

Developmental Trauma

The early chronic unpredictable stressors, losses, and adversities we face as children shape our biology in ways that predetermine our adult health. This early biological blueprint depicts our proclivity to develop life-altering adult illnesses such as heart disease, cancer, autoimmune disease, fibromyalgia, and depression. It also lays the groundwork for how we relate to others, how successful our love relationships will be, and how well we will nurture and raise our own children.

The ACE (Adverse Childhood Experiences Study) shows a clear scientific link between many types of childhood adversity and the adult onset of physical disease and mental health disorders. These traumas include being verbally put down and humiliated: being emotionally or physically neglected: being physically or sexually abused; living with a depressed parent, a parent with a mental illness, or a parent who is addicted to alcohol or other substances; witnessing one's mother being abused; and losing a parent to separation or divorce, losing a parent to death, witnessing a sibling being abused or abusive, violence in one's community, growing up in poverty, witnessing a father being abused by a mother, being bullied by a classmate or teacher. These types of chronic adversities change the architecture of a child's brain, altering the expression of genes that control stress hormone output, triggering an overactive inflammatory stress response for life, and predisposing the child to adult disease.

Brain imaging of kids when they reached adulthood who had traumatic childhood events and found that even exposure to very common but relatively chronic forms of family dysfunction, such as lack of family affection or parental discord, led to changes in the developing brain, decreasing the brain's size and volume. It has allowed researchers to demonstrate the striking scientific relationship between low-dose parental or family unkindness or neglect, damage to the young brain, and later negative health outcomes.

Chronic parental discord; enduring low-dose humiliation or blame and shame; chronic teasing; the quiet divorce between two secretly seething parents; a parent's premature exit from a child's life; the emotional scars of growing up with a hypercritical, unsteady, narcissistic, bipolar, alcoholic, addicted, or depressed parent; physical or emotional abuse or neglect. Although the details of individual experiences of adversity differ from one home to another and from one neighborhood to another, they are all precursors to the same organic chemical changes deep in the gray matter of the developing brain.

While disease develops for many reasons, including lifestyle, genetic, environmental toxins, and diet. But how is it that the early stress we face when we are very young, or teenagers, catches up with us when we are adults, altering our bodies, our cells, and even our DNA?

Your hypothalamus, as well as your pituitary and adrenal glands comprise the HPA stress axis. There is a powerful relationship between mental stress and physical inflammation. When we experience stressful emotions – anger, fear, worry, anxiety, rumination, grief, loss, the HPA axis releases stress hormones, including cortisol and inflammatory cytokines that promote inflammation. The problem is, when you are facing a lot of chronic stress, the stress response never shuts off. You're caught, perpetually, in the first half of the stress cycle. There is no state of recovery. Chronic stress leads to a dysregulation of our stress hormones – which leads to unregulated inflammation. And inflammation translates into symptoms and disease.

Emotional stress in adult life affects us on a physical level in quantifiable, life-altering ways. But when children or teens meet up with emotional stressors and adversity, they leave even deeper scars. These potential stressors include chronic put-downs, emotional neglect, parental divorce, a parent's death, the mood shifts of depressed or addicted parent, sexual abuse, medical trauma, the loss of a sibling, and physical or community violence. In each case, the HPA stress response can become reprogrammed so that it rev up one's inflammatory stress hormone response of the rest of one's life.

In young and growing children, the HPA stress axis is developing – and healthy maturation is heavily influenced by the safety or lack of safety we counter in the day-to-day environment. Early stress causes changes in the brain that reset the immune system so that either you no longer respond to stress or you respond in an exacerbated way and can't shut off that stress response. These epigenetic changes occur when early environmental influences both good and bad permanently alter which genes become active in the body. These epigenetic shifts take place due to a process called gene methylation. It doesn't matter what your genome is; what matters is how your genome is expressed – like a flower opening up. When the brain can't moderate our biological stress response, it goes into a state of constant hyperarousal and reactivity. So, when a child is young and his brain is still developing, if he is repeatedly thrust into a state of fight or flight, this chronic stress state causes these small, chemical markers to disable the genes that regulate the stress response – preventing the brain from properly regulating the response for the rest of his life.

Often, if we had early trauma, our adult HPA axis can't distinguish between real danger and perceived stress.

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