Biology and Environment

The Tutsi Genocide and Epigenetic Transmission of Maternal Stress to Children

In 1994, in a genocidal rampage committed by members of the Hutu majority against the Tutsi people of Rwanda, nearly 1 million perished within a three-month period. The horror was so extreme that in surveys of Rwandans during the years following the genocide, an estimated 40 to 60 percent reported symptoms of post-traumatic stress disorder (PTSD) (Neugebauer et al., 2009; Schaal et al., 2011). In PTSD, flashbacks, nightmares, anxiety, irritability, angry outbursts, and difficulty concentrating lead to intense distress, physical symptoms, and loss of interest in relationships and daily life.

Parental PTSD is a strong predictor of child PTSD (Brand et al., 2011; Yehuda & Bierer, 2009). In both children and adults, PTSD is associated with disruptions in the body’s stress response system, reflected in abnormal blood levels of stress hormones. In appropriate concentrations, stress hormones assist our brains in managing stress effectively. In individuals with PTSD, stress hormone levels are either too high or (more often) too low, contributing to persistently disturbed stress regulation.

Mounting evidence confirms that exposure to extreme adversity increases methylation of a chromosome-5 gene called GR, which plays a central role in stress-hormone regulation.

Might this epigenetic process contribute to parent-to-child transmission of PTSD?

To explore this question, researchers identified 50 Tutsi women who had been pregnant during the genocide (Perroud et al., 2014). Half had been directly exposed to the trauma; the other half had not been exposed due to being out of the country at the time. Eighteen years later, the mothers and their adolescent children were assessed for PTSD and depression by trained psychologists. Blood samples enabled genetic testing for methylation of the GR gene and assessment of stress-hormone levels (which we will discuss further in Chapter 3).

Compared with non-exposed mothers, mothers who witnessed the genocidal carnage had substantially higher PTSD and depression scores, and children of the two groups of mothers differed similarly. Also, as Figure 2.11 reveals, exposed mothers and their children displayed stronger GR methylation. And consistent with methylation’s dampening effect on gene expression, trauma-exposed mothers and their children had much lower stress-hormone levels than their non-exposed counterparts.

These findings are consistent with other evidence, in both animals and humans, indicating that prenatal exposure to the biological consequences of severe maternal stress can induce epigenetic changes, through methylation, that impair functioning of the body’s stress response system (Daskalakis & Yehuda, 2014; Mueller & Bale, 2008). In the Tutsi mothers and children, the effects of genocidal trauma were long-lasting, evident in serious psychological disorders nearly two decades later.

As the researchers noted, more remains to be discovered about exactly how maternal trauma exposure compromised the Tutsi children’s capacity to manage stress. Epigenetic processes, not just prenatally but also at later ages, may have been largely responsible. Alternatively, poor-quality parenting, resulting from maternal anxiety, irritability, anger, and depression, could have been the major influence. More likely, epigenetic changes, inept parenting, and other unfavorable environmental factors combined to place the Tutsi children at high risk for PTSD and depression. In Chapter 3, we will return to the impact of prenatal stress, including evidence showing that its negative impact can be lessened or prevented through social support.

**FIGURE 2.11** Methylation status of the GR gene in trauma-exposed and non-trauma-exposed Tutsi mothers and their children. Mothers who had been directly exposed to the Rwandan Tutsi genocide, as well as their children, showed elevated methylation of the GR gene, which is centrally involved in functioning of the body’s stress response system. (Based on Perroud et al., 2014.)