activation of the stress response will compromise the body's investment in growth.

The process of the body adapting to manage threats, such as increased blood pressure as a response to stress, is what scientists call allostasis. If a threat or hardship is too intense or prolonged, it results in allostatic load or overload.24 As with any overloaded system, allostatic load can lead to breakdowns (i.e., physiological and behavioral changes that can undermine both physical and mental health). Elevated blood pressure, for example, is initially part of the stress response that gets needed blood, nutrients, and oxygen to all cells in the body, but if it is too high for too long it damages arteries, which can lead to a heart attack or stroke.

If the body receives indications that the environment is generally predictable and presents manageable challenges, a child can develop a well-regulated stress response system more readily. If, however, the brain perceives excessive, frequent, or persistent

## **Homeostasis and Allostasis** Homeostasis Seeking balance **Allostasis** Long-term adaptations to maintain balance **Environmental** stressors Allostatic Load/Overload Long-term adaptations become problems **Environmental** stressors

threat, it learns to expect adversity and develops a "shorter fuse" for activating physiological responses throughout the body.<sup>25</sup> These adaptations can lead to costs as well as benefits—they are health-protecting in acute, short-term situations but can become health-damaging if activated at too high a level for

too long.26,27,28 Such tradeoffs often occur when the body adapts to one environment (e.g., a threatening one) but later needs to adjust to different conditions (e.g., a neutral situation).

Consider, for example, children developing under conditions of poverty, where dedicated parents and other caregivers are burdened by the challenges of making ends meet. These challenges are often embedded in structural inequities such as residential segregation, food deserts, and limited employment opportunities. Nutritious food may not be readily available, stable housing may not be assured, and constant economic worries and unpredictability may impose continuous distractions on daily adult/child interactions, which activate multiple components of the stress response. Some children may develop behaviors that help them adapt to and cope with these conditions of scarcity or fear (e.g., binge eating whenever possible), yet these short-term adaptations can become problematic later in life.

For children who are not living under conditions of chronic hardship, activation of stress response systems that are brief and intermittent, followed by a return to balance, leads to healthy adaptations that build resilience—just as a fire drill prepares children for an emergency but then restores order after a short time. In contrast, if stress responses remain activated at high levels for long periods, this can have a significant wear-and-tear effect on the brain and other biological systems. In other words, if children were disrupted by urgent fire drills nonstop for days, weeks, or months, they would be worn down over time and less likely to respond effectively to a

true emergency. In the body, this cumulative burden can lead to both short- and long-term consequences that may include maladaptive behaviors (e.g., difficulties with impulse control, addictions), a "weathering" effect that accelerates the aging process, chronic illness in adulthood, and a shortened lifespan.29

A growing body of evidence from both the biological and social sciences builds on this concept of chronic wear and tear. Beyond the cumulative effects of chronic adversity more generally, this research provides a compelling framework for exploring how well-documented racial disparities in health, independent of socioeconomic status, may be rooted in the effects of both individual and systemic racism on early childhood development.30 At an individual level, multiple studies have documented how the stresses of everyday discrimination on parents or other caregivers can affect caregiving behaviors and adult mental health, and by extension child development. 31,32,33 At an institutional level, researchers are investigating how structural inequities and discriminatory laws affect the context in which families of color raise children. Unequal access to high quality education and health services, economic opportunities, and wealth accumulation, compounded by racial disparities in the child welfare and criminal justice systems, provide multiple examples of how the legacy of racism in policies and systems has created conditions that disproportionately undermine the health and development of children and families of color.34

## How Early Adversity Affects Developing Biological Systems

When stress responses are activated frequently, intensively, and persistently during early childhood, the systems involved can become permanently calibrated to activate more easily and may not turn off as readily as they should.35,36,37 From a biological perspective, this is essential for survival. If the world is a dangerous place, the internal systems designed to protect us need to develop in a

way that anticipates frequent threats. Yet over time, these repeated activations lead to greater risk for stress-associated diseases well into the adult years—conditions such as cardiovascular disease, obesity, type 2 diabetes, respiratory and immunological disorders, and a range of mental health problems.<sup>38,39</sup> That's the trade-off of adapting to significant early adversity.

What might be happening in a young

child's environment that could transform an adaptation that is advantageous in the short term into an unhealthy, chronic stress activation with long-term consequences? Among the possible answers:

- the socioeconomic hardships of poverty;
- the material and psychosocial burdens of intergenerational racism or other forms of institutionalized discrimination;
- the psychological threats of maltreatment and community violence;
- the interpersonal challenges of maternal depression and parental addictions;
- the physiological disruptions of air pollution and environmental toxicants;
- the metabolic consequences of inadequate or excessive nutrition;
- the developmental burdens of chronic disease or disability.

Any of these stressors—particularly when perpetuated by recurrent triggers and/or systemic barriers to effective prevention, reduction, or mitigation—can contribute to an environment that may persistently and intensely activate a developing child's stress response systems.<sup>40</sup>

Poor health outcomes are not inevitable, but they are more likely if we do not adequately support children and families experiencing persistent hardships or challenges.

> Physiological systems typically work most effectively when they operate within a wellregulated range—and significant deviations beyond either end of that range can lead to problems in physical and mental health. For example, an immune system that doesn't react at a sufficiently high level will be unable to fight off serious infection, but one that is hyperreactive could flood the body with disease-causing inflammation. When highly stressful experiences persist, biological dysregulations can result in either direction. The brain, for example, might become overly primed to trigger fight-or-flight responses when threats are relatively low, while the neuroendocrine system that elevates cortisol levels might become blunted and respond less

vigorously after it has been activated repeatedly for a long time. These paradoxically lower cortisol levels are often seen as a result of chronic abuse and neglect.<sup>41</sup> Although the exact causal mechanisms have not been identified, this diminished cortisol activation has been associated with an increase in body fat<sup>42</sup>, social and behavioral problems in maltreated children<sup>43</sup>, and high levels of depressive symptoms in women with low income.<sup>44</sup> When effective treatment is provided, these systems can regain their responsivity.<sup>45,46</sup>

Finally, it is essential to remember that there are many opportunities to build resilience in the face of significant adversity—beginning in early childhood and continuing throughout life—by providing supportive relationships in predictable environments, reducing sources of significant stress, and building a toolkit of adaptive skills. Poor health outcomes are *not* inevitable, but they are more likely if we do not adequately support children and families experiencing persistent hardships or challenges.

Below are descriptions of how significant adversity affects three biological systems—three members of the stress-response team that also includes the lungs, the endocrine system, and the gut microbiome (i.e., bacteria that live in the intestines), among others—which illustrate how they are all interrelated with each other as well as with other systems.

Effects of excessive early adversity on the **developing brain:** The foundations of brain architecture are built during the prenatal, infant, and toddler periods and shaped by experiences, interacting with genes, in an environment of relationships over time. 47,48 During these periods of rapid development, the brain is as adaptable and flexible as it will ever be. This means that the brain's developing circuits are also highly sensitive to the disruptive effects of elevated stress activation, which releases a flood of hormones, immune responses, and neurotransmitters (the chemicals that send signals from one brain cell to another). Three brain systems are particularly susceptible: (1) emotion regulation systems, which include the amygdala, where circuitry for processing fear and threat develops early in life; (2) memory systems,

which include the hippocampus, where circuitry for memory and simple learning (e.g., remembering the location of an object) begins early and continues into later childhood;49 and (3) executive function systems, which include the prefrontal cortex and other brain regions, where circuitry for focused attention, impulse control, and higher level cognitive skills develops well into the adult years. 50,51,52,53,54,55 These executive systems also help moderate stress responses (by regulating other brain regions) as well as the immune response to threat (by influencing the amount of inflammation that is mobilized to protect the body). Inflammation is one of the core features of the fight or flight response, as it prepares the body for potential wound healing and protection from infection, and the brain influences when and how much it is needed. 56,57

Brief stress-system activation is protective in a dangerous environment, as it prepares the body and brain to respond to an acute threat. But these systems need to recover and return to balance after the source of the stress is eliminated or reduced. If they do not—if the stressors are severe, long-lasting, or there is a lack of supportive relationships to help children calm these responses—they can result in a brain that is "stuck" in a state of high alert. Over time, this can have harmful wear-and-tear effects. The earlier in life this kind of frequent, unmoderated response to adversity occurs, the greater the risk for stressrelated health problems that will be more resistant to treatment well into the adult years. Recent research also has found that significant adversity before birth or in early infancy can build a brain that is more susceptible to harm from repeated stressors later in life.<sup>58,59</sup> Remediation may be possible at any age, but outcomes are better and easier to achieve when interventions are provided earlier and promoting the healthy development of biological systems from the beginning is better, and more cost-effective, than trying to fix them later.60

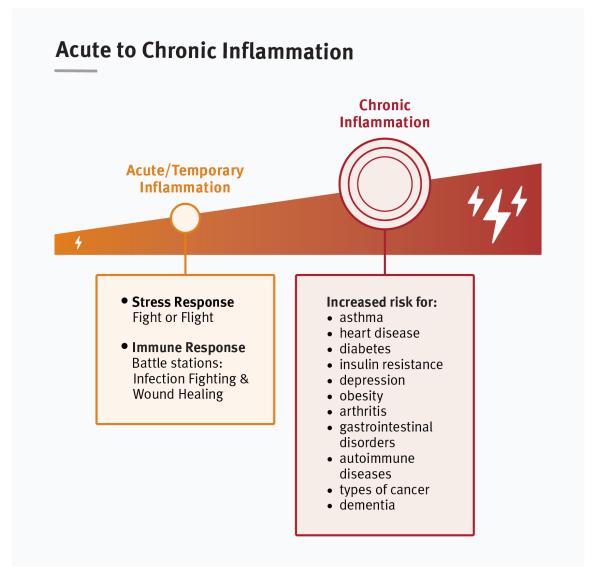
Effects of excessive early adversity on the developing immune system: The immune system defends the body against infection and a variety of toxic substances. One of the most

important components of the immune system's response is inflammation, a physiological function that attacks invading bacteria or viruses, clears out the tissue destruction they cause, and begins the repair process. Acute stress (triggered by experiencing or witnessing a brief but traumatic event) activates an inflammatory response by causing immune cells to "go to their battle stations." Our bodies need this physiological mobilization for survival. Chronic stress (experienced

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over a prolonged period of time in a threatening environment) can cause persistent inflammation. This prolonged state of alert puts powerful inflammatory substances used to kill microbes in constant contact with body organs, which can eventually damage them. At the same time, a constant state of activation also weakens the immune system, making it less efficient in its fight against microbes. 61 This double hit makes children living in adverse environments more susceptible to recurrent infection and more prone to develop chronic inflammatory conditions that may last for a lifetime<sup>62</sup>, including heart disease, diabetes, depression, arthritis, gastrointestinal disorders, autoimmune disorders, multiple types of cancer, and dementia, among many others.

Asthma provides an illustrative example of the consequences of too much inflammation in childhood. Household stress, exposure to auto exhaust and other forms of pollution, tobacco smoke, allergens, and a wide variety of viruses can all contribute to an increased inflammatory response in the lungs—especially in children who carry genes that make them more susceptible to developing asthma. 63,64 This recurrent inflammation, in turn, can stimulate reactions that make the bronchial



muscles twitchy and overreactive to usually innocuous triggers. As a consequence, the airways become too narrow, making it more difficult to breathe, and the child has asthma attacks that require medical intervention. Over time, chronic inflammation and overreaction in the small airways can cause structural changes in the lungs that increase the risk of developing chronic lung disease in the adult years. <sup>65</sup> Bottom line: If challenging *experiences* (e.g., exposure to stress, pollutants, or allergens) prompt a significant *immune response* (i.e., inflammation) early in life, and that response stays activated for too long, it can lead to greater risk for lifelong illness.

As one of the most common chronic illnesses in children, asthma illustrates the powerful influence of gene-environment

interaction on physical health. Research shows that this condition is more frequent and more severe in children living in families with low income, children exposed to poor housing conditions, and children of color, regardless of income, whose families report experiencing discrimination. These findings illustrate the extent to which structural inequities that affect the environments in which families raise children can undermine the foundations of health and well-being in the early years of life. The several seve

Another example of how a child's immune system becomes finely tuned by experiences or environmental exposures comes from recent studies on the bacteria and viruses that normally live in our intestines (what scientists refer to as the "gut microbiome"). Beginning in the late phases of pregnancy, the fetus prepares

to adapt to an outside environment that is packed with a wide variety of microorganisms (or microbes). After birth, the many different ways in which newborns are held, fed, and cared for affect which bacteria and viruses become inhabitants in their bodies. The nature of these microbes is influenced by differences between vaginal and Caesarian deliveries, breast- and bottle-feeding, the type and quality of ingested nutrients, and the physical environment in which caregiving is provided. For example, children who live on farms beginning in early infancy are exposed to a diversity of bacteria and viruses that stimulate adaptive immune responses that result in much lower rates of allergies and asthma as they get older, compared with young children living in rural areas with non-farming families. 68,69

Living in a wide variety of socially nurturing environments, with multiple interactions among caregivers, siblings, pets, and other human beings, allows an infant to acquire a robust and diverse microbiome. In contrast, lack of physical interaction with a variety of other people or obsessive cleanliness can lead to a microbiome that is not sufficiently varied. This results in an immune system that encounters fewer opportunities to distinguish biological "friend" from "foe" and to regulate immune responses in organs even far away from the gut itself, and may therefore respond excessively to usually innocuous viruses and allergens in early life.

#### Effects of excessive early adversity on developing cardiometabolic systems:

This network produces, distributes, and/or regulates the physiological fuel (e.g., oxygen and glucose) cells need throughout the body via the circulating blood stream. When the stress response is activated, this system springs into action (e.g., elevated heart rate, blood pressure, and blood glucose level) to provide increased energy to respond to threat while it ramps down other systems (e.g., digestion) that are relatively less essential in an acute situation. The impact of poor nutrition on cardiovascular health is well-documented.<sup>70</sup> Obesity and elevated blood pressure are also more prevalent in children experiencing the stresses of poverty, racism, unsupportive caregiving, overstimulation from excessive noise and overcrowding, and sedentary behavior from living in a violent neighborhood with no safe space for playing outdoors. 71,72,73,74 There is also growing evidence that inflammation is an important contributor to that increased risk, and the combination of stress and inflammation is especially threatening to health and well-being over time. For example, excessive amounts of stress hormones such as cortisol, combined with chronic inflammation, can result in insulin resistance—a physiological disruption that can lead to metabolic syndrome, obesity, diabetes, and cardiovascular disease, as well as brain changes and cognitive impairment.75,76,77

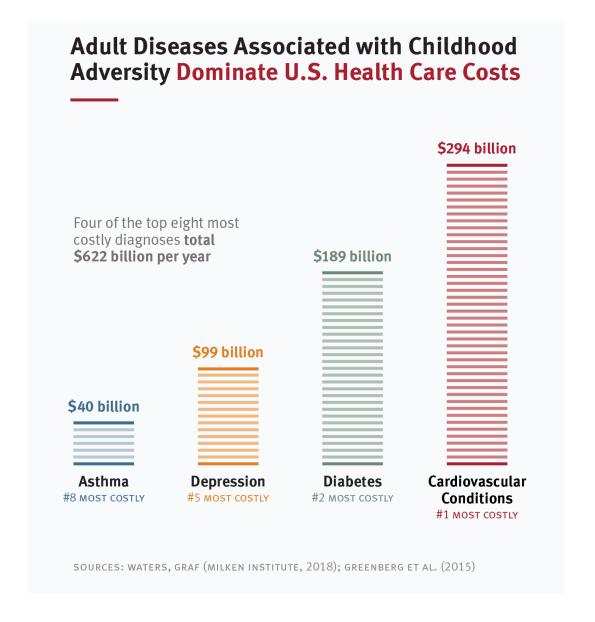
Addressing the early childhood stressors that lead to chronic, lifelong inflammation might dramatically reduce the need for costly treatments for a wide range of multiple health conditions, including cardiovascular disease.

In 2018, the American Heart Association issued a Scientific Statement that cited substantial evidence documenting an association between childhood and adolescent adversity and adult cardiometabolic disorders (e.g., obesity, hypertension, type 2 diabetes, and cardiovascular disease). Based on that growing knowledge base, the Statement asserted the following: "Given that childhood adversities affect cardiometabolic health and multiple health domains across the life course, interventions that ameliorate these initial upstream exposures may be more appropriate than interventions remediating downstream cardiovascular disease risk factor effects later in life." 78,79 In other words, addressing the early childhood stressors that lead to chronic, lifelong inflammation might dramatically reduce the need for costly treatments for a wide range of multiple health conditions, including cardiovascular disease.

# **Common Illnesses in Adults Have Roots in Early Childhood Adversity**

Three Chronic Health Impairments in the United States—cardiovascular disease, diabetes, and depression—together account for nearly \$600 billion in *direct* health care expenditures annually (above and beyond their indirect costs, such as lost productivity). 80,81,82 According to the Centers for Disease Control and Prevention, heart disease and stroke alone kill more than 859,000 people in the United States every year (accounting for one-third of all deaths and an even higher percentage in communities of color) and are also estimated

to account for \$131 billion annually in lost economic productivity. More than 30 million people have diabetes (a disproportionate number of whom are people of color) and another 84 million adults have a condition called prediabetes, up to 70% of whom will eventually develop diabetes. 83,84 All mental health and substance abuse disorders together constitute the most expensive category of chronic conditions, with depression alone incurring \$99 billion in health care costs annually. 85 Beyond their financial cost, these



conditions also have enormous impacts on the quality of life of individuals, families, and communities.

The prevalence of cardiovascular disease, diabetes, and depression in adults is associated with higher rates of adverse experiences in childhood—and advances in biology are beginning to explain how and why that happens. Although they may appear to be unrelated on the surface, all three share a common association with elevated inflammation, which, as described above, can be influenced by recurrent hardships or threats in early childhood. These conditions are far from the only ones that could be listed, but they are among the most common and most costly of many possible examples that all point in the same direction: Efforts to prevent many chronic illnesses in adults need to begin in the early childhood years.

**Cardiovascular disease:** This diverse category of disorders includes medical conditions that involve narrowed or blocked blood vessels that can lead to sudden death or compromised life due to hypertension, chest pains (angina), a heart attack, or a stroke. The process that causes this narrowing or blockage is called atherosclerosis, which can begin early in life. This process involves a buildup of fatty deposits (called plaques) that thicken and stiffen artery walls, which can then lead to decreased blood flow to the heart muscle and brain, as well as to other body tissues.

As with any medical condition, understanding the underlying causes of cardiovascular disease can lead to effective prevention and treatments. Chronic inflammation, which as we've seen has its roots in early childhood, accelerates atherosclerosis by disrupting the walls of arteries and making them more likely to be sites for plaque formation and build-up. Although still an area of extensive scientific research, a biomarker of inflammation called C-reactive protein is measured by some physicians as another way to screen for cardiac risk. These findings make a strong case that detecting and reducing chronic inflammation, starting in early childhood, may be as important as lowering cholesterol for preventing a heart attack.

## Nutritional factors during pregnancy and early infancy have been associated with heart disease later in adulthood.

Other underlying causal factors also point toward early childhood origins. Most of the public is aware that the risk of a heart attack or stroke is increased by an unhealthy diet (i.e., high in fats, salt, and sugar), lack of exercise, excess weight, and smoking. Many people also understand that high levels of "bad" cholesterol in the blood can increase the formation of plaques and thus accelerate atherosclerosis—and that poorly controlled high blood pressure can result in hardening and thickening of the arteries, which can then obstruct blood flow.86 Less well known is the extent to which nutritional factors during pregnancy and early infancy have been associated with heart disease later in adulthood.147 Although more research is needed to fully explain the causal mechanisms underlying this association, one compelling hypothesis<sup>87</sup> points to conditions of food scarcity during pregnancy leading to

#### **WHY EARLY MATTERS: CARDIOVASCULAR DISORDERS**

Heart disease is the leading cause of death for men, women, and most racial and ethnic groups in the United States.132 Toxic stress in childhood is frequently associated with elevated inflammation<sup>133</sup> and atherosclerosis (which decreases blood flow to the heart in adults). Although scientists are still learning how the immune system's response to adversity in the early childhood period influences the development of cardiovascular disease in adults, there is enough evidence now to test the hypothesis that reducing early adversity could lead to breakthrough reductions in adult cardiovascular disease.

The U.S. spent \$294 billion on direct health care costs and at least \$137 billion in lost productivity for cardiovascular conditions in 2018.134 altered fetal growth (e.g., low birth weight) and a metabolic system "programmed" for relatively excessive food intake in early childhood, leading to greater risk for type 2 diabetes and heart disease later in life.

Diabetes: This diagnosis includes several subtypes of chronic disease, all of which result in high levels of blood glucose (sugar) that persist over time. Glucose provides the main source of energy for cells in most body tissues. Insulin, a hormone made by the pancreas, controls the process by which glucose gets into most of those cells. Insulin, in a sense, "opens the doors" to allow glucose to enter into cells. If the pancreas doesn't produce enough insulin, or if the insulin is not opening those cell doors effectively, glucose stays in the blood and its level rises. When high levels of blood sugar are detected, it means that cells throughout the body are not getting enough of the glucose they need to function well.

#### WHY EARLY MATTERS: DIABETES

Extensive evidence indicates that disturbances in a pregnant woman's metabolic systems can "program" greater risk in the fetus for later development of type 2 diabetes and excess body fat. 135 These prenatal influences include insufficient protein and calories in the mother's diet-or too many calories-as well as hormones that affect developing systems that will regulate body weight and energy in the baby after birth. Studies of children born to mothers who were pregnant during times of famine found higher rates of obesity, insulin resistance, and diabetes than in children born in the same area a year or two earlier. 136,137,138 Substantial research findings also demonstrate sensitive periods in early infancy for long term effects of overnutrition (having more calories or nutrients than are needed for healthy growth) on the risk for later obesity and diabetes. 139,140,141

The U.S. spent \$237 billion in direct medical costs and lost \$90 billion in reduced productivity due to diabetes in 2017.142

In type 1 diabetes, which has a strong genetic component, the pancreas produces little or no insulin. In type 2 diabetes, which accounts for about 90% of diabetes diagnoses and is associated more strongly with environmental influences, insulin is produced but it doesn't work as well as it should.88,89 This results in a condition called insulin resistance, in which the insulin is less able to "open the doors" and glucose stays in the bloodstream. The relation between insulin resistance and elevated inflammation is well-documented, although the nature of the association is not yet fully clear.90

The body's main source of glucose comes from what we eat and drink. Nutritious food that is accessible and affordable is an important protective factor against persistently elevated blood sugar and excessive intake of "junk food" increases the risk of disease. Activation of the stress response increases glucose levels in the blood in order to generate more energy to fuel the "fight or flight" response, which prepares our muscles and brain to deal with a sudden threat. But when the stress response persists over time, especially during the early developmental years, prolonged glucose elevation can trigger a cascade of events culminating in insulin resistance, metabolic syndrome, obesity, and eventually a diagnosis of type 2 diabetes. 91,92 Over the full life course, chronically elevated blood sugar can lead to greater risk of cardiovascular disease, kidney disorders, neurological impairments, and vision problems. Persistent insulin resistance, which can affect the brain, is also a risk factor for cognitive impairments, depression, and Alzheimer's disease.93

**Depression:** Clinical depression (also known as major depressive disorder or MDD) is one of the most common mental disorders in the United States and around the world. In the U.S., more than 7% of all adults and 13% of adolescents experienced at least one major depressive episode in 2017. 94 Individuals with diagnosed depression experience a range of symptoms that affect how they feel, think, and manage everyday tasks. Extensive evidence indicates that MDD is caused by a combination of genetic, biological, environmental, and psychological factors that interact in a variety of ways.95 The following are some of the facts about MDD that are well-documented by scientific evidence:

- Adults who experienced serious trauma in childhood are at greater risk (indicating that experiences are an important factor).96,97,98
- It occurs with greater frequency in some families more than others (indicating that genes also play a role).99,100,101
- It is twice as common in women as in men (although the reason is not known, some animal studies have found sex differences in adult behaviors following early life adversity, including more depressive-like patterns in females in contrast to more aggressive behaviors in males). 102,103,104
- It is more common in urban populations than in rural areas (indicating that social and physical environments may also contribute).105
- Acute episodes are reported more commonly in whites in contrast to higher rates of chronic depression in communities of color.106
- Approximately one in seven pregnant and postpartum women nationwide are affected by mood and anxiety disorders, and 40-60 percent of low-income women report such maternal depressive symptoms. 107
- Studies of diverse samples of pregnant and postpartum women have found minimal effects of screening on ameliorating depressive symptoms or increasing use of behavioral health care. 108

#### WHY EARLY MATTERS: DEPRESSION

The prevalence of depressive disorders is markedly increased among people who face adversities related to poverty, homelessness, and exposure to violence. 143,144 Although researchers do not yet fully understand the relative impacts of the age at which the adversity was experienced, the duration of the adverse conditions, or the cumulative build-up of stress over time, recent research has identified *significant adversity in the first* three years after birth as a potential critical period associated with greater risk for clinical depression in adulthood. 145

The U.S. spent \$99 billion on direct health care costs and \$112 billion on indirect costs for MDD in 2010.146

There is extensive evidence that clinical depression, like cardiovascular disease and diabetes, is associated with increased inflammatory activation and insulin resistance. 109,110 Although many questions remain about whether this link reflects a cause or an effect, these associations are well-documented and underscore the importance of learning more about the relations among early life adversity, persistent inflammation, insulin resistance, and impairments in both mental and physical health throughout the adult years.

## Facts About Health That Are Often Misunderstood

The experiences we have early in life are at least as important for the biological foundations of physical and mental health as the lifestyle choices we make as adults.

Critical or sensitive periods provide unmatched opportunities for both positive and negative influences on developing biological systems. Above and beyond well-known impacts on early brain development, increasing evidence is also pointing to the importance of the prenatal period and first few years after birth for the development of core immune functions, metabolic regulation, and other physiological

systems that can affect long-term wellbeing. 111,112,113 Without dismissing the influence of adult lifestyle (including nutrition, exercise, and sleep) on physical health, early adversity can increase the risk for many of the most common chronic diseases that appear later in life and that incur substantial costs to society.

Health-promoting environments early in life are critically important for building a strong foundation, but it's never too late to reduce risk. Although effective interventions can produce improvements in health and behavior

throughout life, the ability of the brain and other biological systems to adapt and change generally decreases as we age. 114 The brain can compensate for early disruptions at later stages, but full "reversals" are rare. 115 Stated simply, life is a continuous "one-way street"—what happens at each stage, including events before conception and during pregnancy, has consequences for what follows. Changing course is possible, but the changes we make later in life must contend with foundations that were laid down in the early years.

Expanding access to health care and decreasing utilization of unnecessary services are not the same as producing documented effects on child health.

Although access and delivery of appropriate services are clearly important goals, direct measures of health status are needed to assess physical and mental well-being, identify problems that require intervention, and quantify the effects of services received. Since the launch of Head Start in 1965, programs have been mandated to promote health and address unmet medical needs by ensuring that all children receive pediatric examinations, immunizations, dental care, and assessments of nutrition, growth, vision, hearing, and speech.116 Over more than half a century, the most common metric used to assess health impacts has been the delivery of health-related services, often supplemented by data on cost savings from decreased emergency room visits and hospitalizations. The need for greater attention to direct measures of child health outcomes (e.g., rates of common diseases) as well as indicators of health risk (e.g., biomarkers of excessive stress activation) is clear.

## **Future Directions for Policy and Practice**

The effects of significant adversity on brain functions associated with early learning, social and emotional development, and kindergarten readiness are well-documented. This knowledge has influenced policy objectives, program design, allocation of resources, and expected returns on investment in the early childhood period for decades. The rapidly moving frontiers of the biomedical sciences now underscore the compelling need for an expanded mindset, informed by a deeper understanding of how early adversity can disrupt multiple biological systems in addition to the brain, with serious consequences for long-term physical and mental health.

This new mindset views investment in the early years as a necessary priority for strengthening the foundations of both health and learning across the lifespan by addressing the common origins of disparities in each. The implications of this rapidly advancing scientific knowledge for a new era in early childhood policy and practice point to the need for: (1) implementing practical strategies for promoting health and preventing disease; as well as (2) overcoming longstanding barriers to change.

### **Implementing Practical Strategies**

Advances in science should be informing the design, testing, iteration, and eventual scaling of innovative intervention strategies to protect the developing brain and other biological systems from the disruptive effects of early adversity.

Above and beyond assuring enriched learning experiences for children and information on child development for parents and other caregivers, the biology of adversity and resilience points to three science-based principles<sup>120</sup> that should be used to inform more effective policies and programs across all sectors to strengthen the early childhood foundations of lifelong health.

• Support responsive relationships.

Reliable "serve and return" interactions between young children and the adults who care for them help to reduce the physiological disruptions of excessive

stress activation and protect developing biological systems, especially in the earliest years. Examples of policies or programs that align with this principle include: (1) giving parents and other primary caregivers the time needed to build the foundations of nurturing relationships with their children (e.g., paid family leave after the birth or adoption of a child); (2) minimizing disruptions of stable, adultchild relationships in child care centers (e.g., reducing staff turnover by providing competitive compensation through wages and benefits, as well as requiring reasonable adult-to-child ratios); (3) providing relationship-focused coaching for primary caregivers when needed; (4) focusing on the need to support continuing contact between children and parents who are separated in the child welfare system or when a parent is incarcerated (both of which are associated with longstanding racial disparities); and (5) protecting family cohesion in the design and implementation of immigration policies, both for newly arriving refugees where the risk of detention and parent-child separation is high, and for mixed-status families where the fear of separation is constant.

**Reduce sources of stress.** Policies and programs that lessen economic and psychosocial burdens on families with young children pay off in two ways. First, they reduce chronic activation of stress systems in both adults and children. Second, they enhance adult capacity for providing responsive caregiving that facilitates healthy child development. Examples include policies and practices that: (1) bolster safety-net policies that address income, nutrition, housing, and medical insurance needs; (2) eliminate punitive or unnecessary administrative regulations (e.g., streamlined eligibility processes for needed services); (3) address community and intimate partner violence; and (4) reduce systemic and economic racism<sup>122</sup> (e.g., fair hiring and lending practices, housing and home ownership programs, community policing initiatives,

and efforts to reduce implicit bias<sup>123</sup>). **Strengthen core life skills.** In order to provide a well-regulated caregiving environment in both the family and community context, adults must be able to set and meet goals, manage their own behavior and emotions, establish daily routines for eating and sleeping, and facilitate social-emotional development and skill-building in children. Wellmatched programs can help both children and adults build and apply these skills (known as executive function and selfregulation) through modeling, coaching, and practice (thereby providing a strengths-based approach similar to the way elite athletes rely on coaching and practice to continuously improve their already well-honed skills). Recognizing that stress can compromise anyone's ability to use the skills that they have can help programs offer supportive, scaffolding techniques, rather than threats of punishment, when existing efforts are not working well.124

This new mindset views investment in the early years as a necessary priority for strengthening the foundations of both health and learning across the lifespan by addressing the common origins of disparities in each.

Primary health care offers a sciencebased delivery channel for reaching the largest number of children at the earliest possible ages in a non-stigmatizing **context.** Team-based care provided through culturally and linguistically responsive, trusted relationships offers a promising model for individualized approaches to building resilience and preventing, reducing, or mitigating the consequences of early adversity. Reducing disparities in child health outcomes at a population level, however, will require a substantial shift in professional training, current practice, and payment systems to address the following challenges:125

- Pressures on physicians that demand brief interactions and high-volume service delivery undermine the ability to build supportive relationships—and more time is needed for families facing adversity. Credible evidence of improved child outcomes (see below) will strengthen the case for adequate funding, and engaging expertise in value-based reimbursement policies will be essential to secure the sustainability of that funding.
- Persistently low rates of routine developmental screening during wellchild visits have been extremely difficult to improve, despite decades of task force recommendations, explicit mandates, and continuing education programs. 126 The emerging availability of biological and behavioral indicators of excessive stress activation and resilience in young children could present game-changing opportunities to generate more actionable and empowering information for both clinicians and parents/caregivers.
- Evaluations of "evidence-based" interventions linked to pediatric practice during the first three years after birth have demonstrated positive impacts on self-reported parenting behaviors but few replicable effects on child outcomes. 127 Direct measurement of key indicators of child health and development (including stress effects and resilience as well as common medical problems) will enhance clinical management and help secure payment for effective services.
- Limited progress in strengthening science-based content on early childhood development (including its underlying biology) and cultural context in pediatric residency programs, as well as in training of other health professions, indicates that a "top down" strategy to transform primary care practice will not be easy to achieve. In contrast, science-informed innovation in well-resourced, community-based laboratories could provide a potentially powerful, "bottom up" strategy to eventual impact at scale.
- Promising elements of this latter strategy include incorporating child development expertise within innovative team models

(e.g., HealthySteps<sup>128</sup>), engaging team members who reflect the cultural and linguistic characteristics of the community, using rigorously validated measures to assess child outcomes directly, and embedding primary health care more seamlessly within community-based systems of services across sectors (e.g., Help Me Grow<sup>129</sup>).

## **Overcoming Longstanding Barriers**

There is an urgent need for more effective strategies to support the health and development of young children by confronting poverty, racism, violence, housing instability, food insecurity, and other sources of chronic adversity that impose significant stresses on families.

Public attention to these social determinants of health is increasing, but simply naming the problem, identifying "high-risk" children and families, and making referrals to services has not resulted in substantial or replicable impacts.

- The longstanding designation of *race* as a risk factor for disparities in health outcomes diverts critical attention away from systemic racism and its deep historical roots as a pernicious cause of stress-related disease.130 Viewed through an equity lens, services and programs must move beyond a sole focus on children and families to an intentional, "upstream" focus on changing macro-level policies that systematically threaten the health and wellbeing of families of color.
- Evidence-based interventions that build resilience in children and caregivers facing adversity can lead to better individual outcomes at a program level, but achieving greater impact at scale will require increased efforts to confront structural inequities—such as unequal access to opportunities in education, health care, and wealth creation—at a societal level. Science alone is unable to address this challenge. But science-informed thinking combined with on-the-ground expertise and the lived experiences of families

raising young children under a wide variety of conditions (many of whom are typically marginalized) can be a powerful catalyst of new strategies at both levels.

All policies and delivery systems serving young children and families across sectors (including but not limited to medical care and early care and education) can and must measure their success by improved child outcomes in both health and learning.

Persistent attempts to increase access to services, reduce their fragmentation, build integrated delivery systems, and secure sustainable funding remain important objectives. But these efforts will not produce greater impacts until the measurement of their success moves beyond serving more children and enhancing interagency collaboration and begins to focus more explicitly on key child outcomes.

Assessing child well-being and determining service needs by focusing

- exclusively on demographic risk factors (e.g., income, race, ethnicity, parent education) or number of adverse childhood experiences (e.g., ACE scores) can result in inappropriate labelling and unnecessary services for children and families who are doing well ("false positives") as well as missed opportunities to provide vital services for those who would benefit from them ("false negatives").
- More informed allocation of resources would be enhanced by supplementing social determinants of health with individual-level data on carefully selected indicators of child and family wellbeing-first to determine both assets and concerns, next to match specific services to identified needs, and then to find out who is benefiting from those services (which should prompt targeted scaling) and who is not (which should catalyze a search for alternative strategies).

## Final Reflections in a COVID-19 World

DISPARITIES IN HEALTH OUTCOMES RELATED to socioeconomic inequalities impose substantial human and financial costs on all societies around the world.131 Significant racial and ethnic differences in hospitalizations and deaths from COVID-19 in the United States have increased attention to this persistent inequity and much initial discussion has focused on conditions that make it more likely that people of color will be exposed to the virus. These include disproportionate employment in "essential" services without adequate protection from infection, residing in tight quarters, and hourly-wage jobs without paid sick leave or the ability to work at home. Inequalities in access to high-quality health care and higher rates of unequal treatment in the health care system have also been highlighted.

A different yet critically important question is why some people who are exposed to COVID-19 are more likely to have serious complications and less likely to survive. The exceptionally high risk of pre-existing medical conditions—including cardiovascular disease, diabetes, respiratory illnesses, and obesity at the top of the list—underscores the importance of the science reviewed in this Working Paper. Health-threatening conditions early in life (including poor nutrition, exposure to pollutants, and high levels of family stress associated with poverty, racism, and other forms of economic or social marginalization) can have disruptive effects on developing immune and metabolic systems, including excessive inflammation, that lead to a variety of health impairments well into the adult years. The implications for greater returns on innovative, science-informed investments in the early childhood period are clear and compelling. The brain is indeed connected to the rest of the body—and early childhood policy in the 21st-century must focus on the overwhelming evidence that early experiences affect the foundations of both educational achievement and lifelong physical and mental health.

## References

- Boyce, W.T., Levitt, P., Martinez, F.D., McEwen, B.S., & Shonkoff, J.P. (2020). More Than Just the Brain (II): Advances in the Developmental Biology of Adversity and Resilience. Under review.
- 2 Gee D.G., Gabard-Durnam, L.J., Flannery, J., Goff, B., Humphreys, K.L., Telzer, E.H., ... Tottenham, N. (2013). Early developmental emergence of human amygdala-prefrontal connectivity after maternal deprivation. Proceedings of the National Academy of Sciences, 110, 15638-15643.
- 3 Blair, C., & Raver, C.C. (2015). School readiness and self-regulation: a developmental psychobiological approach. *Annual Review of Psychology*, 66, 711-731.
- 4 Center on the Developing Child at Harvard University. (2010). The Foundations of Lifelong Health Are Built in Early Childhood. Retrieved from www.developingchild. harvard.edu.
- 5 Knudsen, E.I., Heckman, J.J., Cameron, J.L., & Shonkoff, J.P. (2006). Economic, neurobiological, and behavioral perspectives on building America's future workforce. Proceedings of the National Academy of Sciences, 103(27), 10155-10162.
- 6 The National Academies of Sciences, Engineering, and Medicine (2019). Vibrant and Healthy Kids: Aligning Science, Practice, and Policy to Advance Health Equity. Washington, DC: The National Academies Press.
- 7 O'Donnell, K.J., & Meaney, M.J. (2020). Epigenetics, development, and psychopathology. Annual Review of Clinical Psychology, 16.
- 8 McEwen, B.S., Gray, J.D., & Nasca, C. (2015). 60 years of neuroendocrinology: Redefining neuroendocrinology: Stress, sex and cognitive and emotional regulation. *Journal of Endocrinology*, 226(2), T67-83.
- 9 Goodwin, R.D. (2011). Association between infection early in life and mental disorders among youth in the community: a crosssectional study. BMC Public Health, 11, 878.
- 10 National Scientific Council on the Developing Child. (2012). The Science of Neglect: The Persistent Absence of Responsive Care Disrupts the Developing Brain: Working Paper No. 12. Retrieved from www.developingchild.harvard.edu.
- 11 National Academies of Sciences, Engineering, and Medicine. (2017). Communities in Action: Pathways to Health Equity. Washington, DC: The National Academies Press.
- 12 Campbell, F., Conti, G., Heckman, J.J., Moon, S.H., Pinto, R., Pungello, E., & Pan, Y. (2014). Early childhood investments substantially boost adult health. *Science*, 343(6178), 1478-1485.
- Heckman, J.J. (2012). The developmental origins of health. *Health Economics*, 21(1): 24-29
- 14 Caspi, A., Houts, R.M., Belsky, D.W., Harrington, H., Hogan, S., Ramrakha, S., ... Moffitt, T. (2016). Childhood forecasting

- of a small segment of the population with large economic burden. *Nature Human Behaviour*, 1, 0005.
- Moffitt, T.E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R.J., Harrington, H.L., ... Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences*, 108(7), 2693–2698.
- 16 National Scientific Council on the Developing Child. (2005/2014). Excessive Stress Disrupts the Architecture of the Developing Brain: Working Paper No. 3. Updated edition. Retrieved from www.developingchild. harvard.edu.
- 17 Rhyu, I.J., Bytheway, J.A., Kohler, S.J., Lange, H., Lee, K.J., Boklewski, J., ... Cameron, J.L., (2010). Effects of aerobic exercise training on cognitive function and cortical vascularity in monkeys. *Neuroscience*, 167(4), 1239-1248.
- 18 Smith, K.J., & Ainslie, P.N. (2017). Regulation of cerebral blood flow and metabolism during exercise. *Experimental Physiology*, 102(11), 1356-1371.
- 19 Cassilhas, R.C., Tufik, S., & de Mello, M.T. (2016). Physical exercise, neuroplasticity, spatial learning and memory. Cellular and Molecular Life Sciences, 73(5), 975-983.
- 20 Lubans, D., Richards, J., Hillman, C., Faulkner, G., Beauchamp, M., Nilsson, M., ... Biddle S. (2016). Physical activity for cognitive and mental health in youth: A systematic review of mechanisms. *Pediatrics*, 138(3), e20161642.
- 21 Erickson, K.I., Voss, M.W., Prakash, R.S., Basak, C., Szabo, A., Chaddock, L., ... Kramer, A.F. (2011). Exercise training increases size of hippocampus and improves memory. Proceedings of the National Academy of Sciences, 108(7), 3017-3022.
- 22 Kullmann, S., Heni, M., Hallschmid, M., Fritsche, A., Preissl, H., & Häring, H.U. (2016). Brain insulin resistance at the crossroads of metabolic and cognitive disorders in humans. *Physiology Reviews*, 96(4), 1169-1209.
- 23 McEwen, B.S., & Akil, H. (2020). Revisiting the stress concept: implications for affective disorders. *The Journal of Neuroscience*, 40(1), 12-21.
- 24 McEwen, B.S. (1998). Protective and damaging effects of stress mediators. New England Journal of Medicine, 338(3), 171-179.
- 25 McEwen & Akil (2020)
- 26 Caspi et al. (2016)
- 27 Heckman (2012)
- 28 Zhang, T.Y., Bagot, R., Parent, C., Nesbitt, C., Bredy, T.W., Caldji, C., ... Meaney, M.J. (2006). Maternal programming of defensive responses through sustained effects on gene expression. *Biological Psychology*, 73(1), 72-89.
- 29 Forde, A.T., Crookes, D.M., Suglia, S.F., & Demmer, R.T. (2019). The weathering hy-

- pothesis as an explanation for racial disparities in health: a systematic review. *Annals of Epidemiology*, 33, 1-18.e3.
- Williams, D.R., & Sternthal, M. (2010). Understanding racial-ethnic disparities in health: Sociological contributions. *Journal* of Health and Social Behavior, 51 Suppl, S15-27.
- 31 Heard-Garris, N. J., Cale, M., Camaj, L., Hamati, M. C., & Dominguez, T. P. (2018). Transmitting trauma: A systematic review of vicarious racism and child health. Social Science & Medicine, 199, 230-240.
- 32 Pachter, L.M., & Coll, C.G. (2009). Racism and child health: a review of the literature and future directions. *Journal of Developmental and Behavioral Pediatrics*, 30(3), 255-263.
- 33 Clark, R., Anderson, N.B., Clark, V.R., & Williams, D.R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist*, 54(10), 805-816.
- 34 National Academies of Sciences, Engineering, and Medicine (2017)
- McEwen, B.S. (2006). Protective and damaging effects of stress mediators: Central role of the brain. *Dialogues in Clinical Neuroscience*, 8(4), 367-381.
- 36 Peña, C.J., Kronman, H.G., Walker, D.M., Cates, H.M., Bagot, R.C., Purushothaman, I., ... Nestler, E.J. (2017). Early life stress confers lifelong stress susceptibility in mice via ventral tegmental area OTX2. Science, 356(6343), 1185-1188.
- Bale, T.L. (2014). Lifetime stress experience: Transgenerational epigenetics and germ cell programming. *Dialogues in Clinical Neuro-science*, 16(3), 297-305.
- 38 Hughes, K., Bellis, M.A., Hardcastle, K.A., Sethi, D., Butchart, A., Mikton, C., ... Dunne, M.P. (2017). The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *Lancet Public Health*, 2(8), e356-e366.
- Morris, G., Berk, M., Maes, M., Carvalho, A.F., & Puri, B.K. (2019). Socioeconomic deprivation, adverse childhood experiences and medical disorders in adulthood: Mechanisms and associations. *Molecular Neurobiology*, 56(8), 5866-5890.
- 40 National Scientific Council on the Developing Child (2005/2014)
- 41 McEwen, B.S. (2013). The brain on stress: Toward an integrative approach to brain, body and behavior. Perspectives on Psychological Science, 8(6), 673-675.
- Miller, A.L., Clifford, C., Sturza, J., Rosenblum, K., Vazquez, D.M., Kaciroti, N., & Lumeng, J.C. (2013). Blunted cortisol response to stress is associated with higher body mass index in low-income preschoolaged children. *Psychoneuroendocrinology*, 38(11), 2611–2617.
- 43 Ouellet-Morin, I., Odgers, C.L., Danese, A.,

- Bowes, L., Shakoor, S., Papadopoulos, A.S., ... Arseneault, L. (2011). Blunted cortisol responses to stress signal social and behavioral problems among maltreated/bullied 12-year-old children. Biological Psychiatry, 70(11), 1016-1023.
- Burke, H.M., Fernald, L.C., Gertler, P.J., & Adler, N.E. (2005). Depressive symptoms are associated with blunted cortisol stress responses in very low-income women. Psychosomatic Medicine, 67(2), 211-216.
- Fisher, P.A., Stoolmiller, M., Gunnar, M.R., & Burraston, B.O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. Psychoneuroendocrinology, 32(8-10), 892-905.
- Dozier, M., Peloso, E., Lewis, E., Laurenceau, J.P., & Levine, S. (2008). Effects of an attachment-based intervention on the cortisol production of infants and toddlers in foster care. Development and Psychopathology, 20(3), 845-859.
- 47 Boyce et al. (2020)
- Zhang, T.Y., & Meaney, M.J. (2010). Epigenetics and the environmental regulation of the genome and its function. Annual Review of Psychology 61, 439-466.
- Jabés, A. & Nelson, C.A. (2015). 20 years after "The Ontogeny of Human Memory: A Cognitive Neuroscience Perspective," where are we? International Journal of Behavioral Development, 39 (4), 293-303.
- McEwen, B.S., Nasca, C., & Gray, J.D.(2015). Stress effects on neuronal structure: hippocampus, amygdala, and prefrontal cortex. Neuropsychopharmacology, 41(1), 3-23.
- Eiland, L., Ramroop, J., Hill, M.N., Manley, J., & McEwen, B.S. (2012). Chronic juvenile stress produces corticolimbic dendritic architectural remodeling and modulates emotional behavior in male and female rats. Psychoneuroendocrinology, 37(1), 39-47.
- Sabatini, M.J., Ebert, P., Lewis, D.A., Levitt, P., Cameron, J.L., & Mirnics, K. (2007). Amygdala gene expression correlates of social behavior in monkeys experiencing maternal separation. The Journal of Neuroscience, 27, 3295-3304.
- Herzog, J.I., & Schmahl. C. (2018). Adverse childhood experiences and the consequences on neurobiological, psychosocial, and somatic conditions across the lifespan. Frontiers in Psychiatry, 4(9), 420.
- Duncan, N.W., Hayes, D.J., Wiebking, C., Tiret, B., Pietruska, K., Chen, D.Q., ... Northoff, G. (2015). Negative childhood experiences alter a prefrontal-insular-motor cortical network in healthy adults: A preliminary multimodal rsfMRI-fMRI-MRSdMRI study. Human Brain Mapping, 36(11), 4622-4637.
- Callaghan, B.L., & Tottenham, N. (2016). The neuro-environmental loop of plasticity: A cross-species analysis of parental effects on emotion circuitry development following

- typical and adverse caregiving. Neuropsychopharmacology, 41(1), 163-176.
- Picard, M., McManus, M.J., Gray, J.D., Nasca, C., Moffat, C., Kopinski, P.K., ... Wallace, D.C. (2015). Mitochondrial functions modulate neuroendocrine, metabolic, inflammatory, and transcriptional responses to acute psychological stress. Proceedings of the National Academy of Sciences, 112(48), E6614-E6623.
- Dantzer, R. (2018). Neuroimmune interactions: From the brain to the immune system and vice versa. Physiological Reviews, 98(1),
- 58 Halfon, N., Larson, K., Lu, M., Tullis, E., & Russ, S. (2014). Lifecourse health development: past, present and future. Maternal and Child Health Journal. 18(2), 344-365.
- McEwen & Akil (2020)
- 60 Cameron, J.L., Eagleson, K.L., Fox, N.A., Hensch, T.K., & Levitt, P. (2017). Social origins of developmental risk for mental and physical illness. The Journal of Neuroscience, 37(45), 10783-1079.
- Reid, B.M., Coe, C.L., Doyle, C.M., Sheerar, D., Slukvina, A., Donzella, B., & Gunnar, M.R. (2019). Persistent skewing of the T-cell profile in adolescents adopted internationally from institutional care. Brain, Behavior, and Immunity, 77, 168-177.
- Reichman, N.E., Corman, H., Noonan, K., & Jiménez, M.E. (2018). Infant health and future childhood adversity. Maternal and Child Health Journal, 22(3), 318-26.
- 63 Martinez, F.D., & Guerra, S. (2018). Early origins of asthma. Role of microbial dysbiosis and metabolic dysfunction. American Journal of Respiratory and Critical Care Medicine, 197(5), 573-579.
- Wright, R.J. (2011). Epidemiology of stress and asthma: From contrasting communities and fragile families to epigenetics. Immunology and Allergy Clinics of North America, 31(1), 19-39.
- Martinez, F.D. (2016). Early-life origins of chronic obstructive pulmonary disease. New England Journal of Medicine, 375, 871-878.
- Louisias, M., & Phipatanakul, W. (2017). Managing Asthma in Low-Income, Underrepresented Minority, and Other Disadvantaged Pediatric Populations: Closing the Gap. Current Allergy and Asthma Reports, 17(10), 68.
- Wright, RJ, Subramanian SV. Advancing a multilevel framework for epidemiologic research on asthma disparities. Chest, 132(5 Suppl):757S-769S.
- von Mutius, E., & Vercelli, D. (2010). Farm living: effects on childhood asthma and allergy. Nature Reviews Immunology, 10, 861-868.
- 69 Riedler, J., Braun-Fahrländer, C., Eder, W., Schreuer, M., Waser, M., Maisch, S., ... & ALEX Study Team (2001). Exposure to farming in early life and development of

- asthma and allergy: a cross-sectional survey. Lancet, 358(9288), 1129-1133.
- Casas, R., Castro-Barquero, S., Estruch, R., & Sacanella, E. (2018). Nutrition and Cardiovascular Health. International journal of molecular sciences, 19(12), 3988.
- Danese, A., Dove, R., Belsky, D.W., Henchy, J., Williams, B., Ambler, A., & Arseneault, L. (2014). Leptin deficiency in maltreated children. Translational Psychiatry, 4(9), e446.
- Danese, A., & Tan, M. (2014). Childhood maltreatment and obesity: Systematic review and meta-analysis. Molecular Psychiatry, 19(5), 544-554.
- Evans, G.W., Wachs, T.D. (Eds.) (2010). Chaos and Its Influence on Children's Development: An Ecological Perspective. Washington, DC: American Psychological Association.
- 74 Chen, E., Miller, G.E., Yu, T., & Brody, G.H. (2018). Unsupportive parenting moderates the effects of family psychosocial intervention on metabolic syndrome in African American youth. International Journal of Obesity (Lond), 42(4), 634-640
- Hackett, R.A., & Steptoe, A. (2017). Type 2 diabetes mellitus and psychological stressa modifiable risk factor. Nature Reviews Endocrinology, 13(9), 547-560.
- 76 Kullmann, et al. (2016)
- Dallman, M.F. (2010). Stress-induced obesity and the emotional nervous system. Trends in Endocrinology & Metabolism, 21(3), 159-165.
- Suglia, S.F., Campo, R.A., Brown, A.G.M., Stoney, C., Boyce, C.A.,... Watamura, S.E. (2020). Social determinants of cardiovascular health: Early life adversity as a contributor to disparities in cardiovascular diseases. The Journal of Pediatrics, 219, 267-273.
- Suglia, S.F., Koenen, K.C., Boynton-Jarrett, R., Chan, P.S., Clark, C.J., Danese, A., ... Zachariah, J.P. (2018). Childhood and adolescent adversity and cardiometabolic outcomes: A scientific statement from the American Heart Association. Circulation, 137(5), e15-e28.
- Waters, H., & Graf, M. (2018). The costs of chronic disease in the U.S., Milken Institute and Medical Expenditure Panel Survey. Retrieved from https://milkeninstitute.org/ sites/default/files/reports-pdf/ChronicDiseases-HighRes-FINAL.pdf
- U.S. Department of Health and Human Services, Agency for Healthcare Research and Quality (AHRQ). Medical Expenditure Panel Survey. Retrieved from http://meps. ahrq.gov/mepsweb/
- Greenberg, P.E., Fournier, A-A., Sisitsky, T., Pike, C.T., Kessler, R.C. (2005/2010). The economic burden of adults with major depressive disorder in the United States. Retrieved from <a href="https://www.psychiatrist.com/">https://www.psychiatrist.com/</a> jcp/article/pages/2015/v76n02/v76n0204. aspx.

- 83 Centers for Disease Control and Prevention. (2020). National Diabetes Statistics Report, 2020. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.
- 84 Tabák, A.G., Herder, C., Rathmann, W., Brunner, E J., & Kivimäki, M. (2012). Prediabetes: a high-risk state for diabetes development. *Lancet*, *379*(9833), 2279–2290.
- 85 Dieleman, J.L., Baral, R., & Birger, M. (2016). US Spending on Personal Health Care and Public Health, 1996-2013. *JAMA*, 316(24), 2627-2646.
- 86 Gisterå, A., Hansson, G.K. (2017). The immunology of atherosclerosis. *Nature Reviews Nephrology*, 13(6), 368-380.
- 87 Barker, D.J. (1995). Fetal origins of coronary heart disease. *British Medical Journal*, 311(6998), 171–174.
- 88 Lascar, N., Brown, J., Pattison, H., Barnett, A.H., Bailey, C.J., & Bellary, S. (2018). Type 2 diabetes in adolescents and young adults. The Lancet. Diabetes & Endocrinology, 6(1), 69-80.
- 89 Kautzky-Willer, A., Harreiter, J., & Pacini, G. (2016). Sex and Gender Differences in Risk, Pathophysiology and Complications of Type 2 Diabetes Mellitus. *Endocrine Reviews*, 37(3), 278-316.
- 90 de Luca, C., & Olefsky, J.M. (2008). Inflammation and insulin resistance. FEBS Letters, 582(1), 97–105.
- 91 Gold, S.M., Dziobek, I., Sweat, V., Tirsi, A., Rogers, K., Bruehl, H., ... Convit, A. (2007). Hippocampal damage and memory impairments as possible early brain complications of type 2 diabetes. *Diabetologia*, 50(4), 711-719
- 92 Yau, P.L., Castro, M.G., Tagani, A., Tsui, W.H., & Convit, A. (2012). Obesity and metabolic syndrome and functional and structural brain impairments in adolescence. *Pediatrics*, 130(4), e856-864.
- 93 Rasgon, N.L., & McEwen, B.S.(2016). Insulin resistance—a missing link no more. Molecular Psychiatry, 21(12), 1648-52.
- 94 National Institute of Mental Health, Information Resource Center. (2019). Prevalence of Major Depressive Episode Among Adults. Retrieved from https://www.nimh.nih.gov/health/statistics/major-depression.shtml#part\_155033.
- 95 American Psychiatric Association. (2017). What Is Depression? Retrieved from https://www.psychiatry.org/patientsfamilies/depression/what-is-depression.
- 96 Sheline, Y. I., Liston, C., & McEwen, B. S. (2019). Parsing the hippocampus in depression: Chronic stress, hippocampal volume, and major depressive disorder. *Biological psychiatry*, 85(6), 436–438.
- 97 Chen, Y., & Baram, T.Z. (2016). Toward understanding how early-life stress reprograms cognitive and emotional brain networks. Neuropsychopharmacology, 41(1), 197-206.
- 98 Kessler, R.C., Davis, C.G., & Kendler, K.S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine*, 27(5), 1101-1119.

- 99 McEwen, B.S. (2017). Integrative medicine: Breaking down silos of knowledge and practice an epigenetic approach. *Metabolism*, 69S, S21-S29.
- 100 Kendler, K.S. (1995). Genetic epidemiology in psychiatry; Taking both genes and environment seriously. Archives of General Psychiatry, 52(11), 895-899.
- 101 Sullivan, P.F., Neale, M.C., & Kendler, K.S. (2000). Genetic epidemiology of major depression: review and meta-analysis. *The American Journal of Psychiatry* 157(10), 1552–1562.
- 102 Labaka, A., Goñi-Balentziaga, O., Lebeña, A., & Pérez-Tejada, J. (2018). Biological sex diferences in depression: A systematic review. Biological Research For Nursing, 20(4), 383-392.
- 103 Hodes, G.E., Walker, D.M., Labonté, B., Nestler, E.J., & Russo, S.J. (2017). Understanding the epigenetic basis of sex differences in depression. *Journal of Neuroscience Research*, 95(1-2), 692-702.
- 104 Van Loo, H.M., Aggen, S.H., Gardner, C.O., & Kendler, K.S. (2018). Sex similarities and differences in risk factors for recurrence of major depression. *Psychological Medicine*, 48(10), 1685-1693.
- 105 Weaver, A., Himle, J.A., Taylor, R.J., Matusko, N.N., & Abelson, J.M. (2015). Urban vs rural residence and the prevalence of depression and mood disorder among African American women and non-Hispanic white women. *JAMA Psychiatry*, 72(6), 576-583.
- 106 Bailey, R.K., Mokonogho, J., & Kumar, A. (2019). Racial and ethnic differences in depression: current perspectives. *Neuropsychiatric Disease and Treatment*, 15, 603–609.
- 107 Georgetown University Health Policy Institute. (2019). Maternal Depression Costs Society Billions Each Year, New Model Finds. Retrieved from https://ccf.georgetown. edu/2019/05/31/maternal-depression-costssociety-billions-each-year-new-modelfinds/.
- 108 Smith, M.V., & Lincoln, A.K. (2011). Integrating social epidemiology into public health research and practice for maternal depression. *American Journal of Public Health*, 101(6), 990–994.
- 109 Milaneschi, Y., Lamers, F., Berk, M., & Penninx, B. (2020). Depression heterogeneity and its biological underpinnings: Toward immunometabolic depression. *Biological Psychiatry*, S0006-3223(20)30048-2.
- 110 Danese, A., Moffitt, T.E., Pariante, C.M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. Archives of General Psychiatry, 65(6), 409–415.
- 111 Knop, M.R., Geng, T.-T., Gorny, A.W., Ding, R., Li, C., Ley, S.H., & Huang, T. (2018). Birth weight and risk of type 2 diabetes mellitus, cardiovascular disease, and hypertension in adults: A meta-analysis of 7 646 267 participants from 135 studies. *Journal of the American Heart Association*, 7(23), e008870.
- 112 Olvera Alvarez, H.A., Kubzansky, L.D.,

- Campen, M.J., & Slavich, G.M. (2018). Early life stress, air pollution, inflammation, and disease: An integrative review and immunologic model of social-environmental adversity and lifespan health. *Neuroscience & Biobehavioral Reviews*, 92, 226-242.
- 113 Danese, A., Lewis, S.J. (2017). Psychoneuroimmunology of early-life stress: The hidden qounds of childhood trauma? *Neuropsychopharmacology*, 42(1), 99–114.
- 114 National Scientific Council on the Developing Child. (2007). The Timing and Quality of Early Experiences Combine to Shape Brain Architecture: Working Paper #5. Retrieved from https://developingchild.harvard.edu/.
- 115 Bavelier, D., Levi, D.M., Li, R.W., Dan, Y., & Hensch, T.K. (2010). Removing brakes on adult brain plasticity: from molecular to behavioral interventions. *The Journal of Neuroscience*, 30(45), 14964-14971.
- 116 Zigler, E., & Valentine, J. (Eds.). (1979). Project Head Start: A Legacy of the War on Poverty. The Free Press (US).
- 117 Center on the Developing Child at Harvard University. (2016). From Best Practices to Breakthrough Impacts: A Science-Based Approach to Building a More Promising Future for Young Children and Families. Retrieved from https://developingchild.harvard.edu.
- 118 National Research Council and Institute of Medicine Committee on Integrating the Science of Early Childhood Development, Shonkoff, J. P., & Phillips, D. A. (Eds.). (2000). From Neurons to Neighborhoods: The Science of Early Childhood Development. National Academies Press (US).
- 119 Center on the Developing Child at Harvard University (2016).
- 120 Center on the Developing Child at Harvard University. (2017). Three Principles to Improve Outcomes for Children and Families. Retrieved from: www.developingchild. harvard.edu.
- 121 National Scientific Council on the Developing Child (2012)
- 122 Collins, C., Asante-Muhammed, D., Hoxie, J., & Nieves, E. (2017). The road to zero wealth: How the racial wealth divide is hollowing out America's middle class. Washington, DC: Prosperity Now and Institute for Policy Studies.
- 123 Weir, K. (2016). *Policing in Black & White*. Monitor on Psychology, 47(11). Retrieved from <a href="http://www.apa.org/monitor/2016/12/cover-policing">http://www.apa.org/monitor/2016/12/cover-policing</a>.
- 124 Center on the Developing Child at Harvard University. (2016). Building Core Capabilities for Life: The Science Behind the Skills Adults Need to Succeed in Parenting and in the Workplace. Retrieved from <a href="https://developingchild.harvard.edu">https://developingchild.harvard.edu</a>.
- 125 Shonkoff, J.P., Boyce, W.T., Levitt, P., Martinez, F.D., & McEwen, B.S. (2020). More Than Just the Brain (I): 21st-Century Biology and the Future of Pediatric Primary Care. Under review.
- 126 Hirai, A.H., Kogan, M.D., Kandasamy, V., Reuland, C., & Bethell, C. (2018). Prevalence and variation of developmental screening and surveillance in early child-

- hood. JAMA Pediatrics, 172(9), 857-66. 127 Peacock-Chambers, E., Ivy, K., & Bair-Merritt, M. (2017). Primary care interventions for early childhood development: A systematic review. Pediatrics, 140(6), e20171661.
- 128 Piotrowski, C.C., Talavera, G.A., & Mayer, J.A. (2009). Healthy Steps: A systematic review of a preventive practice-based model of pediatric care. Journal of Developmental and Behavioral Pediatrics, 30(1), 91-103.
- 129 Dworkin, P. H., & Sood, A. B. (2016). A Population Health Approach to System Transformation for Children's Healthy Development. Child and adolescent psychiatric clinics of North America, 25(2), 307-317.
- 130 Trent, M., Dooley, D.G., & Dougé, J. (2019). The impact of racism on child and adolescent health. Pediatrics, 144(2), e20191765.
- 131 Commission on Social Determinants of Health. (2008) Closing the gap in a generation: health equity through action on the social determinants of health. Final report of the Commission on Social Determinants of Health. Geneva, World Health Organiza-
- 132 Heron, M. Deaths: Leading causes for 2017. National Vital Statistics Reports, 68(6). Retrieved from https://www.cdc.gov/heartdisease/facts.htm.
- 133 Rasmussen, L.J.H., Moffitt, T.E., Arseneault, L., Denise, A., Eugen-Olsen, J., Fisher, H.L. ... Caspi, A. (2020). Association of adverse experiences and exposure to violence in childhood and adolescence with inflammatory burden in young people, JAMA Pediatrics, 174(1), 38-47.
- 134 U.S. Department of Health and Human Services, Agency for Healthcare Research and Quality (AHRQ). (2018). Medical Expenditure Panel Survey. Retrieved from http://meps.ahrq.gov/mepsweb/.
- 135 Calkins, K., & Devaskar, S.U. (2011). Fetal origins of adult disease. Current Problems in Pediatric and Adolescent Health Care, 41(6), 158-176.
- 136 Roseboom, T.J., van der Meulen, J.H., Ravelli, A.C., Osmond, C., Barker, D.J., & Bleker, O.P. (2001). Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. Molecular and Cellular Endocrinology, 185(1-2), 93-98.
- 137 Ravelli, G.-P., Stein, Z.A., & Susser, M.W. (1976). Obesity in young men after famine exposure in utero and early infancy. New England Journal of Medicine, 295(7), 349-
- 138 Eriksson, J.G., Forsen, T.J., Osmond, C., & Barker, D.J. (2003). Pathways of infant and childhood growth that lead to type 2 diabetes. Diabetes Care, 26(11), 3006-3010.
- 139 Friedman, J.E. (2018). Developmental programming of obesity and diabetes in mouse, monkey, and man in 2018: Where are we headed? Diabetes, 67(11), 2137-2151.
- 140 Edlow, A.G. (2017). Maternal obesity and neurodevelopmental and psychiatric disorders in offspring. Prenatal Diagnosis, 37(1),
- 141 Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R.A., ... Sultan,

- S.E. (2004). Developmental plasticity and human health. Nature, 430, 419-421.
- American Diabetes Association. (2018). Economic Costs of Diabetes in the U.S. in 2017. Diabetes Care, 41(5), 917-928.
- 143 Nusslock, R., & Miller, G.E. (2016). Earlylife adversity and physical and emotional health across the lifespan: A neuroimmune network hypothesis. Biological Psychiatry, 80(1), 23-32.
- 144 Gilman, S.E., Kawachi, I., Fitzmaurice, G.M., & Buka, L. (2003). Socio-economic status, family disruption and residential stability in childhood: relation to onset, recurrence and remission of major depression. Psychological Medicine, 33(8), 1341-1355.
- 145 Dunn, E., Soare, T., Zhu, Y., Simpkin, A.J., Suderman, M.J., Klengel, T., ... Relton, C.L. (2019). Sensitive periods for the effect of childhood adversity on DNA methylation: Results from a prospective, longitudinal study. Biological Psychiatry, 85(10), 838-849.
- 146 Greenberg, et al. (2005/2010)
- Gluckman, P. D., Hanson, M.A., Cooper, C., & Thornburg, K.L. (2008). Effect of in utero and early-life conditions on adult health and disease. The New England Journal of Medicine, 359(1), 61-73.

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## **Place Matters:**

## The Environment We Create Shapes the Foundations of Healthy Development

**WORKING PAPER 16** 



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#### THE ISSUE:

## The Physical Environments Where Children Live Affect Their Development and Health

We all experience a continuous stream of influences from the physical and social environments in which we live, beginning before birth and continuing throughout our lives. These include a wide range of conditions in the places where children live, grow, play, and learn that get "under the skin" and affect the developing brain and other biological systems—including the immune and metabolic systems—with potential effects in childhood and well into the adult years. 1 Beyond the critically important impacts of caregiver-child relationships on early childhood development, the places where people live affect what they are exposed to, which then affects maturing biological systems—positively or negatively. In short, place matters.

Scientists categorize the physical environment in at least two ways, both of which are shaped by human actions, including intentional decisions around policies that shape the environment in which we live. One category—the natural environment—includes the quality and temperature of our air, the purity and availability of our water supply, and the ways that climate change affects the prevalence and magnitude of natural disasters like floods, hurricanes, and wildfires. Another—the built environment includes the residences in which families live; the density of surrounding buildings; the types of local businesses (and whether they offer job opportunities, access to nutritious food, etc.); the availability of green spaces; the upkeep of roads, bridges, and sidewalks in the neighborhood; and the transportation that people can access to get to where they need to go.2

The qualities of the conditions in which people live are not evenly or randomly distributed. They are shaped by and deeply

rooted in public policies and social history. Extensive research demonstrates how zoning regulations, real estate and banking practices, and government actions—both through historic discrimination and current practices—have discriminated against minoritized racial and ethnic groups. These influences, past and present, continue to shape the natural and built environments where Black and Indigenous individuals, along with other people of color (BIPOC), live today.

The qualities of the conditions in which people live are not evenly or randomly distributed. They are shaped by and deeply rooted in public policies and social history.

For example, policies described as "redlining"—a federally backed program that for nearly 40 years denied mortgage loans and other financial services for residents of areas that were marked on maps as "hazardous" for investment based on residents' race or ethnicity—resulted in neighborhoods that remain predominantly populated by Black residents and other people of color. This segregation has led to unequal access to wealth (through lack of access to high-paying jobs and favorable mortgages), lack of access to high-quality health care and schools, and unequal access to reliable transportation. These previously redlined neighborhoods often lack resources to oppose the building of highways, manufacturing plants, and toxic waste disposal sites in or near their communities. As a result, today, these racially segregated communities are far more likely than predominantly white neighborhoods to experience increased exposure to high levels of air pollution, toxic chemicals, excessive noise, and

higher temperatures, while also having less access to healthy foods, highquality health care facilities, safe areas to play or exercise, and green spaces.<sup>3,4,5</sup> Families struggling with the hardships of intergenerational poverty and with limited political power in rural areas are also more likely to live in close proximity to contaminated groundwater and be exposed to toxicants (i.e., artificial, humanmade toxic products such as pesticides or industrial waste) that can have serious consequences for pregnancy outcomes and the subsequent health of their children. 6,7,8

Understanding the powerful effects that natural and built environments have on the early foundations of health and development calls for increased attention to important influences that fall well beyond the traditional boundaries of the early childhood field.

> In 2004, the National Scientific Council on the Developing Child described the effects of early life experiences on the developing brain in its first Working Paper, Young Children Develop in an Environment of Relationships.9 Over the ensuing two decades, this science-based concept has helped make the case for safe, stable, and nurturing relationships as the "active ingredient" in how environments can positively influence the architecture of the developing brain. More recently, as research on the early origins of health and illness has advanced, so has our understanding of how early experiences affect multiple biological systems in

the body (e.g., immune, metabolic, and respiratory) and how those systems interact with and shape each other as well as the brain. 10,11 But this is not the whole picture. External exposures from the natural and built environments also affect the development of biological systems inside the body and interact with the more personal influences of adult-child relationships in a deeply interconnected way.

The implications of this rapidly growing science are clear. Understanding the powerful effects that natural and built environments have on the early foundations of health and development calls for increased attention to important influences that fall well beyond the traditional boundaries of the early childhood field. This demands the incorporation of a more intentional early childhood perspective within the current concerns of urban planning, rural development, environmental protection, climate change, and anti-discrimination policies, among others. Ensuring "fairness of place"—that vital conditions for wellbeing are available to all children, not just some—requires that a broader range of policy domains work together to redress racist and other discriminatory policies to achieve greater equity. Supporting healthy child development is still about caregiverchild relationships, and it's also about communities, businesses, and governments working together to assure a supportive and healthy environment for all young children—with particular attention to natural and built environments that fall far short of that goal.12

## What Science Tells Us

The conditions of a place can have positive or negative influences on child health and development. Positive influences, beginning in pregnancy and continuing throughout childhood,

include access to nutritious food, clean air and drinking water, safe green space in which to play, reliable transportation, and a home environment free of lead and other heavy metals. Negative influences

include polluted air and water, extreme temperatures, a lack of safe green spaces, high rates of crime and violence, excessive environmental noise that can disrupt normal sleep patterns, lack of access to affordable nutritious food, and a home environment containing toxicants from asbestos, lead, or secondhand smoke.13

An environment that provides many positive influences is more likely to support healthy development, and an environment that imposes many negative influences is more likely to result in a higher prevalence of disease and impairment. For example, access to safe green spaces—such as parks, playgrounds, and recreation areas—is associated with better physical and mental health, lower stress, and lower rates of obesity and type 2 diabetes, among many other benefits.<sup>14</sup> Access to safe green space during pregnancy is associated with decreased risk for low birth weight, which is a known risk factor for a range of health conditions across the life course.15 More frequent exposure to green spaces during childhood is related to lower risk of both obesity and neurodevelopmental problems such as inattentiveness. 16 Based on available evidence, it is reasonable to hypothesize that these benefits can be explained by higher levels of physical activity, calming effects of exposure to nature, mitigation of extreme heat, and reduction of air and noise pollution.

As our knowledge of the health effects of green space grows, the unequal distribution of these spaces demands greater attention. In many cities across the United States, neighborhoods with higher percentages of residents of color, as well as people with lower levels of education and income, have less access to green space and experience higher average temperatures than neighborhoods with higher percentages of white and higher-income residents.<sup>17,18</sup> Moreover, the geography of these differences closely mirrors the boundaries created by legalized, discriminatory zoning and real estate investment practices (described above) that began almost a century ago

and have played a major role in creating the racially segregated neighborhoods and subsequent unequal exposures to adverse environmental conditions that continue to this day. 19 Current zoning practices that place restrictions on minimum lot sizes, building height, and construction of multifamily homes perpetuate unequal types and quality of housing across neighborhoods. These historically discriminatory practices, as well as their modern-day policy counterparts, result in neighborhoods with fewer positive conditions and more harmful environmental influences, and thereby contribute to persistent racial disparities in health, such as higher rates of obesity and diabetes in Black populations compared to white.<sup>20</sup>

Many factors contribute to the early foundations of health and development. That said, abundant research evidence shows that as the number of adverse exposures increases, it becomes less likely that any individual will "weather the storm" and avoid experiencing some negative effects.21 As the demand for deeper understanding of neighborhood influences on child well-being has increased, researchers across disciplines have become more precise about quantifying both positive and negative environmental conditions and their impacts. One of the most prominent examples, the Childhood Opportunity Index (COI), provides a comprehensive tool for evaluating assets and risk factors at the neighborhood level, based on data collected from 72,000 census tracts in the 100 largest metropolitan areas in the United States.<sup>22</sup>

TABLE 1 Neighborhood indicators in the Child Opportunity Index 2.0

Education	Health and Environment	Social and Economic
Early childhood education  • Early childhood education centers  • High-quality early childhood education centers  • Early childhood education enrollment  Elementary education  • Third grade reading proficiency  • Third grade math proficiency  Secondary and postsecondary education  • High school graduation rate  • Advanced Placement course enrollment  • College enrollment in nearby institutions  Educational and social resources  • School poverty  • Teacher experience  • Adult educational attainment	<ul> <li>Healthy environments</li> <li>Access to healthy food</li> <li>Access to green space</li> <li>Walkability</li> <li>Housing vacancy rate</li> <li>Toxic exposures</li> <li>Hazardous waste dump sites</li> <li>Industrial pollutants in air, water or soil</li> <li>Airborne microparticles</li> <li>Ozone concentration</li> <li>Extreme heat exposure</li> <li>Health resources</li> <li>Health insurance coverage</li> </ul>	Economic opportunities  • Employment rate  • Commute duration  Economic and social resources  • Poverty rate*  • Public assistance rate*  • Homeownership rate*  • High-skill employment*  • Median household income*  • Single-headed households

<sup>\*</sup>These five indicators are combined into an economic resource index.

The COI considers the types of resources and conditions in neighborhoods where children live, and the corresponding access to opportunities—or lack thereof that can support healthy development. The 29 elements quantified by the COI include proximity to assets like educational resources (including high-quality early care and education), green spaces, employment opportunities, and healthy foods, as well as exposure to risk factors like hazardous waste, air pollution, and

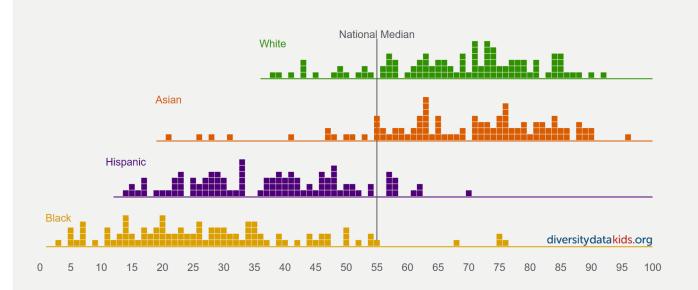
extreme heat.23 Analyses of COI data show significant geographical differences across the United States, with New England and the Great Plains states containing metro areas with the highest scores, while the Central Valley of California and Southern states have metro areas with some of the lowest opportunity scores in the country.

Within these regions, the COI lays bare dramatic differences between neighborhoods populated predominantly by white residents and those that are home

to mostly Black and Hispanic residents (see sidebar). Black and Hispanic children across the US are more than seven and five times more likely, respectively, to live in "very low opportunity" neighborhoods compared to white children.<sup>24</sup> Analysis of COI data shows that children are highly segregated by race/ethnicity, and opportunities are significantly less

available to those who identify as Black or Hispanic compared to those who identify as white or Asian. In the Milwaukee metro area, for example, the typical white child lives in a neighborhood with a Child Opportunity Score of 85 (out of 100), while the typical Black child lives in a neighborhood with a score of only 6.

## Underinvestment Leads to Wide Racial Disparities in Access to Opportunity



The figure above shows the distribution of Child Opportunity Scores across the 100 largest US metro areas by race/ethnicity. The green bars at the top show wide variation in opportunity scores for white children across metro areas, but the distribution is generally above the national median. In other words, in the vast majority of metro areas, the typical white child enjoys neighborhood opportunity higher than the national median. The distribution for Asian children is similar. In contrast, the typical Hispanic or Black child lives in a neighborhood with an opportunity score that is well below the national median. In fact, for the 100 largest metro areas combined, the average Child Opportunity Score is 73 for white children and 72 for Asian children, in sharp contrast to 33 for Hispanic children and 24 for Black children.<sup>25</sup>

Environmental exposures early in life can cause lasting changes in developing biological systems. The brain and other biological systems in the body (e.g., immune, metabolic, and respiratory), as well as the microbiome (i.e., bacteria that develop in the gut and play an important role in health and illness), each have periods when they are most sensitive to environmental influences. During prenatal development, billions of cells are produced that become specialized for different organ systems or functions each establishing unique properties that allow them to function as part of the brain, lungs, immune system, or as hormoneproducing cells, among many other types. In the immune system, for example, these specialized cells are deployed throughout the body and develop molecular "memories" that are essential elements of the body's defense against infection throughout childhood and adolescence.<sup>26</sup>

Ensuring the environments that surround pregnant people are safe, supportive, and free of toxicants is a critical investment in the future health and well-being of all children.

> Ensuring the environments that surround pregnant people are safe, supportive, and free of toxicants is a critical investment in the future health and wellbeing of all children. Exposure to a subset of specific infections or toxic substances, as well as poor nutrition (e.g., scarcity or overabundance of calories), during the prenatal period can have lifelong impacts on developing biological systems and even prime these systems to be more susceptible to similar stressors later in life. For example, undernutrition during critical periods of fetal development may cause lasting changes in metabolic and endocrine regulation that increase the likelihood of obesity and cardiovascular disease later in life. Some toxic substances absorbed during pregnancy can enter the placenta and affect its function, as well as cross into the fetus and disrupt its development directly.

Significant adversity or trauma may also speed up the opening and closing of critical periods in the development of specific brain circuits.<sup>27</sup> This can have negative consequences for both physical and mental health by contributing to earlier onset of puberty and the development of anxiety.<sup>28</sup>

The effects of early exposure to air pollution on the developing brain and respiratory system have been studied extensively and are well understood. Significant air pollution comes from the burning of fossil fuels, including emissions from cars, as well as poorly ventilated wood-burning stoves, and forest fires. Airborne pollutants can be absorbed in a variety of ways and cause problems in specific developing organs as well as entire systems.29 The nature and severity of these effects vary according to when they occur over the course of development. For example, exposure to air pollution prenatally, when the lungs and immune system are especially sensitive to environmental influences, 30 is associated with lower lung volume in early childhood<sup>31</sup> and decreased lung function in the preschool years.<sup>32</sup> Exposure to air pollution in the prenatal period is also associated with increased rates of restricted growth in utero, prematurity, and low birth weight in full-term infants. 33,34,35,36 Children who are exposed to higher rates of outdoor air pollution during the first year after birth may have diminished functional lung capacity as teenagers.<sup>37</sup> Similar exposures throughout early childhood increase the risk of developing pediatric leukemia, elevated blood pressure, and asthma or chronic obstructive pulmonary disease in adolescence or early adulthood. 38,39,40 Because Black children are exposed to air pollution more often than white children, it is not surprising that they are twice as likely to have asthma and four times as likely to die from it.41

There is also evidence that some types of air pollutants can activate the body's stress response by stimulating the hypothalamic-pituitary-adrenal (HPA) axis, triggering the release of stress hormones

such as cortisol.<sup>42</sup> Many developing biological systems, including the brain, are more sensitive to the effects of excessive amounts of stress hormones than more mature systems, particularly in the prenatal period and early years after birth. When the stress response is chronically elevated, it can produce what is known as a "toxic stress response," creating structural irregularities in the brain and negative effects on cognition and mental health.43 as well as broader wear and tear effects across multiple organ systems over time.44 Moreover, the full range of health outcomes that are affected by early environmental influences such as air pollution may not be apparent until much later in life.

Racism influences multiple dimensions of the natural and built environments that affect the foundations of child development and lifelong well-being. In the first decade of the 21st century, the Human Genome Project (an international collaboration that generated the first sequence of the full set of human DNA) demonstrated once and for all that there are no distinct biological boundaries that indicate where one racial category begins and another ends. Racial distinctions, as we know them, are inventions created by societies—and there are no validated genetic criteria for differentiating these categories. 45 Given this scientific consensus, when we study racial and ethnic disparities in health status across groups (as defined by census data or other means of self-identification). these comparisons reflect variation in lived experiences within and across generations, not underlying genetic differences. Stated simply, although race is not an objective biological categorization, the experience of racism gets into the developing body, with significant biological consequences that can begin in the prenatal period.

Many people think of racism as overt bigotry or personally experienced discrimination in the context of everyday social interactions, including implicit bias, microaggressions, and harassment. The

full manifestations of its effects, however, are embedded in a much wider range of conditions, experiences, and exposures that are experienced by families of color with young children. 46 Cultural racism, for example, is experienced as a pervasive ideology that is reflected in the language, symbols, media, and assumptions of the larger society that values whiteness as the desirable standard. Stereotype threat, which occurs when an individual's awareness of a negative stereotype results in worry that their behavior could reinforce that stereotype about their culture, and the internalized racism that it produces, are often invisible to those who do not experience them first-hand.<sup>47</sup>

Structural (or systemic) racism, which is reflected in both the natural and built environments, includes multiple manifestations of how political, economic, and social inequities become deeply embedded in where people live particularly but not exclusively in racially segregated communities—and how systems and institutions operate in ways that provide an advantage to some racial/ ethnic groups and perpetuate an unfair disadvantage to others. These biases have been built deeply into an array of public policies and institutional practices that have been either prescribed explicitly by law (e.g., Jim Crow segregation) or perpetuated implicitly by customary practices (e.g., racial disparities in the criminal justice system as illustrated by unequal sentencing patterns). Many adverse effects of systemic racism have deep historical roots whose impacts continue to the present day, and many present-day policies continue to perpetuate these inequities and their ongoing effects. These include the placement of hazardous waste sites close to communities of color (see box below) and the construction of the US interstate highway system beginning in the 1950s, which located urban routes largely through communities of color and neighborhoods that were previously redlined.48 Current policies that perpetuate inequities through

ongoing discrimination in the housing market include requirements for minimum lot sizes and restrictions on the construction of multi-family homes.49

The cumulative effects of systemic racism, compounded by cultural racism and the everyday personal indignities and threats of individualized discrimination, contribute to a complex mix of physical, social, and economic conditions and experiences that impose substantial hardships on BIPOC families raising young children.<sup>50</sup> In the natural environment, structural racism leads to segregated communities in which minoritized children are exposed to more excessive heat and toxicants (e.g., air pollution,51 industrial waste,<sup>52</sup> insecticides in the case of migrant farm workers<sup>53</sup>) and have less access to clean drinking water<sup>54,55</sup> and violence-free green space. 56,57 In the built environment, structural racism affects the type and quality of residential housing and leads to diminished access to nutritious foods, high-quality health services and child care, educational resources, and economic opportunity.

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> The causal mechanisms that explain how the effects of racism can be built into the body and lead to disparities in the development and health of young children continue to be the focus of extensive research. Like other types of early life adversity that trigger excessive activation of the stress response system, the stresses of racism can lead to biological disruptions that increase the risk for negative impacts on learning, behavior, and both physical and mental health. Some scientists have documented how different forms of adversity affect different

parts of the brain;58 many have focused on the common effects of excessive stress activation inside the body, independent of its causes.<sup>59</sup> Although the biological disruptions caused by racism may be due to its effects on the stress response system, disparities in health outcomes associated with systemic racism can also be explained by profoundly disproportionate exposures to environmental toxicants such as air pollution and contaminated drinking water. Further research will shed greater light on the complex interactions among multiple sources of adversity and resilience that affect the well-being of children and the adults who care for them, particularly in the prenatal and early childhood periods, when developing biological systems are most susceptible to environmental influences.

The timing of environmental experiences and exposures can influence both short- and long-term effects. 60 As noted earlier, humans differ in their sensitivity to influences from the environment at various points in the life course. The sensitivity of the brain and other biological systems is typically greater in the prenatal period than in young children; young children are more susceptible to most adverse exposures than adolescents: and adolescents are more vulnerable to many exposures than adults. 61 Immature biological systems in an embryo or fetus develop at an extremely fast pace, and their development is powerfully shaped by interactions with the environment around them. These systems read conditions in the womb as predictors of what they will encounter after birth and adapt accordingly. This makes these developing systems more susceptible to positive and negative environmental influences, as compared to when they have matured and stabilized.62

Beginning immediately after birth, the protective function of the placenta and uterus is replaced by responsive caregiving, but the external environment also affects babies and toddlers directly through the

air they breathe, the water they drink, and the sound level and temperature of the conditions in which they sleep—all of which can either promote or disrupt the development of their brain circuits, the maturation of their immune system, and the regulation of their metabolism.63

Although the first "place" that affects development directly is the intrauterine environment during pregnancy, the nature and extent of these effects may not be fully apparent until years or decades later.64 Inadequate or excessive nutrition, unmanageable levels of stress, extreme heat, and chemical exposures (e.g., lead) are particularly dangerous during the prenatal period. 65 Over- or under-nutrition is associated with greater risk of obesity, hypertension, and heart disease in adulthood.66 These and other environmental influences (e.g., specific infections, tobacco smoke, pesticides) during pregnancy are also connected to very low or very high birth weights, which can have implications across the lifespan, including greater risk for cardiovascular disease, type 2 diabetes, and mental health conditions.67

One example of how the timing of exposures affects their impact is the effects of lead, an extensively studied toxicant. This heavy metal can be absorbed at any age by the gastrointestinal system through ingestion, in lesser amounts through the respiratory system by inhalation, or in small amounts through skin absorption—and there is no safe level of lead in the blood. A high-profile instance of widespread lead exposure through contaminated water in Flint, Michigan, was identified in 2014, when nearly a quarter of the children in that city showed increased blood lead levels<sup>68</sup>—double the previous rate<sup>69</sup>—in the months following Flint's switch in public water suppliers. Young children, fetuses, and pregnant individuals absorb lead through the gastrointestinal system at substantially higher rates than the general population. As a result, exposure during these sensitive periods can result

in a range of negative impacts on health and learning (including increased risk of preterm birth, miscarriage, decreased fetal growth, learning and behavioral difficulties later in childhood, and increased blood pressure in adulthood<sup>70</sup>), while exposure to the same level of lead in an adult is much less likely to have significant effects.71

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Lead exposure provides a striking example of the effects of discriminatory housing and economic policies on the built environment, which in turn affects child outcomes. Structural racism, through redlining and neighborhood disinvestment, has resulted in children of color living, on average, in older homes that are more likely to contain lead in pipes, paint, and the surrounding soil. Regulatory policies that require landlords of rental properties to abate lead are inconsistently enforced, and when the safeguards around these policies break down, residents are left with limited options for lead abatement. Moreover, these safeguards fail more often in neighborhoods where families living in poverty have fewer resources to put toward lead abatement in their homes.72 In Flint, long-term impacts cannot yet be measured, but research to date has documented a 15% increase in babies born at low birth weight to women who were pregnant when the crisis began and a nearly 20% increase in low birth weight among children born to Black mothers in the area.73

The consequences of exposure to environmental tobacco smoke (ETS) during pregnancy also illustrate increased sensitivity in the prenatal period. Extensive studies have demonstrated that prenatal exposure to ETS—even when the expectant parent does not smoke leads to higher risk of low birth weight,

birth defects, and stillbirth.74 While there is abundant evidence that adults also experience negative health effects from tobacco exposure, those effects (e.g., elevated blood pressure and increased risk of lung cancer and heart disease) are different from those observed early in life. As noted above, air pollution during the prenatal period can directly affect the developing lungs and immune system, increase the risk of low birth weight or neurodevelopmental outcomes like autism, and be a trigger for asthma in susceptible children during childhood (see below). Exposure to air pollution in adulthood does not lead to the same outcomes.75,76

Individuals respond differently to the physical environment, but there are clear patterns of risk that can inform universal action. Even within the same home, or in the face of similar experiences or exposures in a broader context, individual children react differently to both adversity and support. Some are highly sensitive to changes in their environment while others "go with the flow" in difficult situations. Scientists refer to this concept of individual differences as heterogeneity. As a core principle of 21st century biology, it is explained by extensive evidence that all aspects of development and health over the life course are determined by complex interactions among genes, environments, and developmental timing ("GxExT").77

In the case of asthma, each child is born with a unique genetic profile that reflects differential susceptibility to the disease—but whether and how those genetic instructions are carried out is affected by experiences and exposures.78 A fetus in utero or a developing child after birth may be exposed to a range of environmental triggers for asthma (including air pollution—as noted above but also dust, chemicals, viruses/bacteria, vermin, and stress).79 How that fetus or baby is affected by these triggers, however, depends on the magnitude and frequency of the exposures, how specific exposures interact with individual genetic variation,

and whether they occur during critical periods of development. All three factors interacting with each other will determine whether a child is likely to develop asthma, how severe the symptoms might be, and whether the condition becomes chronic.80

Population-level rates of asthma, on the other hand, paint a clear picture of identified risk factors in the broad environment that can be addressed to lower its prevalence across an entire community. For example, multiple studies have shown that higher rates of asthma exist in neighborhoods with more pollution and lower-quality housing.81,82 Programs such as the Community Asthma Initiative in Boston, which provides expanded access to better health care and addresses sources of environmental triggers that are most prevalent in neighborhoods with high rates of this illness, have been shown to reduce rates of asthma across the community. Such programs do not eliminate asthma entirely, because of the complex interaction of factors described above, but by reducing its environmental causes and improving medical treatment, they have been effective in significantly reducing the human and economic burdens of this costly disease at a population level.83,84

Looking at how environmental threats to health play out across a range of contexts and diseases reveals common underlying principles that underscore the way toxic exposures, genetic variation in susceptibility, and developmental timing interact to shape outcomes. In the case of Toms River Township (formerly Dover Township), a predominantly white, middle class, suburban region in New Jersey, public health officials investigated a significant increase in the incidence of childhood cancers and found a link to hazardous chemicals in the local drinking water and soil from a nearby manufacturing site.85 In another example from the Appalachian region of West Virginia known as Chemical Valley, the release of a chemical known as MCHM polluted the local drinking water, groundwater, and soil, leading to

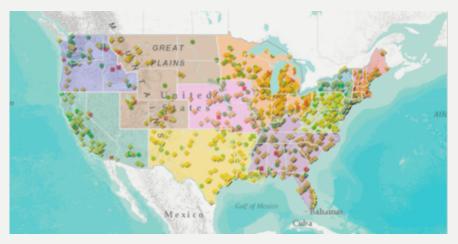
increases in preterm births and low birth weights in full-term newborns, many of whom required complex medical care.86

Without minimizing the serious (and fully preventable) consequences of these toxic exposures, not all children who drank the affected water in the Toms River area developed cancer, and not all fetuses exposed to contaminated drinking water in Chemical Valley were born prematurely. Differences in individual genetic makeup, levels of exposure, and developmental timing explain most, if not all, of the reasons for the variable health effects. Regardless of

this predictable variation in populationlevel risk, a broad public health approach combined with a tailored response to address differential needs is most likely to protect the health and development of all children in a community. In the case of lead, this approach can include housing policies that ensure high-quality pipes and clean water for everyone, mitigation efforts in neighborhoods most likely to have high concentrations of lead paint and soil, and frequent surveillance combined with individualized monitoring and treatment as needed for children with detectable blood levels.

## **Human-Made Toxicants Affect Childhood Development**

While the impacts of adverse environmental influences fall disproportionately on individuals living in poverty, people of color, and other marginalized groups as a result of historical and current discriminatory policies, all communities are potentially susceptible to the health effects of unfavorable environmental conditions. The Toms River Township of Ocean County, New Jersey, is a suburban, predominantly white, middle-class region.



EPA Map of Superfund National Priorities List. Explore this interactive map and additional details here.

Between 1979 and 1999, 102 children under the age of 19 who lived in that area were diagnosed with cancer, an incidence rate that is one-third greater than expected. Rates of brain cancer, leukemia, and other nervous system cancers were particularly high.<sup>87</sup> The state of New Jersey subsequently conducted a study that linked prenatal exposure to two specific sources of contamination in the township's water and air to an increased risk of leukemia in girls.<sup>88</sup> The identification of these contaminants resulted in the closure of two business sites, payment of fines for criminal penalties by the companies involved, financial settlement with families whose children developed cancer, an expanded treatment system for the water supply, and the development of a new water sampling and analysis method that allows for measurement of radioactivity. Despite these efforts, cleanup of the site and disputes about ways to restore its natural resources remain ongoing.<sup>89,90</sup> Environmental hazards can disrupt developing biological systems in a variety of ways beginning very early in life, leading to a range of adverse effects on physical and mental health. News headlines from Wilmington, Massachusetts; Camp Lejeune, North Carolina;

Flint, Michigan; and Jackson, Mississippi, have captured ways that residents have been exposed to toxicants as dramatic examples of preventable tragedies, but they are far from the only ones. As of fall 2022, there were more than 1,300 locations in the US officially designated by the Environmental Protection Agency as National Priority Superfund sites (i.e., locations that contain high levels of hazardous material contamination that require long-term cleanup funded by federal legislation), with another 43 awaiting this formal designation. 91 While contaminated sites can be found in every state and in both rural and urban areas, as well as on hundreds of former military installations, 92 they are not evenly distributed. In 2015, a national analysis of hazardous waste sites found that toxic facilities are usually placed in locations where residents lack social, economic, or political power—and these are disproportionately areas where people of color and people living in poverty reside.93 The Superfund program, instituted by federal legislation in 1980, is one example of a policy response to understanding our shared responsibility for—and benefit from—cleaning up environmental toxicants. Yet, just like exposure to toxicants, our response to these conditions is uneven across groups. For example, in Flint, Michigan, where residents are predominantly Black, it took 79 lawsuits<sup>94</sup> and two years of community activism after several major outbreaks of disease due to contaminated water to initiate a public response that eventually brought lead levels below the toxic range.

## Implications for New Directions in Policy

All children, regardless of where they grow up, should be able to live in an environment that supports their healthy development. And, all communities have natural and built dimensions of their environment that have been constructed and designed through decisions made over time. Just as these dimensions of the environment have been designed over time, they can be re-designed to support healthy development.

All children, regardless of where they grow up, should be able to live in an environment that supports their healthy development.

> Every environment is infused with a combination of positive and negative influences on health and development, but levels of exposure to hazards and access to opportunity are not distributed

equally. Equalizing such environmental opportunities so that all children can grow up in neighborhoods free of toxicants and rich in access to high-quality education and health care will require confronting the causes and consequences of systemic racism, intergenerational poverty, and other structural inequities that lead to preventable disparities in child development and lifelong physical and mental health.

When we respond as a society to a devastating hurricane, wildfire, flood, or blizzard, we target greater support to the communities that have been most severely affected. Similarly, directing greater attention to community conditions where they present the greatest threats to the wellbeing of young children reflects our shared commitment to a healthy and sustainable society. Securing the opportunity for all children to develop in an environment that

helps them to thrive requires attention to universal needs and investment in places that face the greatest hardships and obstacles. Deeply embedded inequities where people live disproportionately undermine the life prospects of children of color and children living in poverty, beginning before they are born. There is an urgent need to address these inequities and provide all children with the opportunity to reach their full potential.

The benefits of high-quality health care, child care, and early education are well-documented. Yet these childfocused programs are situated in a broader environment of risk and protection that also requires focused attention in order to achieve the promise of population-wide improvements in educational achievement, health, and well-being. All sectors of the early childhood ecosystem, including policymakers, service providers, advocates, and private philanthropists, must direct increased attention to and investment in the prevention and reduction of adverse environmental conditions and exposures that get built into the developing body early in life. Strategic investments at the population and community levels, beginning in the earliest periods of development, represent a critical yet currently under-addressed dimension of science-informed early childhood policy that demands fresh thinking.

To succeed in this mission, we must broaden the list of policy domains that are viewed as affecting the foundations of early childhood development and lifelong physical and mental health. Prominent examples include environmental protection, climate change policies and mitigations, housing, zoning, urban planning, economic development, criminal legal reforms, and anti-discrimination policies, among others. All of these areas

are interdependent. Each policy domain must focus on its capacity to dismantle structural factors that lead to the disproportionate exposure of minoritized children to adverse environmental influences. Re-examining policies and their associated systems through an antiracist, early childhood lens will advance our ability to connect the dots among:

- strengthening community assets that support healthy development;
- preventing, reducing, and/or mitigating environmental conditions that threaten human well-being, with particular attention to the most affected communities; and
- understanding how both assets and threats are built into the body, beginning prenatally and in the early childhood period, and result in either a strong or weak foundation for all the learning, behavior, and health that are necessary for a thriving and sustainable society.

These challenges will not be addressed by working within the current boundaries of early childhood policy and practice. The future of science-informed investment in young children and their families and the path to greater impacts at scale requires a coordinated strategy that builds on the current ecosystem of childand family-focused supports and moves "upstream" to incorporate a broader range of policy domains that influence the natural and built environments that affect families raising young children. Through such a coordinated strategy, we can create a society that supports the health and development of all children, one where equal access to opportunity assures a sustainable future for us all.

For more specific policy implications, ideas, and examples, visit developingchild.harvard.edu in 2023 and beyond.

## References

- National Scientific Council on the Developing Child. Connecting the brain to the rest of the body: early childhood development and lifelong health are deeply intertwined: Working paper no. 15. 2020. https:// developingchild.harvard.edu/ resources/connecting-the-brain-tothe-rest-of-the-body-early-childhooddevelopment-and-lifelong-healthare-deeply-intertwined/
- U.S. Environmental Protection Agency. Basic Information About the Built Environment. Updated March 10, 2022. Accessed May 12, 2022. https://www.epa.gov/smm/ basic-information-about-builtenvironment
- Sistrunk C, Tolbert N, Sanchez-Pino MD, et al. Impact of federal, state, and local housing policies on disparities in cardiovascular disease in Black/African American men and women: from policy to pathways to biology. Front Cardiovasc Med. 2022;18(9):756734. doi:10.3389/ fcvm.2022.756734
- Santaliz CA, Lee A, Teteh D, Madak EZ, Treviño L. Endocrinedisrupting chemicals and breast cancer: disparities in exposure and importance of research inclusivity. Endocrinology. 2022;163(5):bgac034. doi:10.1210/endocr/bgac034
- Abdi FM, Andrews K. Redlining has left many communities of color exposed to lead. Child Trends. February 13, 2018. https://www. childtrends.org/blog/redlining-leftmany-communities-color-exposed-
- Collins MB, Munoz, I, JaJa J. Linking 'toxic outliers' to environmental justice communities. Environ. Res. Lett. 2016;11:015004. doi:10.1088/1748-9326/11/1/015004
- Gochfeld M, Burger J. Disproportionate exposures in environmental justice and other populations: the importance of outliers. Am J Public Health. 2011;101 Suppl 1(Suppl 1):S53-S63. doi:10.2105/ AJPH.2011.300121
- Nardone A, Casey JA, Morello-Frosch R, Mujahid M, Balmes JR, Thakur N. Associations between historical residential redlining and current age-adjusted rates of emergency

- department visits due to asthma across eight cities in California: an ecological study. Lancet Planet Health. 2020;4(1):e24-e31. doi:10.1016/S2542-5196(19)30241-4
- National Scientific Council on the Developing Child. Young children develop in an environment of relationships: working paper no. 1. 2004. https://developingchild. harvard.edu/resources/wpl/
- Boyce WT, Levitt P, Martinez FD, McEwen BS, Shonkoff JP. Genes, environments, and time: the biology of adversity and resilience. Pediatrics. 2021;147(2):e20201651. doi:10.1542/ peds.2020-1651
- National Scientific Council on the Developing Child. 2020.
- Shonkoff J. Re-envisioning early childhood policy and practice in a world of striking inequality and uncertainty. Center on the Developing Child at Harvard University. January 2022. https:// developingchild.harvard.edu/reenvisioning-ecd/
- 13 Center on the Developing Child at Harvard University. The foundations of lifelong health are built in early childhood. 2010. https://developingchild.harvard. edu/resources/the-foundations-of-<u>lifelong-health-are-built-in-early-</u> childhood/
- De la Fuente F, Saldías MA, Cubillos C, et al. Green space exposure association with type 2 diabetes mellitus, physical activity, and obesity: a systematic review. Int J Environ Res Public Health. 2020;18(1):97. Published 2020 Dec 25. doi:10.3390/ijerph18010097
- Islam MZ, Johnston J, Sly PD. 15 Green space and early childhood development: a systematic review. Rev Environ Health. 2020;35(2):189-200. doi:10.1515/reveh-2019-0046
- Islam MZ, Johnston J, Sly PD. 2020.
- Hoffman JS, Shandas V, Pendleton N. The effects of historical housing policies on resident exposure to intraurban heat: a study of 108 US urban areas. Climate. 2020;8(1):12. https:// doi.org/10.3390/cli8010012
- Nesbitt L, Meitner MJ, Girling

- C, Sheppard SRJ, Lu Y. Who has access to urban vegetation? A spatial analysis of distributional green equity in 10 US cities. Landscape and Urban Planning. 2019;181:51-79.
- Roberts JD, Dickinson KL, Hendricks MD, Jennings V. "I can't breathe": examining the legacy of American racism on determinants of health and the ongoing pursuit of environmental justice. Curr Environ Health Rep. 2022;9(2):211-227. doi:10.1007/s40572-022-00343-x
- 20 De la Fuente F, et al. 2020.
- Hamby S, Elm JHL, Howell KH, Merrick MT. Recognizing the cumulative burden of childhood adversities transforms science and practice for trauma and resilience. Am Psychol. 2021;76(2):230-242. doi:10.1037/amp0000763
- Acevedo-Garcia D, Noelke C, McArdle N, et al. The geography of child opportunity: why neighborhoods matter for equity. First findings from the Child Opportunity Index 2.0. January 21, 2020. Accessed July 20, 2022. https://www. diversitydatakids.org/sites/default/ files/file/ddk\_the-geography-of-childopportunity\_2020v2\_0.pdf
- Acevedo-Garcia D, McArdle N, Hardy EF, et al. The child opportunity index: improving collaboration between community development and public health. Health Aff (Millwood). 2014:33(11):1948-1957. doi:10.1377/ hlthaff.2014.0679
- Acevedo-Garcia D, et al. 2020.
- Acevedo-Garcia D. et al. 2020.
- Dietert RR, Etzel RA, Chen D, et al. Workshop to identify critical windows of exposure for children's health: immune and respiratory systems work group summary. Environ Health Perspect. 2000;108 Suppl 3(Suppl 3):483-490. doi:10.1289/ ehp.00108s3483
- Tooley UA, Bassett DS, Mackey AP. Environmental influences on the pace of brain development. Nat Rev Neurosci. 2021;22(6):372-384. doi:10.1038/s41583-021-00457-5
- Callaghan BL, Richardson R. The effect of adverse rearing environments on persistent

- memories in young rats: removing the brakes on infant fear memories. Transl Psychiatry. 2012;2(7):e138. doi:10.1038/tp.2012.65
- Brumberg HL, Karr CJ, et al. Ambient air pollution: health hazards to children. Pediatrics. 2021;147(6):e2021051484. doi:10.1542/ peds.2021-051484.
- Krusche J, Basse S, Schaub B. Role of early life immune regulation in asthma development. Semin Immunopathol. 2020;42(1):29-42. doi:10.1007/s00281-019-00774-z
- Mudway IS, Dundas I, Wood HE, et al. Impact of London's low emission zone on air quality and children's respiratory health: a sequential annual cross-sectional study. Lancet Public Health. 2019;4(1):e28-e40. doi:10.1016/S2468-2667(18)30202-0
- Morales E, Garcia-Esteban R, de la Cruz OA, et al. Intrauterine and early postnatal exposure to outdoor air pollution and lung function at preschool age. Thorax. 2015;70(1):64-73. doi:10.1136/thoraxjnl-2014-205413
- 33 Brumberg HL, et al. 2021.
- Stieb DM, Chen L, Eshoul M, Judek S. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. Environ Res. 2012;117:100-111. doi: 10.1016/j.envres.2012.05.007
- Srám RJ, Binková B, Dejmek J, Bobak M. Ambient air pollution and pregnancy outcomes: a review of the literature. Environ Health Perspect. 2005;113(4):375-382. doi:10.1289/ ehp.6362
- Dadvand P, Parker J, Bell ML, et al. Maternal exposure to particulate air pollution and term birth weight: a multi-country evaluation of effect and heterogeneity. Environ Health Perspect. 2013;121(3):267-373. doi: 10.1289/ehp.1205575
- Schultz ES, Hallberg J, Bellander T, et al. Early-life exposure to trafficrelated air pollution and lung function in adolescence. Am J Respir Crit Care Med. 2016;193(2):171-177. doi:10.1164/rccm.201505-0928OC
- Boothe VL, Boehmer TK, Wendel AM, Yip FY. Residential traffic exposure and childhood

- leukemia: a systematic review and meta-analysis. Am J Prev Med. 2014;46(4):413-422. doi:10.1016/j. amepre.2013.11.004
- 39 Kelishadi R, Poursafa P, Keramatian K. Overweight, air and noise pollution: universal risk factors for pediatric pre-hypertension. J Res Med Sci. 2011;16(9):1234-1250.
- Guarnieri M, Balmes JR. Outdoor air pollution and asthma. Lancet. 2014;383(9928):1581-1592. doi:10.1016/ S0140-6736(14)60617-6
- Holsey CN, Collins P, Zahran H. Disparities in asthma care, management, and education among children with asthma. Clin Pulm Med. 2013;20(4):172-177. doi:10.1097/ CPM.0b013e3182991146
- 42 Thomson EM. Air pollution, stress, and allostatic load: linking systemic and central nervous system impacts. J Alzheimers Dis. 2019;69(3):597-614. doi:10.3233/JAD-190015
- 43 National Scientific Council on the Developing Child. Excessive stress disrupts the architecture of the developing brain: working paper no. 3. Updated 2014. https:// developingchild.harvard.edu/ resources/wp3/
- McEwen BS. Stress: Homeostasis, 44 rheostasis, reactive scope, allostasis and allostatic load. 2017. doi:10.1016/ B978-0-12-809324-5.02867-4
- 45 Yudell M, Roberts D, DeSalle R, Tishkoff S. SCIENCE AND SOCIETY. Taking race out of human genetics. Science. 2016;351(6273):564-565. doi:10.1126/science.aac4951
- Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. J Behav Med. 2009;32(1):20-47. doi:10.1007/s10865-008-9185-0
- Shonkoff JP, Slopen N, Williams DR. Early childhood adversity, toxic stress, and the impacts of racism on the foundations of health. Annu Rev Public Health. 2021;42:115-134. doi:10.1146/annurevpublhealth-090419-101940
- Archer, D. White men's roads through black men's homes: advancing racial equity through highway reconstruction. Vanderbilt

- Law Review 73:5:1259-1330.
- Racial Residential Segregation in Greater Boston. Harvard Chan-NIEHS Center for Environmental Health. Updated July 18, 2022. Accessed January 25, 2023. https:// storymaps.arcgis.com/stories/ bd15a5eb9eae49cda09bfa7368272f89
- 50 Center on the Developing Child at Harvard University. Moving upstream: confronting racism to open up children's potential. 2021. https://developingchild.harvard. edu/resources/moving-upstreamconfronting-racism-to-open-upchildrens-potential/
- Chakraborty J, Zandbergen PA. Children at risk: measuring racial/ ethnic disparities in potential exposure to air pollution at school and home. J Epidemiol Community Health. 2007;61(12):1074-1079. doi:10.1136/jech.2006.054130
- 52 Mohai P & Saha R. Which came first, people or pollution? Assessing the disparate siting and post-siting demographic change hypotheses of environmental injustice. Environ. Res. Lett. 2015;10:115008. doi:10.1088/1748-9326/10/11/11500
- Mills PK, Dodge J, Yang R. Cancer in migrant and seasonal hired farm workers. J Agromedicine. 2009;14(2):185-191. doi:10.1080/10599240902824034
- Roberts JD, Dickinson KL, Hendricks MD, Jennings V. "I can't breathe": examining the legacy of American racism on determinants of health and the ongoing pursuit of environmental justice. Curr Environ Health Rep. 2022 Jun;9(2):211-227. doi:10.1007/s40572-022-00343-x
- 55 Masten SJ, Davies SH, McElmurry SP. Flint water crisis: what happened and why? J Am Water Works Assoc. 2016;108(12):22-34. doi:10.5942/ jawwa.2016.108.0195
- Locke DH, Hall B, Grove JM, et al. Residential housing segregation and urban tree canopy in 37 US cities. npj Urban Sustain. 2021;1(15):1-9. doi:10.1038/s42949-021-00022-0
- Rowland-Shea J, Doshi S, Edberg S, Fanger R. The nature gap: confronting racial and economic disparities in the destruction and

- protection of nature in America. Center for American Progress. 2020.
- McLaughlin KA, Sheridan MA. Beyond cumulative risk: a dimensional approach to childhood adversity. Curr Dir Psychol Sci. 2016;25(4):239-245. doi:10.1177/0963721416655883
- Smith KE, Pollak SD. Rethinking concepts and categories for understanding the neurodevelopmental effects of childhood adversity. Perspect Psychol Sci. 2021;16(1):67-93. doi:10.1177/1745691620920725
- Boyce WT, et al. 2021.
- Boyce WT, et al. 2021.
- Fleming TP, Watkins AJ, Velazquez MA, et al. Origins of lifetime health around the time of conception: causes and consequences. Lancet. 2018;391(10132):1842-1852. doi:10.1016/ S0140-6736(18)30312-X
- 63 Boyce WT, et al. 2021.
- Dunkerton S & Aiken C. Impact of the intrauterine environment on future reproductive and metabolic health. The Obstetrician & Gynaecologist. 2022;24(2):93-100. doi:10.1111/ tog.12797
- 65 Center on the Developing Child at Harvard University. 2010.
- Portella AK, Silveira PP. Neurobehavioral determinants of nutritional security in fetal growthrestricted individuals. Ann NY Acad Sci. 2014;1331:15-33. doi:10.1111/ nyas.12390
- Fernandez-Twinn DS, Hjort L, Novakovic B, Ozanne SE, Saffery R. Intrauterine programming of obesity and type 2 diabetes. Diabetologia. 2019;62(10):1789-1801. doi:10.1007/ s00125-019-4951-9
- Ezell JM, Bhardwaj S, Chase EC. Child lead screening behaviors and health outcomes following the Flint water crisis [published correction appears in J Racial Ethn Health Disparities. 2022 Mar 10.]. J Racial Ethn Health Disparities. 2023:10(1):418-426. doi:10.1007/s40615-022-01233-6
- DeWitt RD. Pediatric lead exposure and the water crisis in Flint, Michigan. JAAPA. 2017;30(2):43-46. doi:10.1097/01.JAA.0000511794.60054.
- United States. Agency for Toxic Substances and Disease Registry. Toxicological profile for lead. August

- 2020. doi:10.15620/cdc:95222
- Yeter D, Banks EC, Aschner M. Disparity in risk factor severity for early childhood blood lead among predominantly African-American Black children: The 1999 to 2010 US NHANES. Int J Environ Res Public Health. 2020;17(5):1552. Published 2020 Feb 28. doi:10.3390/ijerph17051552
- 72 Muller C, Sampson RJ, & Winter AS. Environmental inequality: The social causes and consequences of lead exposure. Annual Review of Sociology. 2018;44(1), 263-282. doi:10.1146/ annurev-soc-073117-041222
- Wang R, Chen X, Li X. Something in the pipe: the Flint water crisis and health at birth. J Popul Econ. 2022:35:1723-1749. doi:10.1007/s00148-021-00876-9
- 74 Salmasi G, Grady R, Jones J, McDonald SD; Knowledge Synthesis Group. Environmental tobacco smoke exposure and perinatal outcomes: a systematic review and meta-analyses. Acta Obstet Gynecol Scand. 2010;89(4):423-441. doi:10.3109/00016340903505748
- Volk HE, Lurmann F, Penfold 75 B, Hertz-Picciotto I, McConnell R. Traffic-related air pollution, particulate matter, and autism. JAMA Psychiatry. 2013;70(1):71-77. doi:10.1001/jamapsychiatry.2013.266
- von Ehrenstein OS, Aralis H, Cockburn M, Ritz B. In utero exposure to toxic air pollutants and risk of childhood autism. Epidemiology. 2014;25(6):851-858. doi:10.1097/EDE.0000000000000150
- Boyce WT, et al. 2021.
- National Scientific Council on the Developing Child. Early experiences can alter gene expression and affect long-term development: working paper no. 10. 2010. https:// developingchild.harvard.edu/ resources/early-experiences-canalter-gene-expression-and-affectlong-term-development/
- National Scientific Council on the Developing Child. 2020.
- 80 Martinez FD. Asthma as a developmental disorder. Annu Rev Dev Psychol. 2021;3:229-48. doi:10.1146/ annurev-devpsych-030221-020950
- Lemire E, Samuels EA, Wang W, Haber A. Unequal housing conditions and code enforcement contribute to asthma disparities in Boston, Massachusetts. Health Affairs.

- 2022;41(4): 563-572. doi:10.1377/ hlthaff.2021.01403
- 82 Alexander D & Currie J. Is it who you are or where you live? Residential segregation and racial gaps in childhood asthma. NBER Working Paper 23622. National Bureau of Economic Research. July 2017. https:// www.nber.org/papers/w23622
- 83 Woods ER, Bhaumik U, Sommer SJ, et al. Community asthma initiative: evaluation of a quality improvement program for comprehensive asthma care. Pediatrics. 2012;129(3):465-472. doi:10.1542/peds.2010-3472
- Bhaumik U, Walker SP, Sommer SJ, et al. Social return on investment from an asthma community-based care management intervention program. American Public Health Association Annual Meeting. Denver, CO. November 8, 2010.
- State of New Jersey Department of Public Health. Toms River Township childhood cancer investigation. Accessed October 4, 2022. https:// www.state.nj.us/health/ceohs/ environmental-occupational/ hazardous-waste-sites/ocean/ dovertwp.shtml
- Parsons E & Diamond E. Dirty the waters: mothers' experience of a chemical disaster in West Virginia, USA. WIT Transactions on the Built Environment. 2019;190:79-90. doi:10.2495/DMAN190071
- Mansnerus L. Community; Dover Township's cancer cluster. The New York Times. February 7, 1999. Accessed May 18, 2022. https://www. nytimes.com/1999/02/07/nyregion/ community-dover-township-scancer-cluster.html
- Toms River Township Childhood Cancer Investigation. State of New Jersey Department of Health. Accessed May 18, 2022. https:// www.state.nj.us/health/ceohs/ environmental-occupational/ hazardous-waste-sites/ocean/ dovertwp.shtml
- Childhood cancer incidence update: a review and analysis of cancer registry data, 2001-2005. U.S. Department of Health and Human Services Public Health Service, Agency for Toxic Substances and Disease Registry, Division of Health Assessment and Consultation, 2008.
- NJ Families Blast Deal Over Polluted Toms River Site After Child Cancer Epidemic. NBC10 Philadelphia.

- Accessed February 1, 2023. https:// www.nbcphiladelphia.com/news/ local/toms-river-families-blast-dealover-polluted-site/3483258/
- Superfund: National Priorities List (NPL). United States Environmental Protection Agency. Accessed October 5, 2022. <a href="https://www.epa.gov/">https://www.epa.gov/</a> superfund/superfund-nationalpriorities-list-npl
- Reuben SH. Reducing Environmental Cancer Risk: What We Can Do Now. National Cancer Institute, National Institutes of Health, U.S. Department of Health And Human Services. DIANE Publishing; 2010.
- Mohai P & Saha R. 2015.
- AG looks to settle Flint suits; Worthy joins criminal probe. Associated Press. February 21, 2019. Accessed October 5, 2022. https://apnews.com/article/8e6 45ecb0acf42bebeebb3595007c934

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