

Does the immune system link childhood trauma to adult mental illness?

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Video Abstract

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Abstract

Although traumatic events in childhood can lead to psychiatric disorders later in life, the biological origins for this relationship aren't well defined. Overactivation of the immune system has been implicated in a range of psychiatric conditions, including bipolar disorder, schizophrenia, and anxiety disorder. Despite this well-known association, the events initially causing this activation remain a mystery. But a team of researchers in the Netherlands has uncovered a potential missing link: they found that traumatic events in childhood can cause inflammation that lasts into adolescence. This association is important because prior work has tied inflammation to the development of mood and anxiety disorders. The researchers surveyed one thousand teenagers affected by childhood trauma at ages 12, 14, 16 and 19. In addition, immune system activation at age 16 was evaluated by measuring high-sensitivity C reactive protein, a tell-tale sign of inflammation in the body. They found that, by age 16, the number of traumatic events experienced by a person was linked to the presence of mood and anxiety disorders at age 19. Interestingly, this number was also associated with increased levels of hsCRP. These results suggest that people who experience trauma in childhood still show signs of immune activation as teenagers. The team then looked at whether specific types of trauma were associated with higher levels of inflammation. They found that elevated hsCRP levels in teenagers were most strongly associated with the experience of separation trauma or sexual abuse in childhood, while weaker associations were found for verbal and physical abuse. These findings highlight the deep connection that exists between psychological and physical wellbeing. Further studies more clearly defining the pathway that leads from immune system activation to psychiatric disease could one day lead to early interventions designed to halt this progression.