CHAPTER 5

Beyond genetic race: biocultural insights into the causes of racial health disparities

Christopher W. Kuzawa[1] & Clarence C. Gravlee[2]
[1] *Department of Anthropology, Northwestern University*
[2] *Department of Anthropology, University of Florida*

Introduction

In the United States, it is well documented that blacks have higher mortality and lower life expectancy than other demographic groups (Wong *et al*. 2002). Differences in the burden of cardiovascular diseases, including heart attack, diabetes, and stroke, are among the most important contributors to these differences (Wong *et al*. 2002). The pervasiveness of these biological health disparities has led to debates about the underlying causes, with researchers generally falling into one of several camps. Among many within the medical establishment, there remains a persistent assumption that differences in health predicted by race categorization partially reflect the effects of genetic differences between racial groups (Kistka *et al*. 2007; Kramer *et al*. 2013). These researchers point to the fact that race membership often continues to predict disease risk after adjusting statistically for socioeconomic factors, combined with an assumption that populations carry different genes that influence the risk of developing these diseases (Burchard *et al*. 2003; Risch *et al*. 2002).

From a contrasting perspective, of which George Armelagos was among the leaders, most anthropologists have come to accept that races are best viewed as socially defined categories that do not have an underlying genetic basis (Armelagos and Goodman 1998; Livingstone 1962; Montagu 1942). Support for this position comes from the fact that genetic differences between groups tend to be small, while most genetic variation is found within groups (Brown and Armelagos 2001). At the same time, the race you are assigned to by society is known to shape the opportunities that are open to you, and the types and patterns of stressors that you experience. These in turn can have profound impacts on biology, and it is these differences in environments and experiences, rather

New Directions in Biocultural Anthropology, First Edition.
Edited by Molly K. Zuckerman and Debra L. Martin.
© 2016 John Wiley & Sons, Inc. Published 2016 by John Wiley & Sons, Inc.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

than genes, that most anthropologists view as driving the differing patterns of health and disease seen between individuals with different racial identities.

In this chapter, we build on the anthropological challenge to genetic race and critically evaluate the limitations of this concept as an explanation for contemporary race-related health disparities in racially segregated societies like the United States. We first discuss four factors that limit the utility of the genetic race concept and its applicability to racial health disparities. The first is that, whether we focus on physical features or genes, human variation does not come packaged in types or races, but is instead continuously varying, or clinal, and reflects complex histories of migration and gene flow between continents. Second, race membership – who is ascribed to what race – is socially and culturally defined, and not based upon genetic criteria. Third, unlike simple traits like eye color, most common chronic diseases that contribute to health disparities are "complex" traits that do not relate back to genes in a straightforward fashion. Lastly, in contrast to this, there are two powerful ways by which environments and experiences shape health disparities within and between societies, reflecting how the human body responds to and comes to create a record of these experiences. These include the cumulative negative impact of stressors, or "wear and tear," and alterations in how bodies develop, or plasticity. To illustrate these pathways, we conclude with two case studies which show how the social, cultural, political, and economic realities of race within contemporary US society can lead to biological disparities in health, without needing to invoke genes or racial types.

Background

The illusion of genetic races

Most of us have likely learned that humankind can be divided into three races, reflecting ancestral roots in Europe, Asia or Africa. Although it is still common for many to think in these terms, anthropologists have realized for more than half a century that modern human diversity does not come in such a tidy package (Brown and Armelagos 2001; Livingstone 1962; Montagu 1942). Our drive to divvy up human variation and place it into a few categories speaks to the way that the human mind works and, in particular, how it simplifies phenomena in order to make a complex world more manageable and understandable. Although it may feel natural to think about human variation in terms of categories like black and white, or European, African, and Asian, human diversity is more akin to the continuous color gradations of the visual spectrum than the discrete bands of color – red, orange, yellow, green, blue, indigo, and violet – that our minds impose upon it.

Starting in the mid-twentieth century, Frank Livingstone (Livingstone 1962) noted that as one pans across the globe and views variation in human traits such as skin color, body size, and facial features, they do not come in distinct types but form continuously varying gradients, or *clines*. Flying from Copenhagen to

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Dakar, you would be struck by obvious differences in features of appearance like skin or hair color. However, walking between those two populations, you would experience a continuous but imperceptible pace of change in these traits. Skin color would darken gradually as you moved from northern to southern Europe. It would continue to darken across the Mediterranean, and into Egypt and onward across Africa to the south, until reaching its darkest expression in the equatorial belt. There would be no obvious moment during your journey when you had crossed a threshold differentiating white from black, and the same would hold for other traits as well. This divide would be even murkier if exploring gradients in biology or appearance between Europe and Asia, which form a single supercontinent with greater opportunities for population movement and gene flow between them.

The recent advent of high-throughput genome sequencing technologies has provided a powerful tool to investigate the nature of human biological diversity. Since the initial sequencing of the human genome (Lander *et al*. 2001), technological advances have allowed the sequencing of genomes in thousands of individuals from across the globe. These studies consistently confirm what had previously been shown in earlier studies of blood groups or proteins: only a small part of the variability in human genes is explained by knowing what continent someone is from, or in what group they reside within a continent. Instead, the vast majority of genetic variation is found within human groups (Brown and Armelagos 2001).

While human genetic diversity does not cluster into tidy continental packages, as the traditional concept of race implies, it also turns out that we are an unusually homogenous species from a genetic perspective. There are only an estimated 150 000 chimpanzees living in the wild, compared to more than 7 billion humans. And yet, if you were to select two chimps at random, they would show substantially more genetic differences than would two humans (Prado-Martinez *et al*. 2013). There are many more humans but we all share a more recent common ancestor, and thus are more genetically similar to each other as a result.

Modern humans diverged from ancestral hominids around 200 000 years ago on the African continent. More recently, around 50 000 years ago, humans migrated out of Africa through the Nile corridor to colonize the rest of the globe (Pagani *et al*. 2015). Recent analyses suggest that the split between the original inhabitants of East Asia and Europe occurred between 40 000 and 20 000 years ago, and the split between East Asians and the lineage that peopled the Americas 20 000 years ago (Schiffels and Durbin 2014). Because humans have inhabited Africa for so much longer, the African continent harbors much greater genetic diversity than populations outside of Africa, and nearly all human genetic variants are found there (Long 2013). Populations outside of Africa can largely be viewed as subsets of this ancestral pool of African genetic variation, reflecting the narrower gene pools within the small "founder" populations of migrants that migrated out of Africa (Long 2013).

Local genetic adaptations have since arisen in both non-African and African populations, leading to biological features unique to local populations. One

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

example is skin color, which, as already discussed, follows a gradient across all continents that mirrors the intensity of ultraviolet radiation from sunlight. There is substantial variation in genes that contribute to differences in skin tone within Africa, and that evolved independently as populations moved to different latitudes, with different sun intensities, outside of Africa. This illustrates how natural selection shapes genetic variation *locally*, *within* continents, and thus contributes to the sizeable genetic differences between inhabitants of the same continent, or what would traditionally have been viewed as members of the same race.

Even if human genetic data do not support a simple race-based model of human diversity, they *do* reveal genetic differences between populations, some of which influence biology and thus could impact health. Might this global diversity in local population gene pools now contribute to race-related health disparities in a place like the United States? After all, in the United States, Europeans largely displaced the original Native American inhabitants of the continent, and initiated a slave trade that introduced Africans from a fairly narrow region of equatorial west and central Africa. Given that two geographically distant populations were brought side by side by these historical events – not unlike the experience of our jet-setting traveler between Copenhagen and Dakar – might genetic differences in their ancestral populations in Europe and Africa now contribute to contemporary health differences in the United States?

Race is socially and culturally, not genetically, defined

> Black man, black woman, black baby
> White man, white woman, white baby
> White man, black woman, black baby
> Black man, white woman, black baby
>
> *Dick Gregory (from Public Enemy, Fear of a Black Planet)*

For many people who grew up in the United States, the notion that human races do not have a firm genetic footing may be difficult to accept. We are conditioned to think of racial categories like *black* and *white* as corresponding to deep-seated differences between distinct types of humans. This impulse is part of what Smedley (Smedley and Smedley 2012:18) defines as the racial worldview: "a culturally structured, systematic way of looking at, perceiving, and interpreting" reality. But a fundamental lesson of anthropology is that each society develops its *own* culturally structured way of perceiving and interpreting reality. The racial worldview is but one way: a culture-bound product of unique demographic, political, and economic forces in US history. In other times and places, the same reality of human biological variation has been interpreted through other frames.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

We don't have to look far to find examples of alternative worldviews. In the United States, the rule of hypodescent, or "one-drop rule," automatically assigns anyone with a perceptible trace of African ancestry to the category *black* (Harris 1964). This rule is what makes the civil rights activist and comedian Dick Gregory's quote above ring true – and what explains how President Obama can be regarded as the first African American US president, despite having a mother who is defined as white. No such descent rule is evident in Latin American societies such as Brazil or Puerto Rico, where color terms are based on an individual's physical appearance and even siblings can be assigned to different categories (Duany 2002).

A related distinction between the US racial worldview and other ways of seeing human difference concerns the extent of ambiguity in racial classification. The rule of hypodescent in the United States historically sustains a simple classification scheme grounded in the relatively well-defined *black-white* binary. In Latin America, by contrast, the primacy of appearance over descent leads to the proliferation of color terms that capture subtle variations along a continuum from dark to light. Sanjek (1971), for example, identified 116 terms in the Brazilian "racial lexicon," and Duany (2002) listed 19 "major folk racial terms" in Puerto Rico. Usage of these terms is also more fluid than the US racial worldview allows.

These systems contrast with the United States in yet another important way: in some Latin American societies, including Brazil and Puerto Rico, "money whitens" (Degler 1971). In Brazil, interviewers were more likely to assign respondents to lighter categories if the respondents had more education, especially if they lived in mostly non-white places (Telles 2002), and non-white Brazilian parents with high levels of education are more likely to classify their children as white (Schwartzman 2007). Both examples illustrate how local conceptions of race emerge from cultural norms rather than underlying genetic realities.

Our emphasis on race as a worldview, rather than a genetically grounded set of categories, should not be taken to mean that it is merely a bad idea, or that it does not have real and serious biological *impacts*. The US racial worldview that emerged from the historical and economic realities in English North America (Harris 1964) today remains embedded in systemic racism that is experienced daily (Feagin 2006). Although race as most people understand it may be an illusion, that illusion and the system of oppression in which it is embedded have toxic – and even lethal – consequences for biology and health.

Why most health disparities are not like eye color: moving beyond genetic determinism

The culturally grounded criteria for race membership in any given society thus mean that self-identified race is often a poor proxy for genetic ancestry. In addition, a more fundamental challenge to the role of genes in health disparities comes from the fact that genes rarely have strong, direct effects on the biological

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

and behavioral traits that underlie these diseases. Nearly everyone reading this likely first learned about genetics through examples like eye color, which has an unusually straightforward genetic underpinning. Blue eye color is a homozygous recessive trait, meaning that you must inherit blue eye alleles from both of your parents to have blue eyes. If you inherit only one blue eye allele, you will likely have brown or black eyes, depending on the other inherited allele.

Although this example has traditionally served as a staple of textbook discussions of genetics, it is ultimately misleading when considering the role of genes in most biological and behavioral characteristics. Let us take the example of a leading cause of ethnicity or "race"-based health disparities in contemporary societies: cardiovascular disease. Genome-wide association studies (GWAS) search for statistical relationships between a trait, such as high cholesterol or hypertension, and information on hundreds of thousands or millions of genetic variants measured at single nucleotide positions (single nucleotide polymorphisms, or SNPs) across the genome, often in samples of tens to hundreds of thousands of individuals. For some traits, GWAS has been quite successful in identifying genes that explain variation in a health outcome. For instance, one recent study pooled genetic data from several hundred thousand individuals, from multiple global populations, to disentangle the genetic predictors of cholesterol levels. The authors identified 95 genes that together explained a respectable 10–12% of the variability in each lipid risk factor (Teslovich *et al*. 2010).

Such successes have been the exception rather than the rule, and most attempts to link health disparity-related traits to genes have proven far less successful. Take, for instance, body weight, which, owing to the rising public health burden of obesity, has been subjected to intensive investigation by GWAS. One large GWAS analysis of 250 000 individuals identified 32 genes that together explained only 1.45% of the variation in the body mass index (BMI) across the pooled populations (Speliotes *et al*. 2010). In other words, in this sample *nearly 99%* of the variance in BMI could *not* be explained by knowing which of the measured gene variants individuals carried in their genomes.

What accounts for the marked differences in the success of identifying genetic influences on cholesterol levels and obesity? Although there are many factors involved, a fundamental one is a difference in complexity. Cholesterol is a simple molecule, and its production in the liver, its circulation in the bloodstream, and clearance from the body are governed by relatively few factors. It is precisely this simplicity that accounts for the clinical success of statin drugs in lowering LDL cholesterol levels ("bad cholesterol"), with reductions typically varying from 15% to 50% depending on dosage. This drug blocks the protein product of a single gene (*HMGCR*) that codes for an enzyme required for cholesterol synthesis (HMG-CoA reductase). It should come as no surprise that the *HMGCR* gene was also among the strongest genetic predictors of cholesterol levels identified by the GWAS study mentioned above (Teslovich *et al*. 2010). In contrast, there is no drug for obesity, because there are no comparably simple pathways governing the gain and maintenance of excess body fat in most individuals. Instead, body weight is

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

related to a dizzying array of factors, including our appetites and cravings, our emotions, the amount of heat that our bodies produce after we eat a meal, our gut microbiome, personality type, and other traits that influence our activity level. As we will discuss below, one's risk for obesity is also powerfully shaped by our earliest experiences, beginning in the womb, when fetal development commits to different long-term metabolic settings depending on the nutritional conditions that we experience *in utero*. All of this biological complexity interacts with a similarly complex set of historical, environmental, cultural, political, and economic factors that vary across individuals and populations (Ulijaszek and Lofink 2006).

Thus, knowing someone's genotype will tell you a lot about relatively simple traits like their eye color, skin color, and, to a certain extent, even their cholesterol levels. However, most traits of importance to health disparities, including common and important conditions like obesity, hypertension, depression, and diabetes, are far more complex, meaning that knowing which genes someone carries is less helpful in explaining who becomes sick and who stays healthy.

Why environments matter more than genes for many health disparities

While genes are poor predictors of who suffers from most common chronic diseases, the environments that we inhabit – such as our experience of stress, our diet, and other factors – are comparably strong predictors of our health. There are several factors that help us make sense of why this is. Some of these explanations are widely understood, while others are only recently coming to light as a result of breakthroughs in our understanding of biology and the ways in which our experiences can influence it. As we discuss next, environments can shape patterns of health in two basic ways: first, through the cumulative lived experience of daily life, or what we might call "wear and tear," and second, by altering how our bodies develop biologically. Because both are crucial to understanding the origins of health disparities, we discuss each in turn.

Wear and tear: the cumulative negative impact of adverse environments

Until recently, most research on racial inequalities in health viewed these health disparities as a product of differences in socioeconomic status (SES). The basis of this approach is the observation that, in racially stratified societies like the United States, race is strongly associated with differences in common indicators of SES like income, education, and occupation. These indicators, in turn, are associated with inequalities in health outcomes. It stands to reason, then, that racial inequalities in health may be due at least in part to the unequal distribution of socioeconomic resources along racial lines.

This analytic strategy has produced mixed results. Some studies show that black–white differences in blood pressure, for example, disappear after controlling for individual or neighborhood-level indicators of SES (Morenoff *et al*. 2007;

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Syme *et al*. 1974), but others show that African Americans have higher blood pressure than whites even within levels of education, occupation or income (Hypertension Detection and Follow-up Program Cooperative 1977). In every case, however, accounting for differences in SES substantially weakens the magnitude of blood pressure differences between racially defined groups. This pattern holds for other health outcomes, too (Williams and Collins 1995).

The incomplete success of the SES model prompted two lines of criticism that led to other approaches. The first is that "controlling" for standard indicators like income and education does not address underlying causes (Kaufman and Cooper 1999), since these variables don't play a direct role in health. Consider, for example, that lower SES is associated with *lower* blood pressure in some societies (e.g., Ogunlesi *et al*. 1991). The next step, then, is to specify the intervening behaviors and exposures that shape the social patterning of health in a particular cultural setting.

One approach to this problem has been to focus on health behaviors such as smoking, diet, and physical activity, and related outcomes like obesity. These factors are known predictors of individual and population health, but they actually explain little of the racial disparities in health in the United States. For example, obesity explains 43% of the difference in hypertension prevalence between African American women in the United States and women in rural west Africa (Cooper *et al*. 1997). But *within* the United States, African American women are still almost twice as likely to have high blood pressure, compared to whites, even after controlling for obesity and SES (Bell *et al*. 2004).

A related line of criticism is that focusing too much on SES neglects the social significance of race itself. In racialized societies like the United States, categorizing people as *black* or *white* profoundly affects their life chances, such that racial classification is causally prior to standard measures of SES. As Richard Cooper (1984;721) puts it, "explaining racial differentials by education could in a causal sense be considered 'overcontrol': race is not confounded by the other variables; it is antecedent to them. It is race that influences class standing." And of course, the implications of racial classification are not limited to inequalities in social class. It may also affect health through non-economic forms of discrimination, including access to quality health care (Smedley *et al*. 2002), residential segregation (Massey *et al*. 2001), and exposure to racism in everyday social interaction (Essed 1991).

The most common approach to this problem today regards perceived discrimination as a chronic psychosocial stressor that impacts health and health behaviors. So far, researchers have linked self-reported experiences of discrimination to a range of outcomes, including elevated blood pressure, breast cancer, coronary artery calcification, body mass index, abdominal adiposity, preterm birth, low birth weight, depression, poor sleep, substance use, and poor preventive health behaviors (Gravlee 2009; Lewis *et al*. 2015). The associations between discrimination and health are often complex and sometimes manifest in unexpected directions, but this pattern may reflect the early state of the science.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Ongoing work to improve the measurement of experiences of discrimination holds promise to further illuminate how the wear and tear of systemic racism impacts biological systems and health (Williams and Mohammed 2013).

Developmental programming of later disease risk: biological memories of our early experience

In addition to wear and tear, *developmental plasticity* refers to the ways in which our experiences modify our biology and health by altering how our bodies develop (Kuzawa and Thayer 2011). The human body has capacities for plasticity that influence features of our biology that are straightforward and visible, such as relationships between height, age at puberty, and access to nutrition during childhood (Tanner 1962), but also those which are hidden from view but are crucial determinants of long-term risk for developing many common chronic diseases. Indeed, research in recent decades has shown that prenatal nutrition, stress, and other early life factors can modify fetal development in a way that elevates risk for developing conditions like hypertension, diabetes, heart attack, and stroke decades later, in adulthood (Barker 1994). These relationships reflect plasticity in traits like metabolism, blood pressure regulation, and stress biology, which are altered developmentally in response to conditions experienced during gestation and infancy. In addition, early experiences often lead to chemical modifications to chromosomes, called *epigenetic* changes, that determine which genes are turned on and which are silenced, which can also impact later health (Jenuwein and Allis 2001; Waterland and Michels 2007). In addition to the impact of the prenatal environment, research has also shown the lasting health impacts of early postnatal factors, such as whether we are breastfed or how quickly we gain weight as children (Norris *et al*. 2012).

This research suggests that an individual's risk of developing the most common adult chronic diseases, which are major contributors to health disparities, may be established, in part, by experiences much earlier in the lifecycle, often beginning prior to birth (Armelagos *et al*. 2009). By extension, some of the burden of disease in the current generation of adults may be traced to the social and environmental experiences of their mothers and other recent ancestors. For our present discussion, this means that the types of stressors that tend to be experienced differently according to one's self-identified race, such as differences in discrimination, nutrition or other factors, may impact not only the person who experiences them but also health in the next generation (Kuzawa and Sweet 2009).

Some of the earliest evidence for these relationships came from findings in humans who were born as lower birth weight babies, suggesting that they experienced prenatal nutritional stress (Barker *et al*. 1989). Among the better documented changes observed in adults who were born small is a tendency to have muscle that is resistant to the effects of insulin, meaning that they are less efficient at lowering their blood sugar after a meal. Individuals who experienced prenatal nutritional stress also tend to put on less fat in the lower

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

body or limbs, and to preferentially deposit it instead in the belly (leading to a so-called "apple-shaped" or "android" pattern of unhealthy fat deposition). Fat in this abdominal depot is distinct, because it is perfused with nerve fibers from the brain that release hormones like adrenaline, which allows the brain to rapidly mobilize stored fats for use as energy when the body is confronted with a stressor or challenge. Not only do individuals who were born small deposit more fat in this depot, but when they experience stress their fat cells also mobilize these stored fats for energy use more rapidly, which exacerbates the insulin resistance in muscle and other tissues (Girard and Lafontan 2008).

While the negative health impacts of fetal undernutrition, as reflected in being born small, have received much attention, there are also powerful effects of being overnourished *in utero*, which has growing relevance as rates of overweight and obesity rise across the globe. When a pregnant mother is overweight or obese, she is more likely to have high blood sugar during pregnancy, which at its most extreme manifests in gestational diabetes. This high blood sugar, in turn, can overfeed babies and increase their own risk of developing diabetes and obesity as adults. This has led to the notion that obesity is a condition with a transmissible component (Benyshek 2013). That the tendency of obese mothers to give birth to babies with a higher risk of becoming obese is at least partially non-genetic is demonstrated by the finding that offspring born after formerly obese mothers have lost weight as a result of gastric bypass surgery are much less likely to become obese compared to their siblings born prior to their mother's surgery, when the mothers were heavier and had elevated glucose and insulin during pregnancy (Smith *et al*. 2009).

In a similar fashion, when a woman experiences stress during pregnancy this can change how the offspring later responds biologically to stress (O'Connor *et al*. 2013; Tollenaar *et al*. 2011). In one recent study of an ethnically diverse sample in New Zealand, Zaneta Thayer found that women who reported deprivation or self-reported discrimination during pregnancy had higher cortisol levels during their pregnancy (Thayer and Kuzawa 2014, 2015). She followed up with the babies of these women, and found that those born to more stressed mothers produced more cortisol (measured in saliva) in response to the vaccinations that they received during their six-week pediatrician visits. This study shows that there are biological differences in how bodies respond to stress that are easily detectable soon after birth, and that are predicted by the stressful experiences of the mother during her pregnancy. Because cortisol is involved in a range of diseases, children born to mothers who experienced psychosocial stress during pregnancy may, as a result, be more prone to poor health in later life (Kuzawa and Sweet 2009; Thayer and Kuzawa 2011).

Can the biological "memories" of stress reach grandoffspring?

This work on developmental plasticity is showing how early life stressors, starting in the womb, can shape adult health by durably modifying development and long-term health. While these pathways of "biological memory" show how the

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

mother's nutrition or stress might influence the health of her adult offspring, the story unfortunately does not stop here, because among the offspring are females who go on to become pregnant and have offspring of their own. This leads to an important question: might the original stress experienced by the mother also affect the health of her *grandoffspring*?

Although few studies in humans have investigated this, there are good reasons to suspect that the effects of the original stressor could be passed on, albeit more weakly, across several generations. This is because some of the long-term effects of an adverse gestational environment on the offspring's adult health, such as insulin resistance, high blood pressure or inflammation, can negatively impact the gestational environments experienced by the next generation (Drake and Walker 2004). For instance, when a female fetus is exposed to a diabetic gestational environment, her heightened adult risk of diabetes increases the likelihood that her offspring – the grandoffspring of the originally diabetic mother – will also be exposed to a high-glucose, high-insulin gestational environment, thus perpetuating the pattern (Aerts and van Assche 2006; Castro and Avina 2002). Similarly, the heightened cortisol production in babies born to mothers who report having experienced discrimination during pregnancy, discussed in the New Zealand study (Thayer and Kuzawa 2015), might one day lead their bodies to expose the next generation to higher levels of cortisol during their own pregnancies.

Although evidence in humans still remains sparse, recent studies in other species, such as mice, even show that *male* experience can modify epigenetic factors that are transferred via sperm to offspring, and which go on to influence health across multiple generations (Kuzawa and Eisenberg 2014). Through epigenetic pathways of this sort, it is believed that early life stressors not only impact one's own adult health but can be transformed into intergenerational pathways for the perpetuation of health disparities across several generations (Drake and Walker 2004; Kuzawa and Sweet 2009).

These examples illustrate how differences in our lived experiences can lead to biological differences, operating both through wear and tear and through changes in how the body develops. Given these biological sensitivities to environmental stress, we should not be surprised that members of the most socially disadvantaged groups within a society tend to also suffer from a greater burden of common chronic and psychological diseases.

We now conclude this chapter with two case studies that illustrate the power of these two pathways – wear and tear and developmental plasticity – to shape health along lines of socially defined race.

Case study #1: hypertension in the African Diaspora

A key test case for understanding the genetic and environmental influences on racial inequalities in health is the puzzle of hypertension, or chronic high blood

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

pressure, in the African Diaspora. This puzzle is significant not only because of its staggering toll on black lives but also because it is a focus of debate about the meaning of race and racism in contemporary science and medicine (see also Chapter 4).

Throughout the Americas, populations of African ancestry have higher rates of hypertension than do other groups in the same societies, as exemplified in the United States by the 45% higher hypertension rates among African Americans than among whites (Gillespie *et al*. 2013). Many clinicians and health researchers assume that people of African descent are genetically predisposed to develop high blood pressure (Pickering 2001). Wilson and Grim (1991) proposed that this predisposition may have resulted from the African slave trade itself: high mortality from salt-depleting conditions during the Middle Passage may have selected for salt-retaining genotypes among enslaved Africans, which would increase susceptibility to hypertension in today's salt-rich environment. This so-called slavery hypothesis earned a place in clinical textbooks even before it was published in the peer-reviewed literature (Kaufman and Hall 2003), but it has been heavily critiqued on historical, evolutionary, and biological grounds (Armelagos 2005; Poston *et al*. 2001). By contrast, there is ample evidence that social factors contribute to hypertension within and between populations (Dressler 1999), but there remains no consensus about the causes of excess hypertension in the African Diaspora.

Debate about the relative importance of genetic versus environmental factors converges on the relationship between darker skin color and hypertension within populations of African descent in the Americas. This pattern has been interpreted as evidence of either genetic or sociocultural mechanisms (Boyle 1970; Tyroler and James 1978), but most early studies were unable to test these alternatives because they conflated two dimensions of skin color: the trait of skin pigmentation versus the cultural significance of skin color as a criterion of social status in color-conscious societies.

To address this problem, Gravlee and colleagues (Gravlee 2005; Gravlee and Dressler 2005; Gravlee *et al*. 2005) integrated ethnographic, epidemiological, and genetic techniques to disentangle the importance of biological and cultural factors as an influence on blood pressure in Puerto Rico. What they found was that, for Puerto Ricans defined as *blanco* (white) or *trigueño* (an intermediate term), higher SES was associated with lower blood pressure. For Puerto Ricans defined as *negro* (black), however, higher SES meant higher blood pressure, on average. This interaction makes sense in light of the ethnographic observation that racism becomes a more salient aspect of people's experience in Puerto Rico as they climb the social ladder. In a follow-up study, Gravlee *et al*. (2009) extended this analysis to include DNA-based estimates of African genetic ancestry. Their results confirmed the basic anthropological critique of race by showing that culturally constructed categories like race or *color* are poor predictors of genetic ancestry. They also reported that the interaction between SES and *color* predicted blood pressure better than did genetic ancestry.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Although cultural categories are poor predictors of genetic variation, it is precisely because we take those categories for granted that they impact people's lives in ways that our bodies register. The everyday wear and tear of being defined as black in a racially stratified society takes its toll in measurable biological outcomes that deleteriously impact health.

Case study #2: does the experience of racial discrimination in the United States have intergenerational health consequences?

In addition to wear and tear, another challenge to the presumed genetic contribution to the black–white disparity in cardiovascular health is the fact that US blacks *also* have lower birth weights than their white counterparts (Alexander *et al*. 1999) which, as reviewed earlier, elevates risk for hypertension, diabetes, and other cardiovascular diseases (Barker 1994). Although one may question whether the black–white difference in birth weight could be traced to genetic differences between these groups, there is strong evidence that this is not the case. For instance, recent US immigrants from the Caribbean and Africa were found to have birth weights nearly identical to those of US whites upon arrival (David and Collins 1997). However, among offspring born in the United States, birth weights began to shift towards the lower average African American birth weight (Collins *et al*. 2002).

The finding that the birth weights of immigrants and their descendants converge with those of their US ethnic counterparts provides compelling evidence that the black–white difference in birth weight is not likely due to genes (David and Collins 1997). Although the causes of the lower birth weights of African Americans and the offspring of African immigrants are not fully understood, factors associated with minority status, such as discrimination and racism, have been shown to predict prematurity and fetal growth restriction (Collins *et al*. 1998; Dole *et al*. 2004). Because a stressful fetal environment and fetal growth restriction, in turn, increase adult risk for conditions like hypertension, stroke, diabetes, and heart attacks, these findings suggest that the experience of race and racial discrimination could have impacts that transcend the exposed generation and potentially impact health in offspring and even grandoffspring (Kuzawa and Sweet 2009).

Discussion and conclusion

In this chapter, we have explored why the disparities in health that we see in relation to racial identity often have little to do with genes. Whether one studies bodily traits or genes, there is little evidence that race is a useful way to categorize this variation. Instead, most human traits vary as continuous gradients in

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

which the large majority of variation is found within, rather than between, the populations that inhabit different continents. We also see that, within a racially segregated society like the United States, one's racial identity is not based upon genes but upon cultural and social criteria that reflect political, economic, and historical factors that lead to markedly different patterns of opportunity and stress among members of these groups. Our case studies illustrate important ways by which these differences in lived experience can lead to biological differences operating through wear and tear or by modifying early developmental biology. These are examples of pathways that connect the social and cultural realities of race as a lived experience to patterns of biology and disease, and thus illustrate how "race becomes biology" (Gravlee 2009).

References

Aerts, L. and van Assche, F.A. (2006) Animal evidence for the transgenerational development of diabetes mellitus. *International Journal of Biochemistry and Cell Biology*, **38** (5–6), 894–903.

Alexander, G.R., Kogan, M.D., Himes, J.H., Mor, J.M. and Goldenberg, R. (1999) Racial differences in birthweight for gestational age and infant mortality in extremely-low-risk US populations. *Paediatric and Perinatal Epidemiology*, **13** (2), 205–217.

Armelagos, G.J. (2005) The slavery hypertension hypothesis – natural selection and scientific investigation: a commentary. *Transforming Anthropology*, **13** (2), 119–124.

Armelagos, G.J. and Goodman, A.H. (1998) Race, racism, and anthropology, in *Building a New Biocultural Synthesis: Political-Economic Perspectives on Human Biology* (eds A. Goodman and T. Leatherman), University of Michigan Press, Ann Arbor, MI, pp. 359–377.

Armelagos, G.J., Goodman, A.H., Harper, K.N. and Blakey, M.L. (2009) Enamel hypoplasia and early mortality: bioarchaeological support for the Barker hypothesis. *Evolutionary Anthropology*, **18** (6), 261–271.

Barker, D. (1994) *Mothers, Babies, and Disease in Later Life*. BMJ Publishing, London.

Barker, D.J., Osmond, C., Golding, J., Kuh, D. and Wadsworth, M.E. (1989) Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *BMJ*, **298** (6673), 564–567.

Bell, A.C., Adair, L.S. and Popkin, B.M. (2004) Understanding the role of mediating risk factors and proxy effects in the association between socio-economic status and untreated hypertension. *Social Science and Medicine*, **59** (2), 275–283.

Benyshek, D.C. (2013) The "early life" origins of obesity-related health disorders: new discoveries regarding the intergenerational transmission of developmentally programmed traits in the global cardiometabolic health crisis. *American Journal of Physical Anthropology*, **152** (suppl. 57), 79–93.

Boyle, E. Jr. (1970) Biological pattern in hypertension by race, sex, body weight, and skin color. *JAMA*, **213** (10), 1637–1643.

Brown, R. and Armelagos, G. (2001) Apportionment of racial diversity: a review. *Evolutionary Anthropology*, **10**, 34–40.

Burchard, E.G., Ziv, E., Coyle, N., *et al.* (2003) The importance of race and ethnic background in biomedical research and clinical practice. *New England Journal of Medicine*, **348** (12), 1170–1175.

Castro, L.C. and Avina, R.L. (2002) Maternal obesity and pregnancy outcomes. *Current Opinion in Obstetrics and Gynecology*, **14** (6), 601–606.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Collins, J.W. Jr, David, R.J., Symons, R., Handler, A., Wall, S. and Andes, S. (1998) African-American mothers' perception of their residential environment, stressful life events, and very low birthweight. *Epidemiology*, **9** (3), 286–289.

Collin,s J.W. Jr, Wu, S.Y. and David, R.J. (2002) Differing intergenerational birth weights among the descendants of US-born and foreign-born Whites and African Americans in Illinois. *American Journal of Epidemiology*, **155** (3), 210–216.

Cooper, R. (1984) A note on the biologic concept of race and its application in epidemiologic research. *American Heart Journal*, **108** (3, Part 2), 715–723.

Cooper, R., Rotimi, C., Ataman, S., *et al*. (1997) The prevalence of hypertension in seven populations of West African origin. *American Journal of Public Health*, **87** (2), 160–168.

David, R.J. and Collins, J.W. Jr. (1997) Differing birth weight among infants of U.S.-born blacks, African-born blacks, and U.S.-born whites. *New England Journal of Medicine*, **337** (17), 1209–1214.

Degler, C. (1971) *Neither Black Nor White: Slavery and Race Relations in Brazil and the United States*. University of Wisconsin Press, Madison, WI.

Dole, N., Savitz, D.A., Siega-Riz, A.M., Hertz-Picciotto, I., McMahon, M.J. and Buekens, P. (2004) Psychosocial factors and preterm birth among African American and White women in central North Carolina. *American Journal of Public Health*, **94** (8), 1358–1365.

Drake, A. and Walker, B. (2004) The intergenerational effects of fetal programming: non-genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *Journal of Endocrinology*, **180** (1), 1–16.

Dressler, W.W. (1999) Modernization, stress, and blood pressure: new directions in research. *Human Biology*, **71** (4), 583–605.

Duany, J. (2002) *The Puerto Rican Nation on the Move: Identities on the Island and in the United States*. University of North Carolina Press, Chapel Hill, NC.

Essed, P. (1991) *Understanding Everyday Racism: An Interdisciplinary Theory*. Sage Publications, Newbury Park, CA.

Feagin, J.R. (2006) *Systemic Racism: A Theory of Oppression*. Routledge, New York.

Gillespie, C.D., Hurvitz, K.A., Centers for Disease Control and Prevention. (2013) Prevalence of hypertension and controlled hypertension – United States, 2007–2010. *Morbidity and Mortality Weekly Report Surveillance Summary*, **62** (suppl 3), 144–148.

Girard, J. and Lafontan, M. (2008) Impact of visceral adipose tissue on liver metabolism and insulin resistance. Part II: Visceral adipose tissue production and liver metabolism. *Diabetes and Metabolism*, **34** (5), 439–445.

Gravlee, C. (2005) Ethnic classification in southeastern Puerto Rico: the cultural model of "color". *Social Forces*, **83** (3), 949–970.

Gravlee, C. and Dressler, W.W. (2005) Skin pigmentation, self-perceived color, and arterial blood pressure in Puerto Rico. *American Journal of Human Biology*, **17** (2), 195–206.

Gravlee, C.C. (2009) How race becomes biology: embodiment of social inequality. *American Journal of Physical Anthropology*, **139** (1), 47–57.

Gravlee, C., Dressler, W.W. and Bernard, H. (2005) Skin color, social classification, and blood pressure in southeastern Puerto Rico. *American Journal of Public Health*, **95** (12), 2191–2197.

Gravlee, C.C., Non, A.L. and Mulligan, C.J. (2009) Genetic ancestry, social classification, and racial inequalities in blood pressure in Southeastern Puerto Rico. *PLoS ONE*, **4** (9), e6821.

Harris, M. (1964) *Patterns of Race in the Americas*. Walker and Company, New York.

Hypertension Detection and Follow-up Program Cooperative Group. (1977) Race, education, and prevalence of hypertension. *American Journal of Epidemiology*, **106**, 351–361.

Jenuwein, T. and Allis, C.D. (2001) Translating the histone code. *Science*, **293** (5532), 1074–1080.

Kaufman, J. and Cooper, R. (1999) Seeking causal explanations in social epidemiology. *American Journal of Epidemiology*, **150** (2), 113–120.

Kaufman, J.S. and Hall, S.A. (2003) The slavery hypertension hypothesis: dissemination and appeal of a modern race theory. *Epidemiology*, **14** (1), 111–118.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Kistka, Z.A., Palomar, L., Lee, K.A., *et al.* (2007) Racial disparity in the frequency of recurrence of preterm birth. *American Journal of Obstetrics and Gynecology*, **196** (2), e131–136.

Kramer, H., Dugas, L. and Rosas, S.E. (2013) Race and the insulin resistance syndrome. *Seminars in Nephrology*, **33** (5), 457–467.

Kuzawa, C.W. and Eisenberg, D.T. (2014) The long reach of history: intergenerational and transgenerational pathways to plasticity in human longevity, in *Sociality, Hierarchy, Health: Comparative Biodemography* (eds M. Weinstein and M. Lane), National Research Council Press, Washington, DC, pp. 65–94.

Kuzawa, C.W. and Sweet, E. (2009) Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *American Journal of Human Biology*, **21** (1), 2–15.

Kuzawa, C.W. and Thayer, Z. (2011) Timescales of human adaptation: the role of epigenetic processes. *Epigenomics*, **3** (2), 221–234.

Lander, E.S., Linton, L.M., Birren, B., *et al.* (2001) Initial sequencing and analysis of the human genome. *Nature*, **409** (6822), 860–921.

Lewis, T.T., Cogburn, C.D. and Williams, D.R. (2015) Self-reported experiences of discrimination and health: scientific advances, ongoing controversies, and emerging issues. *Annual Review of Clinical Psychology*, **11** (1), 407–440.

Livingstone, F.B. (1962) On the non-existence of human races. *Current Anthropology*, **3** (3), 279–281.

Long, J.C. (2013) The aimless genome, in *Anthropology of Race: Genes, Biology and Culture* (ed. J. Hartigan), School for Advanced Research Press, Santa Fe, NM, pp. 169–186.

Massey, D., (2001) Residential segregation and neighborhood conditions in U.S. metropolitan areas, in *American Becoming: Racial Trends and Their Consequences, Vol I* (eds N. Smelser, W. Wilsonand F. Mitchell), National Academy of Sciences Press, Washington, DC, pp. 391–434.

Montagu, A. (1942) *Man's Most Dangerous Myth: The Fallacy of Race*. Columbia University Press, New York.

Morenoff, J., House, J., Hansen, B., Williams, D., Kaplan, G. and Hunte, H. (2007) Understanding social disparities in hypertension prevalence, awareness, treatment, and control: the role of neighborhood context. *Social Science and Medicine*, **65** (9), 1853–1866.

Norris, S.A., Osmond, C., Gigante, D., *et al.* (2012) Size at birth, weight gain in infancy and childhood, and adult diabetes risk in five low- or middle-income country birth cohorts. *Diabetes Care*, **35** (1), 72–79.

O'Connor, T.G., Bergma,n K., Sarkar, P. and Glover, V. (2013) Prenatal cortisol exposure predicts infant cortisol response to acute stress. *Developmental Psychobiology*, **55** (2), 145–155.

Ogunlesi, A., Osotimehin, A.F., Abbiyessuku, F., *et al.* (1991) Blood pressure and educational level among factory workers in Ibadan, Nigeria. *Journal of Human Hypertension*, **5**, 375–380.

Pagani, L., Schiffels, S., Gurdasani, D., *et al.* (2015) Tracing the route of modern humans out of Africa by using 225 human genome sequences from Ethiopians and Egyptians. *American Journal of Human Genetics*, **96** (6), 986–991.

Pickering, T.G. (2001) Why is hypertension more common in African Americans? *Journal of Clinical Hypertension*, **3** (1), 50–52.

Poston, W.S., Pavlik, V.N., Hyman, D.J., *et al.* (2001) Genetic bottlenecks, perceived racism, and hypertension risk among African Americans and first-generation African immigrants. *Journal of Human Hypertension*,**15** (5), 341–351.

Prado-Martinez, J., Sudmant, P.H., Kidd, J.M., *et al.* (2013) Great ape genetic diversity and population history. *Nature*, **499** (7459), 471–475.

Risch,, N., Burchard, E., Ziv, E. and Tang, H. (2002) Categorization of humans in biomedical research: genes, race and disease. *Genome Biology*, **3** (7), 1–12.

Sanjek, R. (1971) Brazilian racial terms: some aspects of meaning and learning. *American Anthropologist*, **73**, 1126–1143.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Schiffels, S. and Durbin, R. (2014) Inferring human population size and separation history from multiple genome sequences. *Nature Genetics*, **46** (8), 919–925.

Schwartzman, L. (2007) Does money whiten? Intergenerational changes in racial classification in Brazil. *American Sociological Review*, **72**, 940–963.

Smedley, A. and Smedley, B.D. (2012) *Race in North America: Origin and Evolution of a Worldview*. Westview Press, Boulder, CO.

Smedley, B., Stith, A. and Nelson, A. (2002) *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. National Academies Press, Washington, DC.

Smith, J., Cianflone, K., Biron, S., *et al*. (2009) Effects of maternal surgical weight loss in mothers on intergenerational transmission of obesity. *Journal of Clinical Endocrinology and Metabolism*, **94** (11), 4275–4283.

Speliotes, E.K., Willer, C.J., Berndt, S.I., *et al*. (2010) Association analyses of 249,796 individuals reveal 18 new loci associated with body mass index. *Nature Genetics*, **42** (11), 937–948.

Syme, S., Oakes, T., Friedman, G., Feldman, R., Siegelaub, A. and Collen, M. (1974) Social class and racial differences in blood pressure. *American Journal of Public Health*, **64** (6), 619–620.

Tanner, J.M. (1962) *Growth at Adolescence; With a General Consideration of the Effects of Hereditary and Environmental Factors upon Growth and Maturation from Birth to Maturity*. Blackwell Scientific, Oxford.

Telles, E. (2002) Racial ambiguity among the Brazilian population. *Ethnic and Racial Studies*, **25** (3), 415–441.

Teslovich, T.M., Musunuru, K., Smith, A.V., *et al*. (2010) Biological, clinical and population relevance of 95 loci for blood lipids. *Nature*, **466** (7307), 707–713.

Thayer, Z.M. and Kuzawa, C.W. (2011) Biological memories of past environments: epigenetic pathways to health disparities. *Epigenetics*, **6** (7), 798–803.

Thayer, Z.M. and Kuzawa, C.W. (2014) Early origins of health disparities: material deprivation predicts maternal evening cortisol in pregnancy and offspring cortisol reactivity in the first few weeks of life. *American Journal of Human Biology*, **26** (6), 723–730.

Thayer, Z.M. and Kuzawa, C.W. (2015) Ethnic discrimination predicts poor self-rated health and cortisol in pregnancy: insights from New Zealand. *Social Science and Medicine*, **128**, 36–42.

Tollenaar, M.S., Beijers, R., Jansen, J., Riksen-Walraven, J.M. and de Weerth, C. (2011) Maternal prenatal stress and cortisol reactivity to stressors in human infants. *Stress*, **14** (1), 53–65.

Tyroler, H.A. and James, S.A. (1978) Blood pressure and skin color. *American Journal of Public Health*, **68** (12), 1170–1172.

Ulijaszek, S.J. and Lofink, H. (2006) Obesity in biocultural perspective. *Annual Review of Anthropology*, **35**, 337–360.

Waterland, R.A. and Michels, K.B. (2007) Epigenetic epidemiology of the developmental origins hypothesis. *Annual Review of Nutrition*, **27**, 363–388.

Williams, D.R. and Collins, C. (1995) US socioeconomic and racial differences in health: patterns and explanations. *Annual Review of Sociology*, **21**, 349–386.

Williams, D.R. and Mohammed, S.A. (2013) Racism and health I: pathways and scientific evidence. *American Behavioral Scientist*, **57** (8), 1152–1173.

Wilson, T.W. and Grim, C.E. (1991) Biohistory of slavery and blood pressure differences in blacks today. *A hypothesis. Hypertension*, **17** (1 Suppl), I122–128.

Wong, M.D., Shapiro, M.F., Boscardin, W.J. and Ettner, S.L. (2002) Contribution of major diseases to disparities in mortality. *New England Journal of Medicine*, **347** (20), 1585–1592.

10.1002/9781118962954.ch5, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/9781118962954.ch5 by Northwestern University, Wiley Online Library on [23/01/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License