Health disparities refer to differences between groups of people. These differences can affect how frequently a disease affects a group, how many people get sick, or how often the disease causes death. Over the past 20 years, researchers, advocates, and public health officials have documented, and tried to address, the striking health disparities between racial/ethnic minority populations and Whites. The differences between Blacks and Whites are particularly striking and have been frequently documented in research studies. Some of these disparities include rates of infant mortality, obesity, cardiovascular disease, metabolic syndrome and diabetes, resulting in overall premature mortality.

Disparities in Infant Mortality Rates

The rate of infant mortality is 5.5 per 1000 for Whites and 12.4 for Blacks. This rate actually reflects an improvement. Until last year, it was 13.8 per 1000. (The CDC credits the increase in breastfeeding rates among African Americans for this decrease.) This dismal statistic accounts for the fact that the U.S. is ranked 41st in the world in terms of infant mortality. Infant mortality is a key index of overall health of a country. Our rank puts us behind many developing countries. Much of the high mortality rate among Black infants is due to high rates of preterm birth. According to the World Health Organization, preterm birth is the number one cause of infant mortality. Here too there is a disparities.

Racial/Ethnic Differences in Obesity Rates

Obesity rates also show striking ethnic-group differences, with African Americans having significantly higher rates than Whites. Interestingly, a similar pattern appears for indigenous people in Australia and Maori and Pacific Islanders in New Zealand.

There is also a similar pattern based on socioeconomic status, with lower income people having significantly higher rates.
Diabetes and Heart Disease

We see similar patterns in rates of diabetes, metabolic syndrome, and heart disease, particularly for African Americans and American Indians. For example, the rate for diabetes in the U.S. is 7.6 per 1000 for Whites, 13.2 for non-Hispanic Blacks, and 15.6 for American Indians/Alaska Natives (Centers for Disease Control and Prevention, 2014b). Not surprisingly, African American men tend to die at a younger age than men or women of other ethnic groups (Centers for Disease Control and Prevention, 2014a). (Hispanic women actually have the greatest longevity.)

What may surprise you is that these various manifestations of health disparity have the same underlying physiology. Inflammation, or more specifically, the upregulation of the inflammatory response system, underlies them all. To understand these inflammation effects, we need to draw from the research in the field of psychoneuroimmunology. Trauma intersects with these physiological effects in some interesting ways and enhances them (Kendall-Tackett, 2010b).

The Physiology of Stress

In a simplified form, we can think of the stress response as having three key components (Kendall-Tackett, 2010b).

The first is the *catecholamine*, or fight-or-flight response. In response to perceived threat, our bodies secrete three neurotransmitters: epinephrine, norepinephrine, and dopamine.

The second component of the stress response is the *HPA axis*. HPA stands for hypothalamic-pituitary-adrenal. This is a cascade response, meaning that in response to threat, the hypothalamus secretes the stress hormone CRH, which causes the pituitary to secrete the stress hormone ACTH, which causes the cortex of the adrenal gland to secrete the stress hormone cortisol.

The third component of the stress response is the *immune response*. In response to threat, the immune system increases inflammation by releasing proinflammatory cytokines, messenger molecules of the immune system. These molecules have two important functions: fighting infections and healing wounds. When under threat, the body prepares for possible attack—and injury—by being ready to heal wounds.

All components of the stress response are adaptive, meaning that they increase the likelihood of survival. This three-part stress response is meant to be acute: it turns on and it turns back off when the threat is over. The problem is that chronic stress, trauma, or even daily social rejection can keep this system activated, and that increases the likelihood of disease.
The Metabolic Syndrome

One other physiological state is necessary to describe, and that is the metabolic syndrome. The metabolic syndrome is the precursor syndrome to type-2 diabetes and is also a risk factor for heart disease (Haffner & Taegtmeyer, 2003). There are four key symptoms of the metabolic syndrome: insulin resistance, high LDL and VLDL cholesterol, high triglycerides, and visceral (abdominal) obesity. Metabolic syndrome, particularly insulin resistance, is related to increased inflammation and inflammation increases insulin resistance. Both are related to a chronically upregulated stress system.

Social Rejection and Inflammation: The Key to Understanding Health Disparities

In the introduction to the recent book, Social Pain (Jenson-Campbell & MacDonald, 2011), the authors describe how we are designed to be in relationship with each other. That being socially connected increases our chances of survival and that being part of a group provides resources, protection, and safety. When we perceive that we are not part of a group, we experience this rejection in the same part of our brains that process physical pain: the anterior cingulate cortex. Physiologically, humans experience social rejection as a threat to their physical survival (Dickerson, 2011; Eisenberger, 2011; Panksepp, 2011).

Microaggressions and Physical Health

Given that social rejection is perceived as a threat to survival, it’s reasonable to hypothesize that it would increase inflammation. And it does. Health psychologists have documented that “perceived discrimination” (i.e., perceiving yourself as low in the social hierarchy) increases inflammation. In one study of 296 African Americans, Lewis et al. (2006) found that self-reported experiences of discrimination increased C-reactive protein (CRP; a common marker of chronic inflammation. It predicts risk of cardiovascular disease). Their measure included the following questions:

- You are treated with less courtesy than other people.
- You are treated with less respect than other people.
- You receive poorer service than other people at restaurants and stores.
- People act as if they think you are not smart.

In another study (McDade, Hawkley, & Cacioppo, 2006), perceived low social status was related to elevated C-reactive protein. This was a 3-year longitudinal study of 188 middle-aged and older adults. African Americans, women, and those with low education levels had the highest CRP. Another study (Hong, Nelesen, Krohn, Mills, &Dimsdale, 2006) found that perceived low social status was related to vascular inflammation, with elevated levels of the inflammatory molecules, ET-1 and sICAM. These effects were independent of hypertension status or ethnicity.

The effects of perceived discrimination can show up rather early. In a study of high school students (Goodman, McEwen, Huang, Dolan, & Adler, 2005), low parental education (a marker of socioeconomic status) predicted metabolic and cardiovascular risk factors including higher insulin levels, higher glucose, greater insulin resistance, higher HDL and lower LDL cholesterol, higher waist circumference, and higher BMI.
The Role of Sleep

Sleep is another factor that can be affected by everyday experiences of discrimination, and it too has a major impact on health (Kendall-Tackett, 2009b). For example, sleep problems can make you fat. In a meta-analysis of 36 studies (N=634,511 adults and children), short sleep duration (< 5 hours) was related to obesity worldwide (Cappuccio et al., 2008).

Sleep problems increase symptoms of metabolic syndrome and inflammation, thereby increasing the risk of diseases, such as heart disease and diabetes (Suarez & Goforth, 2010). One study found that short sleep duration was related to metabolic syndrome in middle-aged adults (Hall et al., 2008). These symptoms included abdominal obesity, elevated fasting glucose, and high triglycerides. Suarez and Goforth (Suarez & Goforth, 2010) noted that even subclinical sleep disorders increase risk for cardiovascular disease, hypertension, type-2 diabetes, metabolic syndrome, and all-cause mortality. And even short periods of sleep deprivation (e.g., 1 or 2 days) can elevate cortisol and glucose levels, and increase insulin resistance (McEwen, 2003).

Ethnic Differences in Sleep

Given these health effects, it’s interesting to note striking ethnic group differences in sleep. These could be the result of daily exposure to microaggressions or a result of trauma (or both). Either of these appear to upregulate the inflammatory response system. For example, a study of Black and White adults (N=187) found that Blacks had shorter sleep duration and lower sleep efficiency than whites (Mezick et al., 2008). On average, Blacks took 25 minutes to fall asleep compared to 16 minutes for Whites. The percentage of slow-wave sleep was 3.6% for Blacks and 6.8% for Whites. Both are markers of sleep quality. Longer sleep latency (time to get to sleep) and lower percentage of slow-wave sleep both reflect poor sleep quality and a higher state of hyperarousal consistent with the daily experiences of perceived threat. This difference persisted even after controlling for SES. Another study of 97 Black and White adults had similar findings (Beatty et al., 2011). Perceived unfair treatment for both groups was associated with poorer sleep quality, more daytime fatigue, shorter sleep duration, and a smaller proportion of REM. Blacks had lower sleep time and poorer sleep efficiency overall.

Ethnic-Group Differences in Trauma

Trauma can also increase the risk of diseases, such as diabetes and heart disease, and it does it by increasing inflammation. For example, data from year 32 of the Dunedin Multidisciplinary Health and Development study, a birth-cohort study from Dunedin, New Zealand, revealed that those who experienced adverse childhood experiences (defined in this study as low SES, maltreatment, or social isolation) had higher rates of major depression, systemic inflammation, and having at least 3 metabolic risk markers (Danese et al., 2009).

Data from the Nurses’ Health Study II, a study of more than 73,000 nurses, revealed that physical and sexual abuse in childhood or as a teen increased the risk of type-2 diabetes, even after adjusting for age, race, body type at age 5, parental
education, and parental history of diabetes (Rich-Edwards et al., 2010). Severity of abuse increased symptoms dramatically. There was a 50% increase in diabetes risk for those who experienced severe physical abuse and a 69% increase in risk in those who experienced repeated forced sex. Body Mass Index (BMI) was also influenced by past abuse. Physically and sexually abused girls had higher BMIs and the trajectories grew wider as the girls grew (i.e., they gained weight at a faster clip). This was particularly true for those who experienced repeated forced sex.

Unfortunately, there are ethnic group differences in experiences of trauma and the impact that it has. For example, a study of 177 Blacks and 822 Whites compiled a composite of early life adversities and 5 measures of inflammation. They found that early-life adversity predicted higher levels of inflammation for Blacks, but not Whites (Slopen et al., 2010). Researchers from the Black Women’s Health Study (N=33,298) found that early-life sexual and physical abuse was related to overall and central obesity (Boynton-Jarrett, Rosenberg, Palmer, Boggs, & Wise, 2012). This relationship existed even after controlling for lifestyle factors.

Research in perinatal health suggests that Black women may have more lifetime exposure to trauma, and this directly affects their rates of preterm birth. For example, in a national survey of 1,581 pregnant women (709 were Black women), there was more lifetime PTSD and trauma exposure for Black women (Seng, Kohn-Wood, McPherson, & Sperlich, 2011). Current prevalence for PTSD was 4 times higher for Black women. The rates did not differ by SES and are explained by greater trauma exposure. Child abuse was the most common cause of PTSD for both groups.

High rates of trauma and PTSD during pregnancy are concerning because of their relationship to both low birthweight and gestational age. A prospective, 3-cohort sample of first-time pregnant women compared 255 women with PTSD; 307 trauma-exposed, resilient women (no PTSD); and 277 non-trauma-exposed women (Seng, Low, Sperlich, Ronis, & Liberzon, 2011). They found that babies born to PTSD+ women weighed 283 g less than those born to resilient women and 221 g less than those born to non-exposed women. PTSD was also associated with a shorter gestation. These findings suggest trauma exposure and PTSD in pregnancy increased the risk for preterm birth, and both are more common in Black women.

Inflammation is a possible mechanism for this relationship. A study of mothers with stress and depression revealed high levels of the inflammatory molecules IL-6 and TNF-α (Coussons-Read, 2005). In addition to fighting infections and healing wounds, these molecules also ripen the cervix, increasing the likelihood of preterm birth.

Along these same lines, a randomized clinical trial using DHA-enhanced eggs also suggest that inflammation is related to preterm birth (Smuts, 2003). In this trial, 291 mothers were asked to eat one egg a day for the last trimester of their pregnancies. The eggs were either regular or were enriched with the omega-3 fatty acid DHA. DHA is highly anti-inflammatory. The mothers were participating in the WIC program (the Women, Infants, and Children supplemental feeding program) in Kansas City. Approximately 70% of the sample was African American. This simple, cheap intervention increased gestation length by 6 days.

How Shall We Then Treat?
Understanding the mechanism underlying health disparities gives us some possible places to intervene. While we continue to work for social justice in the wider society, there are ways we can intervene when working with a particular client or patient.

We must start by acknowledging the health effects of discrimination and recognizing how it contributes to health disparities. In fact, given these health effects, we might even argue that microaggressions rise to the status of trauma exposure (i.e., the body is perceiving that it these events are a physical threat).

To address preterm birth, we need to be proactive in screening for depression and PTSD in pregnancy. We also need to counter the effects of chronic inflammation directly by supplementing with omega-3 fatty acids (particularly DHA and EPA) (Kendall-Tackett, 2010a). Most American women are deficient in these and they are safe to use in pregnant women (see Kendall-Tackett, 2009a). In postpartum women, breastfeeding downregulates the stress response and decreases inflammation. It is important to continue to support community organizations that are increasing breastfeeding rates in the African American community. This will help with the health of both mothers and babies (Groer & Kendall-Tackett, 2011).

Finally, we need to recommend activities that we know downregulate the stress and inflammatory response systems, including both exercise and long-chain omega-3s (Kendall-Tackett, 2009b). Both help and will improve the health of African Americans and hopefully decrease health disparities.
References


Centers for Disease Control and Prevention. (2014b). Racial and ethnic differences in diagnosed diabetes among people aged 20 years or older, United States, 2010-2012.


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