GENETIC TESTING AND MOLECULAR BIOMARKERS
Volume 20, Number 9, 2016

Pp. 483–484
DOI: 10.1089/gtmb.2016.29021.sjt

PERSPECTIVE

Epigenetics and Racial Health Inequities

Sakura Oyama[1] and Sharon F. Terry[2]

The disproportionate disease and mortality burden of African Americans are among the most challenging public health issues in the United States today. Nationally, African Americans have an age-adjusted all-cause mortality rate that is 1.5 times that of non-Hispanic whites. African Americans are 30% more likely to die from cardiovascular disease, 40% more likely to be obese, and 60% more likely to be diagnosed with diabetes (Mensah *et al.*, 2005). In 2000, the U.S. Congress established the National Center for Minority Health and Health Disparities at the National Institute of Health to help spearhead efforts to study the causes of these health inequalities and recommend effective interventions (National Institutes of Health, n.d.).

Some researchers have attempted to attribute some or all of racial health disparities to differences in innate genetic predisposition. However, many studies directly undermine this hypothesis. First, research has consistently demonstrated that about 85% of all human genetic variation exists within human populations, whereas about 15% of variation exists between populations (Templeton, 1998). In fact, the field of genetics has all but debunked race as a biological concept, revealing that human genetic variation exists along a continuum and cannot be separated into distinct racial categories.

Immigrant studies have also largely dismissed innate genetic predisposition as an explanation for racial health disparities by revealing that health inequalities are more strongly influenced by contemporary environments than by common geographic origin. For example, in the United States, the birth weights of African American infants are lower than those of Caucasian infants. This is notable, as birth weight is strongly associated with mortality risk during the first year, developmental problems in childhood, and disease risk in adulthood. Yet, immigrant studies have shown that birth weight patterns of infants of African-born black women and U.S.-born white women are more similar to each other than to birth-weight patterns of infants of U.S.-born black women (David and Collins, 1997). This shows that health is primarily influenced by social, rather than innate genetic, factors.

But how exactly do social environments become embodied and influence health? Some researchers are hopeful that epigenetics may hold the answer. A growing body of work seeks to investigate the potential for social environments to induce durable, epigenetically based changes in gene regulation that are linked with changes in physiology and behavior.

Examining the epigenetic consequences of psychosocial stress has proven particularly useful in understanding the relationship between health and institutional and interpersonal racism. Most of these studies have focused on methylation at the glucocorticoid receptor, a gene important for regulating stress physiology. For example, several studies on teenage suicide victims have found that a history of childhood abuse was associated with methylation differences at the glucocorticoid receptor locus in the hippocampus (McHowan *et al.*, 2009; Beach *et al.*, 2010).

Other researchers have investigated the intergenerational transmission of epigenetic markers of psychosocial stress. Connie Mulligan's research has focused on women in the eastern Democratic Republic of Congo, a war-torn region in which women often fall victim to extreme violence. Her team identified a significant correlation between maternal prenatal stress and newborn methylation on the promoter of the glucocorticoid receptor gene (Mulligan *et al.*, 2012). Another study found that maternal depression during pregnancy predicted stress reactivity and methylation of the glucocorticoid receptor locus in buccal cells of infants measured 3 months after birth (Oberlander *et al.*, 2008).

Decreased glucocorticoid receptor function contributes to human platlet antigen axis hyperactivity and altered cortisol profiles, which have, in turn, been linked to negative health outcomes (Oberlander *et al.*, 2008). Although further research is needed to determine the long-term health effects of methylation changes at the glucocorticoid receptor locus, these studies suggest that psychosocial stressors during early life, whether in the womb or during childhood, can create lasting changes in epigenetic profiles that can be sustained across multiple generations.

This epigenetic research demonstrates that social health gradients are based, in part, on conditions experienced in the past, whether within one's lifetime or by recent ancestors. However, this research should not be used to undermine the need for current public health interventions. Furthermore, findings from epigenetic studies should be used to promote widespread social reforms that fight the larger geographic, sociocultural, economic, and political contexts in which health disparities are embedded. Reforms in public policies in a wide range of sectors, from education and housing to mass incarceration, must be addressed to create positive social environment for racial minorities that can counter generations of injustice. The importance of environmentally driven

[1]Washington University in St. Louis, St. Louis, Missouri.
[2]Genetic Alliance, Washington, District of Columbia.

epigenetic change will play an essential role, helping policy makers, as well as the public, to understand how socially and economically structured environments influence patterns of health and disease.

References

Beach SRH, Brody GH, Todorob AA, *et al.* (2010) Methylation at SLC6A4 is linked to family history of child abuse: An examination of the Iowa Adoptee sample. Am J Med Genet B Neuropsychiatr Genet 153:710–713.

David RJ, Collins JW Jr. (1997) Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. N Engl J Med 337:1209–1214.

McHowan PO, Sasaki A, D'Alessio AC, *et al.* (2009) Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. Nat Neurosci 12: 342–348.

Mensah GA, Mokdad AH, Ford ES, *et al.* (2005) State of disparities in cardiovascular health in the United States. Circulation 111:1233–1241.

Mulligan CJ, D'Errico NC, Stees J, *et al.* (2012) Methylation changes at NR3C1 in newborns associate with maternal prenatal stress exposure and newborn birth weight. Epigenetics 7:853–857.

National Institutes of Health (n.d.) National Institute of Minority Health and Disparities. June 21, 2016.

Oberlander T, Weinberg J, Papsdorf M, *et al.* (2008) Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR2C1) and infant cortisol stress responses. Epigenetics 3:97–106.

Templeton AR (1998) Human races: a genetic and evolutionary perspective. Am Anthropol 100:632–650. Web.

Address correspondence to:
Sharon F. Terry, MA
Genetic Alliance
4301 Connecticut Avenue, NW, Suite 404
Washington, DC 20008

E-mail: sterry@geneticalliance.org