

Infant stress affects teen brain

Two-decade study reveals neural connection between early stress and anxiety and depression in girls.

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For some girls, stressful experiences in the first year of life seem to drive hormonal changes later in childhood. And these chemical changes, in turn, lead to abnormal brain connectivity and signs of anxiety and depression at age 18, suggests a study published today in *Nature Neuroscience*¹.

Researchers have long known that stress early in life is a risk factor for a host of psychological and physical problems, from mood disorders and substance abuse to obesity and cardiovascular disease. The new study proposes that for some individuals, childhood experiences permanently alter the way the body and brain cope with stress.

“This is one of the first demonstrations that early stress seems to have an impact on the the way this regulatory circuitry is set up in late adolescence,” says Richard Davidson, a neuroscientist at the University of Wisconsin-Madison and one of the leaders of the study.

In 1989, Davidson’s colleague Marilyn Essex launched what is now called the Wisconsin Study of Families and Work. Her team has since collected an array of medical and demographic information on several hundred children from birth to early adulthood.

In a study of this sample published in 2002, Essex reported that 4-year-olds exposed to high levels of everyday stress—such as maternal depression, parental arguments and financial woes—as infants and toddlers have high amounts of the stress hormone cortisol in their saliva. These high cortisol levels correlated with aggression, impulsivity and other behavioural problems when the children were observed two years later².

The new study builds on these findings, reporting that in girls, this increased cortisol when young affects brain function 14 years later.

The researchers scanned the brains of fifty-seven 18-year-olds while they lay quietly for 7 minutes. This procedure measures ‘resting-state functional connectivity’, or the synchrony of spontaneous activity between brain regions. Regions that are humming in sync at rest are thought to be strongly connected at other times (though scientists still have much to learn about the so-called resting brain: see ‘[Idle minds](#)’).

The study showed that 18-year-old girls who had had high cortisol levels at age 4 have weak connectivity between the amygdala, a deep nub of the brain known for processing fear and emotions, and the ventromedial prefrontal cortex, an outer region involved in curbing the amygdala’s stress response.

But without taking cortisol into account, early stress, in itself, is not significantly correlated with the differences in brain activity seen at age 18. “So what really matters is individual differences for individual children,” notes Kathleen Thomas, a developmental psychologist at the University of Minnesota in Minneapolis, who was not involved in the work.

The study also found that girls who have higher scores on anxiety tests have weaker synchrony between these two regions than do girls with lower scores. Intriguingly, the opposite pattern was found for depressive symptoms: higher depression scores correlate with stronger connectivity. That’s puzzling, because depression and anxiety tend to go hand in hand, and depressive symptoms are also linked to early stress, Thomas notes. It could be that both strong synchrony and weak synchrony are signs of an important connection between the two regions, she says.

The study is also notable for parsing sex differences, says Bruce McEwen, a neuroscientist at Rockefeller University in New York, who was not involved in the work. Although there’s no obvious explanation, anxiety and mood disorders are more prevalent in women,



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Depression and anxiety in teenagers is linked to stress early in life.

whereas antisocial behavior and substance abuse are more common in men. “It fits with the idea that they both feel what’s going on, but have different strategies for expressing their unhappiness and maladjustment,” he says.

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References

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2. Essex, M. J., Klein, M. H., Cho, E. & Kalin, N. H. *Biol. Psychiatry* **52**, 776–784 (2002).