

## Q&A

### **Neuroscience, Molecular Biology, and the Childhood Roots of Health Disparities: Building a New Framework for Health Promotion and Disease Prevention**

Shonkoff, Boyce & McEwen (*JAMA*, June 3, 2009)

*Abstract: The origins of many adult diseases can be traced to negative experiences early in life, so confronting the causes of adversity before and shortly after birth may be a promising way to improve adult health and reduce premature deaths.*

#### **Q: What diseases have their origins in early childhood?**

A: A variety of studies have shown that early childhood adverse experiences dramatically increase the risk, later in life, of diabetes, hypertension, cardiovascular disease, stroke, and certain cancers, as well as depression, anxiety disorders, and substance abuse.

#### **Q: What early childhood experiences lead to the increased risk of these diseases?**

A: Exposure to psychologically and physically stressful events, such as chronic neglect, family chaos, violence, mental illness and substance abuse, as well as exposure to toxins, prenatal drug and alcohol exposure, and malnutrition. Conditions such as these can either accumulate, leading to a “weathering” effect on the body, or, if they occur during sensitive periods in early brain development, can become embedded in the body, to be expressed years, or even decades, later.

#### **Q: How does early childhood stress increase the risk of disease?**

A: Not all stress is bad. “Positive stress,” from such experiences as learning a difficult new skill or meeting new people, is healthy and growth-promoting. It teaches our stress response systems how to activate and return to normal. “Tolerable stress” is caused by more serious events, such as the loss of a loved one, a natural disaster, or a serious illness or injury. With the buffering support of caring adults, a child’s stress response system can be brought back to normal and lasting damage (such as post-traumatic stress disorder) is prevented. But “toxic stress” refers to strong, frequent, and/or prolonged activation of the body’s stress response system due to extreme poverty, abuse, neglect, parental mental illness or substance abuse, and exposure to violence. Without the protective support of adults, this extreme, sustained activation of the stress response system disrupts brain architecture, affects other organ systems, and establishes a lower threshold for stress-response activation that persists throughout life. These effects can trigger a range of stress-related diseases.

#### **Q: What does this have to do with government policies and programs?**

A: Strategies for health promotion and disease prevention, currently focused on immunizations, early identification and treatment, and guidance toward health-promoting behaviors, can be effective, but also have serious limitations: They rely on changing behavior as individuals grow older, they must overcome biological vulnerabilities established early in life, and they shift responsibility onto individuals and away from the

conditions that shaped them in the first place. Instead, policies and programs could attack adult diseases at their roots by reducing the sources of toxic stress in early childhood, improving the quality and availability of early childhood programs, and focusing the child welfare system on improved child development, not just child safety.

**Q: How widespread is toxic stress among children?**

A: Some of the most common potential causes of toxic stress in young children include neglect and abuse, which occurs in 7.5 % of US children age 2-5; parental substance abuse, which occurs in 9.8 % of US households with children under age 5; serious post-partum depression, which occurs in 13% of new mothers; and continuing depression beyond the newborn period, which has been reported to affect 40-50 % of low-income mothers. Other possible precipitants include extreme poverty, emotional abuse, family violence, and other forms of parental mental illness. When there is strong, stable support from extended family and friends, other caregivers, and community services, these circumstances can be prevented from precipitating stress at levels that are toxic to brain development—and in other cases, early intervention targeting the specific sources of the stress must be provided for both parents and children.

**Q: Why do some children experience adversity and grow up healthy, while others do not?**

A: The trajectories of any individual child’s development are the result of a complex interaction between genes and experiences. Certain genes may make individuals more or less vulnerable to the effects of stress or other influences, but one’s experiences—particularly during critical and sensitive periods of brain development—determine whether or how those genes are expressed. There is no such thing as genetic “inevitability”—to the extent that we can control those experiences, we can affect how genetic differences are expressed.

**Q: Are there any treatments that can help, or does the window of opportunity to reverse the effects of early adverse experiences close at some time?**

A: It’s never too late to remediate—it just gets harder, and in extreme cases, it may not be possible to remediate fully. The brain keeps learning and adapting throughout life, but its ability to change decreases with age—and if damage is incurred during critical periods of brain and biological systems development, it may not be possible to reverse. That said, studies do show that more intensive and responsive caregiving can remediate the effects of early stress and neglect. There is some evidence from the burgeoning field of epigenetics that caregiving traits can, in fact, be transmitted across generations—so providing responsive caregiving to a child who has initially experienced neglect can result in benefits not only for that child, but for the future offspring of that child as well. *For a broader perspective on public health solutions, see the commentary by James Mercy and Janet Saul of the Centers for Disease Control and Prevention, “Creating a Healthier Future Through Early Interventions,” also in this issue of JAMA.*

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