



Brain Development: Conception to Age 3

Decades of research show that the environment of a child's earliest years can have effects that last a lifetime. Thanks to recent advances in technology, we have a clearer understanding of how these effects are related to early brain development. Neuroscientists can now identify patterns in brain activity that are associated with various types of negative early experiences.¹

Although the dangers of early stress, poverty, neglect and maltreatment have long been recognized, we can now 'see' their effects using brain scanning technology. Although scientists do not yet understand exactly how experiences affect development, dramatic advances continue to be made, and brain research continues to enhance education and intervention efforts.

The organization of a child's brain is affected by early experiences.

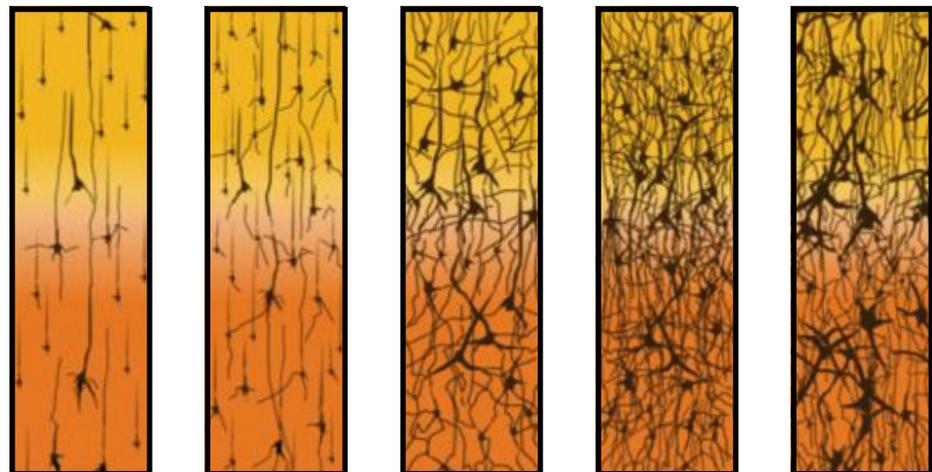
Specialized brain cells called neurons send and receive information by forming connections with one another. Although a newborn's brain already has about all of the neurons it will ever have, it continues to grow at an amazing rate. It doubles in size in the first year, and by age three it reaches 80 percent of its adult volume.²⁻⁴

Even more importantly, connections are formed at a faster rate during these years than at any other time. In fact, the brain creates many more connections than it needs: at age two or three, the brain has up to twice as many connections as it will have in adulthood (FIGURE 1). These surplus connections are gradually eliminated throughout childhood and adolescence, a process sometimes referred to as blooming and pruning.⁵

The excess connections produced by a child's brain in the first three years make the brain especially responsive to external input. During this period, the brain can "capture" experience more efficiently than it will be able to later, when the pruning of unused connections is underway.⁵

FIGURE 1:
Synapse Density
Over Time

Source: Corel, J.L.
The postnatal
development of the
human cerebral cortex.
Cambridge, MA:
Harvard University
Press, 1975.



Newborn

1 Month

9 Months

2 Years

Adult

Genetic and environmental factors work together to shape early brain development.

Although the first stages of brain development are strongly affected by genetic factors, genes do not design the brain completely.^{6,7} Instead, genes allow the brain to fine-tune itself according to the input it receives from the environment. The brain's ability to shape itself lets individuals adapt to their surroundings more readily and more quickly than they could if genes alone determined the brain's wiring.⁸ The interplay of genetic and environmental factors is becoming better understood thanks to recent research in a relatively new scientific field called epigenetics.

The field of epigenetics has changed our understanding of how genes interact with the environment.

Epigenetics is the study of enduring changes in gene activity that do not change the DNA code itself. Many environmental factors and experiences result in a chemical 'mark' on certain genes, and this epigenetic change can influence the activity, or 'expression', of the gene.⁹

Roughly speaking, epigenetic processes are the software that directs the functioning of a gene's DNA hardware. Because the development of all cells, tissues and organs is affected by when and how specific genes are expressed, epigenetic processes can be a powerful influence on health and well-being.

Animal research shows that epigenetic changes can be passed from one generation to the next.

So far, much of what we know about epigenetics comes from research on animals. Numerous studies show how genetic activity can be altered by exposure to different foods, toxins, and experiences. One experiment involved genetically identical pregnant mice who had yellow fur, were overweight, and showed higher-than-average susceptibility to certain diseases. Half of these mice received a normal diet during pregnancy while the other half was fed a diet high in compounds known to affect gene expression.

The offspring of the first group resembled their mothers in color, obesity, and vulnerability to disease. The offspring from the second group were more likely to have brown fur, normal weight, and no increased disease risk (FIGURE 2). But like their mothers, all of the offspring in both groups had identical DNA sequences. The differences in color, weight, and health were due to differences in the activity of a specific gene. The compounds in the experimental diet caused chemical changes that inhibited this gene's expression in the second group of mothers, and this epigenetic process affected their offspring.

Remarkably, these offspring eventually gave birth to babies that showed the same traits—brown fur, normal weight, and low disease risk—even though this third generation received a normal diet. This experiment and others like it show that although epigenetic changes do not alter the DNA sequence itself, they can be passed down to the next generation.

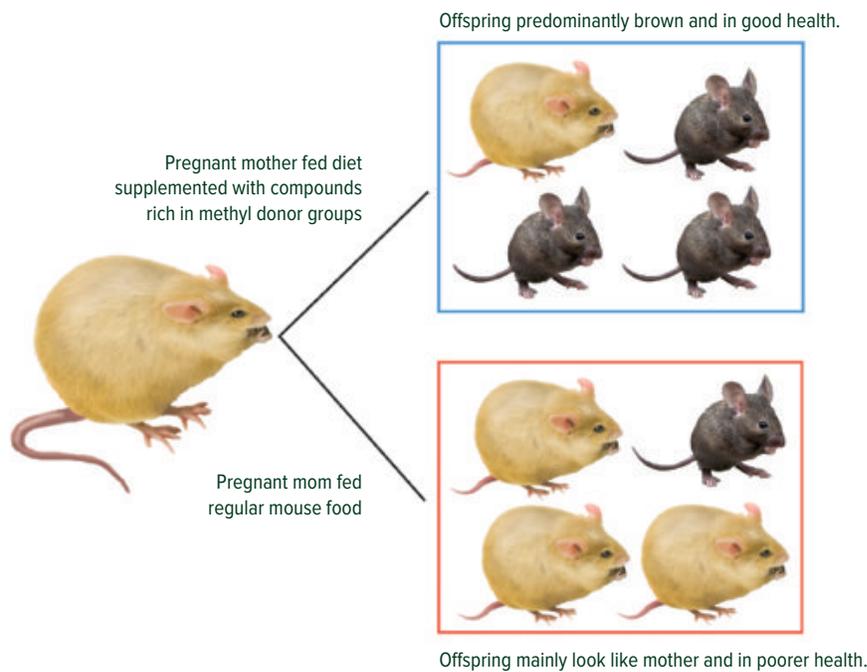


FIGURE 2:
Female Agouti
Mouse (Fully
Expressing a Gene
That Causes Yellow
Coat, Susceptibility
to Diabetes and
Obesity.)

Source: Illustration by Bill Day adapted from Waterland, RA., Jirtle, RL. Transposable elements: Target for early nutritional effects on epigenetic gene regulation. *Molecular and Cellular Biology*. 2003;23(15):5293-5300.

In another series of experiments, mice that received generous amounts of licking and grooming from their mothers were less anxious and had lower levels of stress hormones than those raised by mothers who showed anxious behavior and rarely nurtured their babies. A second phase of the experiment was designed to determine how much of this difference in stress was due to epigenetic factors. In this second procedure, offspring from the two types of mothers were exchanged immediately after birth.

The results showed the importance of early experience for the expression of specific genes. Babies born to high-nurturing mothers but raised by low-nurturing mothers developed high levels of anxious behavior similar to their foster moms. Babies born to low-nurturing mothers but raised by high-nurturing mothers showed less anxiety. In these offspring, a specific gene related to stress regulation was highly expressed, while in babies raised by low-nurturing moms it remained inactive.¹⁰

Epigenetics is strongly related to early brain development.

We know that children's experiences during the first years of life are strongly associated with long-term cognitive, emotional, and social outcomes.¹¹ And we know that the quality of a child's early experiences affects the development and function of the growing brain. But discovering how these processes occur has been difficult. The growing body of research on epigenetic processes, which are especially active early in development,¹² is likely to provide new answers to how adversity threatens optimal development.

For ethical and practical reasons, it is harder to study the gene/environment relationship in humans than in animals. Still, scientists have already found convincing evidence of epigenetic effects in human development. In one study, women who were pregnant during a severe famine tended to give birth to underweight infants. When these babies grew up and became parents themselves, they also tended to have underweight children, even though their own food intake since birth had not been affected by the famine.¹³ Other studies have found that childhood abuse is associated with lifelong decreased activation of a gene that protects against high levels of stress hormones.⁹ Recent research has found that parents' stress levels during their children's first three years were associated with epigenetic markers that were still apparent when children reached age 15.¹⁴

Studies show that high stress and low nurturing in the first stages of life impair brain development through epigenetic changes that reduce the growth of brain cells and the formation of connections. These effects are especially dramatic in brain areas related to memory, learning, and social and emotional adjustment.¹⁰

Epigenetic research supports the importance of a preventive approach to child health and well-being.

Epigenetic processes indicate that development is remarkably flexible. But this doesn't mean that undesirable epigenetic changes can simply be reversed by changing a child's environment later in development. Epigenetic changes—and their effects on behavior and health—are relatively stable once they occur. Moreover, such changes can be transmitted from generation to generation. Whether they can become permanent is not yet known, but even when the conditions that created an epigenetic mark no longer exist, it is likely to take several generations before it begins to fade.⁹

In other words, epigenetics makes a strong argument that prevention is the best policy approach for protecting young children from the effects of harmful influences. Early exposure to chronic stress, negative parenting, inadequate nutrition, and other environmental hazards can have long-term effects on adult health and emotional well-being. A better understanding of epigenetic changes may help inform us how to develop more effective interventions to protect young children from adverse experiences in the first years of life.¹⁵

References

1. Lipina SJ, Colombo JA. *Poverty and Brain Development During Childhood: An Approach From Cognitive Psychology and Neuroscience*. Washington, DC: American Psychological Association; 2009.
2. Gilmore JH, Lin W, Prasatwa MW, et al. Regional gray matter growth, sexual dimorphism, and cerebral asymmetry in the neonatal brain. *Journal of Neuroscience*. 2007;27(6):1255-1260.
3. Nowakowski RS. Stable neuron numbers from cradle to grave. *Proceedings of the National Academy of Sciences of the United States of America*. 2006;103(33):12219-12220.
4. Rakic, P. No more cortical neurons for you. *Science*. 2006;313:928-929.
5. Huttenlocher P. *Neural Plasticity: The Effects of the Environment on the Development of the Cerebral Cortex*. Harvard University Press; 2002.
6. Kagan J, Herschkowitz N, Herschkowitz E. *A Young Mind in a Growing Brain*. Mahwah, NJ: Lawrence Erlbaum Associates; 2005.
7. Elman JL, Bates EA, Johnson MH, et al. *Rethinking Innateness: A Connectionist Perspective on Development*. Cambridge, MA: MIT Press; 1996.
8. Pascual-Leone A, Amedi A, Fregni F, et al. The plastic human brain cortex. *Annual Review of Neuroscience*. 2005;28:377-401.
9. McGowan PO, Szyf M. The epigenetics of social adversity in early life: Implications for mental health outcomes. *Neurobiology of Disease*. 2010; 39(1): 66-72.
10. Meaney, M. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, 81(1), 41–79.
11. Duncan GJ, Ziol-Guest KM, Kalil A. Early childhood poverty and adult attainment, behavior, and health. *Child Development*. 2010; 81: 306–325.
12. Fagiolini M, Jensen CL, Champagne FA. Epigenetic influences on brain development and plasticity. *Current Opinion in Neurobiology*. 2009; 19:1-6.
13. Francis DD. Conceptualizing child health disparities: a role for developmental neurogenomics. *Pediatrics*. 2009; 124: S196–S202.
14. Essex MJ, Boyce WT, Hertzman C, et al. Epigenetic vestiges of early developmental adversity: Childhood stress exposure and DNA methylation in adolescence. *Child Development*. 2011; in press.
15. Shonkoff JP, Levitt P. Neuroscience and the future of early childhood policy: Moving from why to what and how. *Neuron*. 2010; 67: 689-691.