



VCU

Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations

Graduate School

2020

Associations Between Stress, Racial Discrimination, and Cytokine Levels in Black Americans

Takia Williams
Virginia Commonwealth University

Follow this and additional works at: <https://scholarscompass.vcu.edu/etd>



Part of the [Mental and Social Health Commons](#), [Psychiatry and Psychology Commons](#), [Psychology Commons](#), [Public Health Commons](#), [Social Statistics Commons](#), and the [Urban Studies and Planning Commons](#)

© The Author

Downloaded from

<https://scholarscompass.vcu.edu/etd/6181>

This Thesis is brought to you for free and open access by the Graduate School at VCU Scholars Compass. It has been accepted for inclusion in Theses and Dissertations by an authorized administrator of VCU Scholars Compass. For more information, please contact libcompass@vcu.edu.

Associations Between Stress, Racial Discrimination, and Cytokine Levels in Black Americans

Takia S. Williams

Virginia Commonwealth University

Acknowledgments

This project was supported by internal funding from VCU as well as CTSA award No. UL1TR000058 from the National Center for Advancing Translational Sciences. Its contents are solely the responsibility of the authors and do not necessarily represent official views of VCU, the National Center for Advancing Translational Sciences or the National Institutes of Health.

Correspondence should be addressed to Takia Williams and Marcia Winter, Virginia

Commonwealth University Box 842018, Richmond, VA, 23284.

DISCRIMINATION, INFLAMMATION, BLACK AMERICAN	2
ABSTRACT	3
INTRODUCTION	4
THREAT, INFLAMMATION, AND “WEATHERING” IN BLACK AMERICANS	6
CURRENT STUDY AIMS	8
METHODS	9
PARTICIPANTS	9
PROCEDURES	10
MEASURES	10
DATA ANALYSIS	13
RESULTS	14
DISCUSSION	15
LIMITATIONS AND FUTURE DIRECTIONS	16
IMPLICATIONS	19
TABLES	24
VITA	25

Abstract

Inflammation is a common pathophysiological pathway for a number of chronic diseases and is influenced by exposure to stress. Although there are racial disparities in health outcomes, relatively little is known about factors that may influence the inflammatory response in Black American individuals. This study examined whether racial discrimination and other forms of stress are associated with the balance of pro- and anti- inflammatory cytokines in Black American adults. Data from 22 participants were drawn from a larger study of Black American children (ages 5-12) and their primary caregivers drawn from low income neighborhoods in Richmond, Virginia. Caregivers reported demographics, frequency of experiences of discrimination, and stressful life events; and gave blood samples via venipuncture. Inflammation was assessed using a multiplex assay to capture both pro- and anti- inflammatory cytokines.

Results showed that there was no statistically significant correlation between the balance of pro- and anti- inflammatory cytokines and social adversity (racial discrimination and stressful life events together). However, there was a significant, positive correlation between the balance of pro- and anti- inflammatory cytokines and racial discrimination over and above the effects of stressful life events. Higher exposure to racial discrimination was associated with more pro- and anti- inflammatory cytokines being expressed therefore leading to more inflammation and potentially contributing to health disparities in Black Americans.

Introduction

Black Americans have greater prevalence and earlier onset of disability and chronic illness and significantly lower life expectancy than other ethnic groups (Williams, 2012). Racial discrimination and social inequalities have been implicated in many health disparities within the Black American community. Racial discrimination is considered a fundamental cause of adverse health outcomes for ethnic minorities and inequities in health. Racial and ethnic minorities have health that is worse overall than the health of White Americans. A lot of this is based on the continual years of racial and social injustice and a formidable challenge to equitable health care (Noonan et al., 2016). Stress is considered one of the most significant health problems in modern society. It can be characterized as any changes in the homeostasis of an individual that require an adaptive response. Contextual stressors, such as financial strain, victimization and racial discrimination encounters, have also been shown to contribute to biological dysregulation and increased susceptibility to infectious and chronic disease, and inflammation is seen as a key mechanism that connects such stressors to health outcomes (Beach et al., 2019).

Chronic inflammation contributes to several of the diseases showing racial disparities, and racial differences in stress exposure that stimulate pro-inflammatory processes that may contribute to variability in health outcomes. In the case on chronic stress, it has been suggested that prolonged activation of the stress response can promote a state of glucocorticoid resistance that involves an inability of cortisol to inhibit pro-inflammatory signaling pathways, leading to a loss of normal negative association between cortisol concentration and various indicators of inflammatory immune activation.

In recent years, behavioral scientists have documented the importance of social adversity as a predictor of systemic inflammation (Browning, Cagney, & Iveniuk, 2012; Cole, 2014).

Inflammation is the body's response to a threat, whether it's a foreign invader, such as a bacteria, virus, or cancer, or even a psychological or emotional stressor. In response, the immune system sends out an army of chemicals, called pro-inflammatory cytokines, to attack the invaders. One reason why stress is proposed to result in inflammation is through dysregulation of the stress hormone cortisol (Stevens & Wands, 2012). Cortisol can help control blood sugar levels, regulate metabolism, help reduce inflammation, and assist with memory formulation. All of these functions make cortisol a crucial hormone to protect overall health and well-being. A physiologic stress response may be evoked by fear or perceived threat to safety, status, or well-being and elicits the secretion of epinephrine and norepinephrine and neuroendocrine hormones (cortisol) to promote survival. However, although a stress-induced increase in cortisol secretion is adaptive in the short term, excessive or prolonged cortisol secretion may have crippling effects, both physically and psychologically (Stevens & Wands, 2012). Cortisol in turn is not only important in mobilizing resources to respond to the stressor, but also plays a fundamental role in limiting the inflammatory response and the further production of cytokines.

Elevated levels of inflammatory cytokines coupled with an insufficient regulating response are thought to be prompted by the contextual stressors as a conserved response to adversity (Cole, 2014). As a mechanism that increases their vulnerability to cardiometabolic risk and poor health, psychosocial stressors which disproportionately affect Black Americans have been suggested. Cardiometabolic risks include insulin resistance, glucose intolerance, dyslipidemia, and hypertension (Brody et al., 2016). Blacks have a 30% greater chance of dying from cardiovascular disease (Department of Health and Human Services, 2016) and a twofold greater risk of diabetes (Konen, Summersom, Bell, & Curtis, 1999). Chronic illness and

disability place a heavy burden upon Black families and communities, and it appears that matters are getting worse, not better, over time (Geronimus & Thompson, 2004).

Threat, Inflammation, and “Weathering” in Black Americans

Explanation of the relation between adversity and inflammation emphasizes the evolutionary development of the immune system to address threatening conditions (Simons et al., 2018). In the evolution of humans, we are adapted to respond to an environment characterized by heightened stress by increasing the body’s pro-inflammatory response. This response is involved in helping to heal wounds and fight off bacterial infections and would be adaptive by complementing automatic behavioral fight-or-flight stress responses.

It is important to note that the health risk inequities experienced by Black Americans likely stem at least in part from contextual stress associated with economic hardship. “Social environments that pose a persistent threat of hostility, denigration, and disrespect promote chronically high levels of inflammation. This is, of course, everyday life for members of ethnic minorities living in a racially charged society” (Simons et al., 2018, p. 1994). Consistent with this reasoning, an emerging area of research has focused on racial discrimination as a qualitatively unique source of psychosocial stress that Black Americans face. Beyond a risk factors perspective, the “weathering hypothesis” of Geronimus and colleagues (Geronimus, 1992; Geronimus et al., 2006; Geronimus & Thompson, 2004) emphasizes the health inequality experienced by Blacks in a society where they suffer social, economic, and political segregation. Although health risk behaviors exert some influence, the fundamental explanation for health inequalities in the US is a racial divide where Black Americans occupy a marginalized, stigmatized, and subordinate status in relation to Whites. The weathering theory considers Black Americans' high levels of illness and disability as a reaction to social inequalities and everyday

slights, stereotypes, and other challenges to one's identity comprising the Black experience (Williams & Mohammed, 2008). Consistent with this hypothesis, several studies have reported a relation between discrimination and poor health outcomes among Black Americans. Exposure to racial discrimination was found to be correlated with a variety of health-relevant biological processes in Black American Adults, including glucocorticoids, pro-inflammatory cytokines and other inflammatory markers. Elevations in circulating markers of inflammation (e.g., C-reactive protein [CRP], interleukin 6 [IL-6], tumor necrosis factor α [TNF α]) have been associated with cardiovascular disorders (CVD), type II diabetes, osteoporosis, rheumatoid arthritis, Alzheimer's disease and most cancers (Franceschi & Campisi, 2014). There is some evidence that Blacks tend to have higher levels of inflammation than whites (Paalani et al., 2011). These results point to the significance of exploring the link between racism and high inflammation as an avenue for understanding Black Americans' higher prevalence of diseases.

There have been problems in the way that past research has conceptualized inflammation. In recent years, a profusion of studies has investigated the potential link between social adversity and elevated inflammation. Whether adversity was defined as racial discrimination or some other stressful condition, inflammation has almost always been assessed using a single marker usually CRP or interleukin-6 (Beach et al., 2019). Further, the system consists of both proinflammatory and anti-inflammatory factors, with the latter serving to regulate and limit the inflammatory process. Many studies have indicated that it is the balance of pro- and anti-inflammatory cytokines that is crucial for health. In contextual stress and inflammation, you can characterize the response broadly by assessing a number of pro- and anti-inflammatory cytokines using a "multiplex" assessment approach (e.g., Tighe, Ryder, Todd, & Fairclough, 2015). Inadequate concentrations of anti-inflammatory cytokines can result in excess inflammation, potentially

producing harm to the host. For example, IL-4, IL-10, and IL-13 all have strong inhibitory effects on proinflammatory cytokines by suppressing monocyte-derived cytokines such as IL-1, TNF, IL-6, IL-8, and macrophage inflammatory protein 1 (Beach et al., 2019). Because anti-inflammatory cytokines can temper the impact of proinflammatory cytokines, their presence may be as important or more important than proinflammatory cytokines alone in capturing the potential for inflammation to influence health (Beach et al., 2019). Likewise, a lack of balance between pro- and anti-inflammatory cytokines has been linked to onset of a variety of chronic illnesses (e.g., see Andargie & Ejara, 2015).

Current Study Aims

Given the potential importance of the balance of pro- and anti-inflammatory cytokines, the current investigation assessed inflammation using a multiplex assay to capture a number of cytokines and assess both pro- and anti-inflammatory cytokines (Franceschi & Campisi, 2014; Morrisette-Thomas et al., 2014) and combine them into an index of the balance of pro- to anti-inflammatory activity.

As outlined by Simons et al. (2018), compelling support for a weathering hypothesis would require evidence that (a) across the life course, race-related stressors trigger biological changes predictive of chronic illness and disability; (b) this association is maintained after controlling for traditional health risk factors such as SES, smoking, poor diet, and lack of health care; and (c) the impact of race-related stressors on pathological biological processes is greater than that of traditional health-risk factors. While meeting all of those goals is beyond the scope of the current study, the aims are mindful of how results could contribute to testing a weathering hypothesis.

The overarching aim of this study is to examine whether racial discrimination is associated with the balance of pro- and anti-inflammatory cytokines in Black American adults.

More specifically, it is hypothesized that, over and above the contribution of any covariates:

(1) There will be statistically significant, positive correlations between the balance of pro- and anti-inflammatory cytokines and social adversity (racial discrimination and stressful life events).

In other words, as the set of adversity increases so does inflammation.

(2) The association between racial discrimination and the balance of pro- and anti-inflammatory cytokines will be statistically significant over and above the effects of stressful life events.

Methods

Participants

Participants were drawn from the IRB-approved Families of Richmond, VA (FoR-VA) and Families of Richmond, VA Extension (FoR-VA-X) studies. The FoR-VA study took place in Richmond, VA in the research wing of the community-based center of Psychological Services and Development at Virginia Commonwealth University. The research team was given a list of children ages 5-12 who had been seen at the VCU Medical Center for well-child or asthma focused visits during the last year. From the list, only those living in low-income zip codes were chosen to be recruited by phone. Recruitment flyers were used in schools and community centers in these zip codes as well. Inclusion criteria included a child aged 5-12 who had asthma (or didn't) and had no diagnosed developmental disabilities that would preclude meaningful participation; parents and children also needed to speak English. At the time of consent, parents indicated whether they were willing to be contacted and invited to participate in future studies. Those who said yes, and whose children did not have asthma, were later called and invited to participate in the FoR-VA-X study. The FoR-VA-X study took place in the research center of

the Children's Hospital of Richmond (CHOR). This study utilized FoR-VA and FoR-VA-X data from adults (parents) from whom discrimination, stress, and cytokine data was available ($n=22$).

Procedures

Participants in the FoR-VA study came to a Psychology Department building near the VCU campus. After obtaining informed consent and assent, the parent and child completed the questionnaires, interviews, and serum samples during their visit. For the purpose of this study, the Experience of Discrimination (EOD), Stressful Life Events Checklist (SLECC), Health Behaviors, and demographic measures were analyzed. Questionnaire measures were administered interview style (i.e., read aloud by a researcher who recorded verbal responses) to children and to parents, unless parents expressed a strong desire to complete them independently.

Of the 96 families who participated in FoR-VA, 40 were eligible to participate in the FoR-VA-X study and 27 returned to the research center in the Children's Hospital of Richmond for the FoR-VA-X study. Adults and children completed questionnaires and gave blood samples via venipuncture. Phlebotomists collected peripheral blood into standard 6ml EDTA purple-top tubes from 26 adults. Whole blood was delivered to the local lab following each family visit. Lab technicians spun each EDTA tube and aliquoted 400ul of plasma, which was then frozen at -80°C until ready for BioPlex batch analysis. Of the 27 participants enrolled, one did not identify as Black or African American and we were missing data from four others, yielding a final sample size of 22 for this study.

Measures

Demographic Characteristics (FoR-VA study): Parents reported socio-demographic and family characteristics, including their sex, race and ethnicity, and education, as well as their family's household composition and income.

Potential Covariates (FoR-VA-X study): *Physical measurements* were taken by the researcher: participant height (inches), weight (pounds), waist circumferences (inches), and forehead temperature (degrees Fahrenheit). *Participant age and smoking status* were self-reported (i.e., participants were asked whether they smoked or used tobacco products, to which they responded yes or no).

Stressful Life Events Checklist (SLECC; FoR-VA-X study). The SLECC adapted from the Life Events Checklist (LEC) which included 30 items intended to measure both discrete and chronic life events/stressors experienced by children and families, such as a family member being sick, having to move, or having serious financial problems in the family. Primary caregivers reported whether or not the child had experienced each stressor or event in the past six months. Studies to date using this scale, and the slightly adapted SLECC (Caserta et al., 2008) have used a total score (sum of events experienced). Since the development of the SLECC, it has been adapted in a few ways. First it was expanded to 32 items (e.g., Magnus et al., 1999) then to 34 items, which includes two “blanks” to record other significant life events that respondents endorse (Caserta et al., 2008). Each response was scored 0 if the event did not happen and 1 if it did; scores were then summed to create an overall index of stressful life events in which higher scores indicate more stressful events experienced.

Experience of Discrimination (FoR-VA study). The psychometrically established “Experiences of Discrimination” (EOD) measure was used to measure participants’ self-reported experiences of discrimination (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). For this study, the frequency score was utilized. Participants were asked, “Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of your race, ethnicity, or color?” Nine

situations (e.g., “getting housing” and “from the police or in the courts”) were rated as YES/NO and then as happening “Once,” “Two or three times” and “4 or more times.” Responses were scored per standard EOD protocol by assigning the value of 0 to “never,” 1 to “once,” 2.5 to “2-3 times,” and 5 to “4 or more times,” and then summing across items to yield a total score in which higher values represent more experiences of discrimination.

Cytokines (FoR-VA-X study). The Bio-Plex Pro Human Cytokine Assays system was used to measure ten cytokines that have been identified in psychological studies as important in inflammatory responses and immune system regulation IL-1 β , IL-2, IL-4, IL-5, IL-6, IL-10, IL-12, IL-13, IFN- γ , and TNF- α . The assays utilize a combination of fluorescent flow cytometry and enzyme-linked immunosorbent assay (ELISA) technology. Cytokines and chemokines are extracellular mediators and regulators within a signaling network between cells and are key modulators of inflammation, participating in acute and chronic conditions via a complex of interactions.

Cytokines were batch processed and run in duplicate. Cytokine concentrations were derived from 5PL standard curves and reported in pg/ml. Following procedures used in past human research (e.g., Beach et al., 2019) cytokines were eliminated if they were present at out-of-range levels in 50% or more of samples. Of the five remaining cytokines, IL-1 β , IL-6, IL-12, and TNF- α were classified as primarily pro-inflammatory and IL-13 as anti-inflammatory. The following equation represents the calculation of a ratio of pro-inflammatory (numerator) to anti-inflammatory (denominator) cytokines:

$$\frac{(IL-1\beta + IL-6 + IL-12 + TNF-\alpha)}{IL-13}$$

IL-13

Thus, higher scores indicate a greater pro-inflammatory response as well as lack of a potentially mitigating anti-inflammatory response.

Data Analysis

Data analysis was conducted using SPSS 26.0 software from IBM (2019). Variable skewness and kurtosis were examined, and results showed that all measures were normally distributed (i.e., skewness < 2.0 , kurtosis < 7.0 ; Tabachnick and Fidell, 2012). Prior to hypothesis testing, two-tailed bivariate correlation analysis and independent samples t-tests were run for each potential covariate with the outcome variable to guide the selection of covariates for inclusion.

To test study hypotheses, a hierarchical regression analysis was conducted in which the pro-anti-inflammatory ratio was entered as the dependent variable, covariates (sex, height, and weight) were entered at Step 1, and stressful life events and frequency of discrimination were entered at Step 2. Hypothesis 1 would be supported if – at Step 2 – the overall model was statistically significant. Hypothesis 2 would be supported if, at Step 2, the association between racial discrimination and the balance of pro- and anti-inflammatory cytokines was statistically significant over and above the effects of stressful life events.

Results

Participants were between the ages of 26 and 67 ($M_{age} = 38.79$, $SD = 11.23$); 96% identified as female and 4% (one participant) identified as male. Reflecting the study recruitment strategy, nearly all families (91%) had yearly incomes that would be classified below the national poverty level: families reported annual family incomes as follows: 0-5999 (33.3%), 6000-11999 (28.6%), 12000-23999 (23.8%), 24000-35999 (14.3%). Years of education reported ranged from “5” to “17 or more” ($M = 12.10$, $SD = 2.86$) with 22.7% having a high school diploma or GED, 40.9% having below a high school diploma, and 36.4% having education beyond high school. While collecting the biometrics forehead temperatures were taken. No participants had a forehead temperature higher than 37°C at the time of blood draw.

Participants reported that they had faced racial discrimination and their families experienced stressful life events. Experiences of Discrimination (EOD) scores ranged from 28 to 42.5 ($M = 35.52$, $SD = 4.31$) out of a possible score range of 0 - 45. SLECC scores ranged from 1 to 18 ($M = 8.04$, $SD = 4.23$) out of a possible score range of 0 - 34.

The pro-anti ratio was not significantly associated with waist circumference ($R = -.258$, $p = .246$) or smoking status ($t = -.453$, $p = .668$). It was marginally associated with both height ($R = -.380$, $p = .081$) and weight ($R = -.402$, $p = .063$). To be conservative and given the wide age range plus the inability to properly test for differences between female and (the one) male participants, participant age and sex were included as covariates along with height and weight.

Table 1 shows the results of the regression analysis. Hypothesis 1 was not supported, $F(6,15) = 1.67$, $p < .05$). Hypothesis 2 was supported; higher frequency of racial discrimination was significantly associated with larger pro- anti-inflammatory cytokine ratio over and above the effects of stressful life events and covariates ($t = 2.164$, $p = .047$, $\beta = .479$).

Discussion

We hypothesized direct associations between the balance of pro- and anti- inflammatory cytokines and social adversity (i.e., stressful events and racial discrimination together). In addition, we hypothesized that there would be an association between racial discrimination and the balance of pro- and anti- inflammatory cytokines over and above the effects of stressful life events. The hypothesized model of direct associations between pro- and anti- inflammatory cytokines and social adversity was not statistically significant. Stressful life events were not directly associated with inflammation. However, there was a significant, positive correlation between the balance of pro- and anti- inflammatory cytokines over and above the effects of stressful life events. Identifying factors related to inflammation among Black American adults may yield insights into mechanisms underlying racial health disparities. High levels of interpersonal racial discrimination and the development of a positive racial identity operate jointly to determine inflammation levels that may have been found to forecast chronic diseases such as coronary disease and stroke (Lewis et al., 2010).

Although the results to this study are preliminary, if this study was replicated it indicates that discrimination may play an important role in inflammation and therefore in health outcomes in Black Americans. By focusing on just Black Americans, we were able to examine a relevant construct (i.e., racial discrimination) in an at-risk group that is disproportionately exposed to these experiences. This could help lead to more in-depth research studies.

However, in this study the effects of stressful life events appeared to be minimal. Since stress has been associated with inflammation in past studies, the fact that we did not find that most likely has something to with our study's methodology or sample size. For example, many of the items on the SLECC are events that are stressful for children (e.g., child having few

clothes to wear) that arguably might not be as stressful for these parents. It may be necessary to follow up to see which items on the SLECC were endorsed by participants in this study. In addition, other markers of stress (e.g., those associated with poverty like food and housing instability) may be more important to these families because almost all of them (91%) live in poverty.

Limitations and Future Directions

The main limitation of this study was our sample size. Having such a small sample size makes it difficult to know if the lack of findings were true or due to a Type II error. Future studies with larger samples would be able to properly test the study hypotheses more in depth and be able to look at cytokine levels individually instead of (or in addition to) grouping them into Pro- and Anti- Inflammatory Cytokines.

Another limitation was the method of recruitment. The study was aimed to recruit families living in high-poverty, urban areas. Therefore, our results had a higher percentage of Black Americans and may not generalize to other Black American families. Also, our research team did not have any Spanish speakers. So, for the participants that were Black and Hispanic/Latino that may have identified as Black American but spoke predominately Spanish were not able to be recruited. Research is also needed to identify the mechanisms and processes that give rise to those situations where whites are more adversely affected by risk factors than racial minorities.

Furthermore, because this study is cross-sectional, we do not know the direction of effects for the relationship between racial discrimination and inflammation in Black Americans. Future studies should conduct longitudinal research to examine whether stress/racial discrimination is related to increases or fluctuations in inflammation and cytokine levels over

time. Having a longitudinal study over a period of time would allow examination of the association of contextual stress (i.e., financial strain, victimization, and racial discrimination) with inflammation. During this study, we can compare high poverty, Black American adults to more affluent Black American adults who are less stressed but also experiencing some form of racial discrimination. It would be informative to examine each cytokine separately and see how it relates to stress and racial discrimination in both groups.

Additional steps could be taken in this research to see if there is any correlation between the caregiver and the child when it comes to inflammation, for example inheritability and shared contextual stress. It would also be beneficial to understand when Black American children start to experience “weathering” and take into account the compounding of risk factors over a period of time. Future research could profitably explore the extent to which these patterns could reflect weaker, habituation effects for blacks due to earlier exposure and elevated levels of exposure, and/or the conditions under which the presence of cultural, SES, psychosocial, and religion and other resources can weaken the negative health effects of certain risk factors. For instance, high levels of interpersonal racial discrimination and the development of a positive racial identity operate jointly to determine inflammation levels that may have been found to forecast chronic diseases such as coronary disease and stroke (Lewis et al., 2010).

In addition, we can look at specific age groups within Black Americans. Older Black Americans in particular are an important sub-population to consider in studies of discrimination and health because these individuals came of age prior to the United States civil rights movement, at a time when discriminatory treatment against Black Americans was legally sanctioned. Opportunity now exists to cleave together a comprehensive understanding of the ways in which discrimination has harmful effects on health. This work would be furthered by

interdisciplinary research collaborations, for example between psychologists studying stress and immunologists conducting research through systemic (e.g., physiological and cellular) lenses. This could also promote research to better understand the link between experiences of stress and inflammation, for example through dysregulations in cortisol or other body systems. Diet is also an important factor in regulation of pro-inflammatory cytokines. The impact that inflammation in the gut is having on our general health is substantial. When you consider that a significant portion of the immune system is dedicated to maintaining a host relationship with the gut microbiome, it may be unsurprising that gut microbiota is heavily involved in local inflammatory responses to acute injury and/or infection. Understanding the microbiota similarities and differences across races and ethnicities has the potential to advance approaches aimed at personalized microbial discovery and treatment, particularly those involved in health disparities. Inclusion of individuals from diverse cultural and social backgrounds in microbiome studies is a key step in advancing our understanding of health disparities. This is especially true in cases where investigators can link prevalence differences in a specific health condition or disease with identifiable population groups. Uncovering the role of the microbiome in health disparities could enhance our understanding of why some populations have poorer survival rates, greater severity of disease, and overall elevated disease risks compared to others.

The microbiota may assist in regulating immune responses including tolerance and may play a protective role in acute inflammatory responses. Thus, factors such as diet, lifestyle, and other health-related behaviors likely influence ethnic differences in health and combined with other exposures to potentially modify the microbiome over time, resulting in poorer health outcomes. Inclusion of individuals from diverse cultural and social backgrounds in microbiome studies is a key step in advancing our understanding of health disparities. This is especially true

in cases where investigators can link prevalence differences in a specific health condition or disease with identifiable population groups (Liu, 2017). Uncovering the role of the microbiome in health disparities could enhance our understanding of why some populations have poorer survival rates, greater severity of disease, and overall elevated disease risks compared to others. Also, exploring the microbiome and the differences therein is likely to be important in efforts to reduce and eliminate health disparities while shedding light on how social and environmental exposures interact with biology to affect disease risk and outcome. An individual's microbiome is likely to be an important contributor to certain health disparity diseases and conditions. Researchers studying the microbiome have captured limited information on socioeconomic, psychosocial, cultural, and behavioral factors as well as diet in diverse study populations.

Implications

The findings of the current study suggest that higher exposure to racial discrimination may contribute to physiological wear and tear on the body, leading to alteration of immune function. These findings, if replicated in larger, longitudinal studies, would add to the literature by suggesting risk factors for disparities in health outcomes. This could influence what is targeted in interventions that aim to improve Black American health outcomes by emphasizing repeated exposure to racial discrimination.

References

- Andargie, T. E., and Ejara, E. D. (2016). Pro- and Anti-inflammatory Cytokines in Visceral Leishmaniasis. *Journal of Cell Science and Therapy*, 06(02). doi: 10.4172/2157-7013.1000206
- Beach, S. R. H., Lei, M. K., Simons, R. L., Barr, A. B., Simons, L. G., Cutrona, C. E., and Philibert, R. A. (2019). Perceived relationship support moderates the association of contextual stress with inflammation among African Americans. *Journal of Family Psychology*, 33(3), 338–348. doi: 10.1037/fam0000509
- Brody, G. H., Yu, T., Chen, E., Ehrlich, K. B., and Miller, G. E. (2018). Racial discrimination, body mass index, and insulin resistance: A longitudinal analysis. *Health Psychology*, 37(12), 1107–1114. doi: 10.1037/hea0000674
- Browning, C. R., Cagney, K. A., and Iveniuk, J. (2012). Neighborhood stressors and cardiovascular health: Crime and C-reactive protein in Dallas, USA. *Social Science and Medicine*, 75(7), 1271–1279. doi.org/10.1016/j.socscimed.2012.03.027
- Caserta, M. T., O'Connor, T. G., Wyman, P. A., Wang, H., Moynihan, J., Cross, W., Tu, X., and Jin, X. (2008). The associations between psychosocial stress and the frequency of illness, and innate and adaptive immune function in children. *Brain, behavior, and immunity*, 22(6), 933–940. doi.org/10.1016/j.bbi.2008.01.007
- CDC Health, United States. (2016): Chartbook on long-term trends in health. US Department of Health and Human Services. Retrieved from www.cdc.gov/nchs/data/abus/abus16.pdf
- icon

Cole, S. W. (n.d.). Human Social Genomics. Retrieved from

<https://journals.plos.org/plosgenetics/article?id=10.1371/journal.pgen.1004601>

Franceschi, Claudio, Campisi, and Judith (2014, May 8). Chronic inflammation (inflammaging)

and its potential contribution to age-associated diseases. Retrieved from

<https://doi.org/10.1093/gerona/glu057>

Geronimus A.T. (1992). The weathering hypothesis and the health of African American women and infants: evidence and speculations. *Ethnicity Disease*, (2), 207–221

Geronimus, A. T., Hicken, M., Keene, D., and Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *American journal of Public Health*, 96(5), 826–833. doi: 0.2105/AJPH.2004.060749

Geronimus, Arline and Thompson, J. (2004). To denigrate, ignore, or disrupt: racial inequality in health and the impact of a policy-induced breakdown of African American communities. *Du Bois Review*, (1). (247 – 279). doi:10.1017/S1742058X04042031.

Konen, J.C., Summerson, J.H., Bell, R.A., and Curtis, L. G. (1999). Racial differences in symptoms and complications in adults with type 2 diabetes mellitus. *Ethnicity and Health*, 4 (1-2), 39-49. doi: 10.1080/13557859998182

Krieger, N., Smith, K., Naishadham, D., Hartman, C., and Barbeau, E. M. (2005). Experiences of discrimination: Validity and reliability of a self-report measure for population health research on racism and health. *Social Science and Medicine*, 61(7), 1576–1596. doi:10.1016/j.socscimed.2005.03.006

Lewis, T. T., Aiello, A. E., Leurgans, S., Kelly, J., and Barnes, L. L. (2010). Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein

- levels in older African-American adults. *Brain, behavior, and immunity*, 24(3), 438–443.
doi: 10.1016/j.bbi.2009.11.011
- Liu R. T. (2017). The microbiome as a novel paradigm in studying stress and mental health. *The American psychologist*, 72(7), 655–667. doi.org/10.1037/amp0000058
- Morrisette-Thomas, V., Cohen, A. A., Fülöp, T., Riesco, É., Legault, V., Li, Q., Milot, E.,
Dusseault-Bélanger, F., and Ferrucci, L. (2014). Inflamm-aging does not simply reflect
increases in pro-inflammatory markers. *Mechanisms of ageing and development*, 139,
49–57. doi.org/10.1016/j.mad.2014.06.005
- Noonan, A. S., Velasco-Mondragon, H. E., and Wagner, F. A. (2016). Improving the health of
African Americans in the USA: an overdue opportunity for social justice. *Public health
reviews*, 37, 12. doi.org/10.1186/s40985-016-0025-4
- Paalani, M., Lee, J. W., Haddad, E., and Tonstad, S. (2011). Determinants of inflammatory
markers in a bi-ethnic population. *Ethnicity and Disease*, 21(2), 142–149.
- Simons, R. L., Lei, M.-K., Beach, S. R. H., Barr, A. B., Simons, L. G., Gibbons, F. X., and
Philibert, R. A. (2018). Discrimination, segregation, and chronic inflammation: Testing
the weathering explanation for the poor health of Black Americans. *Developmental
psychology*, 54(10), 1993–2006. doi.org/10.1037/dev0000511
- Stephens, M. A., and Wand, G. (2012). Stress and the HPA axis: role of glucocorticoids in
alcohol dependence. *Alcohol research: current reviews*, 34(4), 468–483.
- Tabachnik, B. G. and Fidell, L. S. (2012). *Using Multivariate Statistics* (6th edition). Pearson.
- Tighe, P. J., Ryder, R. R., Todd, I., and Fairclough, L. C. (2015). ELISA in the multiplex era:
Potentials and pitfalls. *PROTEOMICS - Clinical Applications*, 9(3-4), 406–422. doi:
10.1002/prca.201400130

Williams, D. R., and Mohammed, S. A. (2008). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, 32(1), 20–47. doi:

10.1007/s10865-008-9185-0

Williams, D. R., Priest, N., and Anderson, N. B. (2016). Understanding associations among race, socioeconomic status, and health: Patterns and prospects. *Health psychology*, 35(4), 407–411. doi.org/10.1037/hea0000242

Tables

Table 1

Regression Analysis of Stress Predicting the Pro- Anti-Inflammatory Cytokine Ratio

Level	<i>t</i>	<i>p</i>	β	<i>F</i>	<i>df</i>	<i>p</i>	Adj <i>R</i> ²
Covariates – Overall Model				1.113	4,17	.383	.021
Age	-.031	.975	-.007				
Sex	-.626	.540	-.171				
Weight	-1.134	.273	-.371				
Height	-.780	.446	-.216				
Stress – Overall Model				1.666	6,15	.197	.160
Stressful life events	.874	.396	.206				
Racial discrimination	2.164	.047	.479				

Note: Results for Hypothesis 1 and Hypotheses 2 are in bold text

Vita

Takia Williams was born on October 17, 1995, in Amelia Court House, Virginia. She graduated from Amelia County High School in Amelia, Virginia, in 2014. She received her Bachelor of Science in Biology from Alderson Broaddus University in 2017. She began the Microbiology and Immunology program at Virginia Commonwealth University in August 2018 and will be receiving her Master of Science degree in Microbiology and Immunology in May 2020. Takia is currently on track to apply to Veterinary School to obtain her Doctor of Veterinarian Medicine degree.