Epigenetic embodiment of early life adversity as a mechanism for perpetuating health disparities

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In recent decades, interdisciplinary researchers have debated the root causes behind social and racial disparities in health. Much of this research has focused separately on genetic or psychosocial explanations, with little integration of these two types of data. The study of epigenetic processes naturally bridges these areas by offering a mechanism for the biological embedding of stressful environmental exposures. An emerging literature on adverse prenatal and early childhood environments suggest that these early time periods may be particularly sensitive windows during which epigenetic marks are established. Findings from epigenetic anthropology studies will be presented that demonstrate epigenetic consequences resulting from exposure to social adversity *in utero,* and in early childhood, examined at both the level of individual gene regions, and through epigenome-wide analyses. These studies reveal small changes in DNA methylation in cord blood of neonates or in buccal cells of children in response to exposures such as prenatal depression, war trauma, racial discrimination, and time spent in institutional care. Taken together, these findings suggest that psychosocial stressors in the social environment of a child can alter the epigenome early in life, with health consequences potentially expanding into adulthood. Thus, epigenetic mechanisms may in part explain how unequal burdens of stress experienced by certain racial or social groups could perpetuate health disparities across generations.