UNIVERSITY OF CALIFORNIA,

IRVINE

Intergenerational associations between parental adversity and offspring health outcomes in African-American families

DISSERTATION

submitted in partial satisfaction of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

in Psychology and Social Behavior

by

Josiah A. Sweeting

Dissertation Committee:

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2022

DEDICATION

To

my parents, sisters, and many loved ones

in recognition of their unwavering support

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ACKNOWLEDGEMENTS

To say my journey to a Ph.D. has been a truly wild, unpredictable, and LONG ride would be an understatement to say the absolute least. Fortunately, I have had the most meaningful and vital support system at every step of the way; the adage “it takes a village” could not capture my situation more perfectly. The individuals who I have been blessed to have in my corner have nurtured me as a human being while also continually pushing me to evolve both personally and professionally. Everything I have accomplished is a culmination of the constant dedication, love, and support that my village has given me, and my only hope is that I have made every single one of them proud.

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VITA

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PUBLICATIONS

Sweeting, J. A., Akinyemi, A. A., & Holman, E. A. (2022). Parental preconception adversity and offspring health in African Americans: A systematic review of intergenerational studies. *Trauma, Violence, & Abuse,* 0, 1-16. doi:10.1177/15248380221074320

Sweeting, J. A., Garfin, D. R., Holman, E. A., & Silver, R. C. (2020). Associations between exposure to childhood bullying and abuse and adulthood outcomes in a representative national U.S. sample. *Child Abuse & Neglect, 101, 104048*. doi:10.1016/j.chiabu.2019.104048

Lacey, T. E., **Sweeting, J.**, Kingston, R., Smith, M., & Markham, C. M. (2018). Neuropeptide Y impairs the acquisition of conditioned defeat in Syrian hamsters. *Neuroscience Letters, 690,* 214-218. doi: 10.1016/j.neulet.2018.09.049

Jones, N. M., Wojcik, S. P., **Sweeting, J.**, & Silver, R. C. (2016). Tweeting negative emotion: An investigation of Twitter data in the aftermath of violence on college campuses. *Psychological Methods, 21,* 526-541. doi: 10.1037/met0000099

PRESENTATIONS

Acevedo, A. M., Marshburn, C. K., **Sweeting, J.**, Williams, D. P., & Thayer, J. F. (2020, March). Invited participant, annual meeting of the American Psychosomatic Society symposium "*Understanding discrimination in the context of the Generalized Unsafety Theory of Stress*," Long Beach, CA (cancelled due to COVID-19).

Sweeting, J., Akinyemi, A., & Holman, E. A. (2019, March). *Understanding physical health disparities in African Americans and Native Americans: A systematic review of the role of stress/trauma*. Poster presented at the annual meeting of the American Psychosomatic Society, Vancouver, BC.

Pratt, J. B., Davison, M., **Sweeting, J.**, & Villalta, S. (2017, June). Invited participant, annual Law and Society Conference panel "*Look at 13th Documentary: Millennial Scholars at UC Irvine*," Mexico City, Mexico.

Sweeting, J., Garfin, D. R., Holman, E. A., & Silver, R. C. (2016, May). *Long-term effects of exposure to bullying and family violence in childhood*. Poster presented at the annual meeting of the Western Psychological Association, Long Beach, CA.

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ABSTRACT OF THE DISSERTATION

Intergenerational associations between parental adversity and offspring health outcomes in African-American families

By

Josiah A. Sweeting

Doctor of Philosophy in Psychology and Social Behavior

University of California, Irvine, 2022

Roxane Cohen Silver, Distinguished Professor of Psychological Science, Chair

Across two studies, this dissertation examined how life adversity for African Americans contributes to their health outcomes and how adverse experiences occurring in one generation may be associated with the health outcomes of subsequent generations. In Chapter 2, a systematic review exploring the empirical literature on associations between parental preconception adversity and offspring physical health in African-American families was conducted. Thirty-eight articles representing 30 independent studies met inclusion criteria. Ultimately, twenty-five studies (83%) reported that parental preconception adversity was associated with child health; six studies (20%) reported that parental preconception adversity was not associated with at least one offspring outcome; several studies reported both. Only six studies (20%) reported an association specific to African Americans. In Chapter 3, a dyadic sample of African-American mothers and adult children (N = 57 dyads) was used to investigate whether several types of maternal adversity were related to their child's health, as well as if the specific timing of adversity played a role in offspring health outcomes. Findings showed that greater maternal preconception general adversity (IRR, 1.05; 95% CI: 1.00-1.11) was associated with a

higher number of doctor-diagnosed offspring health ailments after controlling for adversity during other time periods and offspring adversity. Greater maternal post-conception law enforcement adversity was associated with better self-rated health in their offspring (unstandardized b = -.23, SE = .07, z = -3.10, p = .002). Taken together, these findings highlight the importance of both timing and type of maternal adversity when exploring links to offspring health. Findings also demonstrate how maternal adversity can be linked to adult offspring health while controlling for offspring's own adversity exposure. The significance of utilizing a more fine-grained approach to examining links between parental adversity and offspring health in African Americans is discussed.

Chapter 1: Introduction

Introduction

Research has identified health disparities across several demographic indicators, but among the most widely studied factors in the literature are the concepts of ethnicity and race. Although often used interchangeably, it is important to distinguish the notion of ethnicity from race. Ethnicity encompasses the social science construct referring to one's chosen cultural identity, and members of this group tend to shape this identity through learning as opposed to biological predispositions (Valdez & Golash-Boza, 2017). In contrast, race generally describes the biological, observable physical characteristics of an individual (Valdez & Golash-Boza, 2017) and this construct represents the topic of focus in this dissertation.

Health disparities have been identified extensively for African Americans in relation to whites. For example, African Americans tend to have poorer cardiovascular and endocrine health across several indicators including diabetes mellitus, hypertension, obesity, and stroke (Benjamin et al., 2017; Carnethon et al., 2017; Cunningham et al., 2017; Howard et al., 2017; Ogden, Carroll, Kit, & Flegal, 2014; Ogden et al., 2016). Furthermore, they tend to experience greater risk of certain infectious diseases compared to whites, including the Hepatitis C virus, Hepatitis B, pneumonia, and tuberculosis (Hall, Rosenberg, & Sullivan, 2018; Hayes et al., 2018; Kim et al., 2017; Yuen, Kammerer, Marks, Navin, & France, 2016; Zou et al., 2019). Overall cancer incidence, as well as a higher prevalence of several chronic conditions including asthma, Alzheimer's disease and dementia, HIV, and obstructive sleep apnea, have also been demonstrated in the literature for African Americans compared to whites (Allgood, Hunt, & Rucker, 2016; American Cancer Society, 2019; Chen & Panegyres, 2017; Cunningham et al., 2017; Mehta & Yeo, 2017; Ruiter, DeCoster, Jacobs, & Lichstein, 2010; Siddiqi, Hu, Hall, & CDC, 2015; Steenland, Goldstein, Levey, & Wharton, 2016).

Another important factor that has been linked to health status and strongly linked to race is socioeconomic status (SES). SES is a "complex and multi-dimensional concept comprising a range of factors encompassing economic resources, power and/or prestige that can influence health at different times in the life course, at different levels (e.g., individual, household, neighborhood)" (pg. 2, Williams, Priest, & Anderson, 2016). Previous work has suggested that individuals with a lower SES are significantly more likely to develop mental health problems compared to high SES individuals (Devenish, Hooley, & Mellor, 2017; Reiss, 2013; Sweeting, Garfin, Holman, & Silver, 2020) and have more physical health ailments, including several cardiovascular disease risk factors such as obesity and metabolic syndrome (Mozaffarian et al., 2016; Sweeting et al., 2020). Given the many health disparities identified for African Americans, SES may be another factor that tends to disproportionately contribute to their unfavorable health outcomes in several ways. For example, sizable differences have been observed in SES levels, with rates of college graduation being nearly twice as high for whites compared to African Americans (Williams et al., 2016). Furthermore, data on household wealth and assets from the 2016 census showed that African Americans had an average net worth of $14,100 compared to $187,300 for whites (U.S. Census Bureau, 2019). In relation to whites, African Americans also receive less income at the same education levels and have less purchasing power due to higher costs of goods and services in the residential settings where they disproportionately reside (Williams, Mohammed, Leavell, & Collins, 2010). This notion was reflected by the U.S. Census Bureau in 2018 when it identified African Americans as having the highest poverty rate at nearly double the rate of whites while also having the lowest real median household income of any racial group (U.S. Census Bureau, 2019a).

However, it is important to note that health disparities linked to SES differences within a given racial group are greater than the health disparities observed between racial groups (Adler, 2009). In other words, differences in health status and mortality between the most and least affluent people within any racial group are larger than the differences between different racial groups at equivalent levels of SES. For example, Adler (2009) explained that the difference in life expectancy at age 25 between white and African-American men was 4.4 years, but the difference between men with higher compared to lower incomes at the same age within African-American or white groups was nearly double that difference (7.9 and 8.6 years for whites and African Americans, respectively). Consequently, African Americans may experience health disparities when compared to whites as a function of their race, but SES differences may also account for a substantial portion of these disparities.

Potential explanations for health disparities

Researchers have highlighted several factors that can significantly impact health outcomes. Another research area in which substantial disparities for African Americans have been demonstrated is the experience of stress and trauma throughout the lifespan. Prior work has shown that African-American adults report a significantly higher prevalence and greater clustering of high stress scores compared to whites across community, financial, and relationship stress domains (Boardman & Alexander, 2011; Sternthal, Slopen, & Williams, 2011). A wealth of findings from research studies have specifically highlighted the relatively high frequency at which this racial group is exposed to violence, both as witnesses and victims. In childhood, African Americans have been shown to have a higher risk of adverse experiences in the form of witnessing domestic violence, serious injury, or murder, being threatened with a weapon, and being held captive (Roberts, Gilman, Breslau, Breslau, & Koenen, 2011; Schilling, Aseltine, &

Gore, 2007). Based on data from the National Crime Victimization Survey, African Americans aged 12 and older are also more likely to report being a victim of a violent crime (Truman & Langdon, 2015).

Similar to the aforementioned link between race and SES in health outcomes, an association has also been identified with respect to stress and trauma exposure and health. Individuals from lower SES backgrounds may have less financial control over the environments in which they are able to reside. Furthermore, they may be exposed to greater sources of stress and trauma in the form of poorer quality neighborhoods and living conditions. This notion has been supported by several studies showing that low SES is associated with higher levels of perceived stress, being a victim of nonfatal violent crimes, and being a victim of homicide (Cohen & Janicki-Deverts, 2012; Lee, Coe, & Ryff, 2017; Ulmer, Harris, & Steffensmeier, 2012). For children, specifically, those in low-SES households tend to experience more adverse events, have fewer supportive interactions with parents, greater exposure to harsh parenting and interpersonal conflict, lower parental involvement in their education, and a greater likelihood of maltreatment (Evans & Kim, 2013; Hornby & Lafaele, 2011; Jonson-Reid, Drake, & Kohl, 2009; Topitzes, Pate, Berman, & Medina-Kirchner, 2016). In addition, having fewer economic resources negatively impacts family cohesion and hinders the formation of marriage, which has been identified as an essential component of financial stability and social support (Caughy et al., 2012; Watson & McLanahan, 2011). Based on U.S. Census Bureau data highlighting the substantial socioeconomic disparities they face compared to other racial groups (U.S. Census Bureau, 2019a; U.S. Census Bureau, 2019b), African Americans may experience greater stress and trauma exposure than whites due to their racial identity as well as their SES.

Beyond general stress, trauma, and violence exposure, historical trauma is another negative experience that has been identified as highly salient to the collective experience of African Americans in the United States. The notion of historical trauma was first conceptualized in the 1960's based on the widespread prevalence of persistent trauma among Holocaust survivors and their families following World War II (Sotero, 2006). This concept was later expanded and used to describe the cumulative emotional and psychological wounding of Indigenous Native Americans occurring across the lifespan and multiple generations that originated from exposure to massive group trauma experiences (e.g., enslavement, community massacres, forced relocation; Brave Heart, 1998; Brave Heart & DeBruyn, 1998; Evans-Campbell, 2008).

Building on these ideas, Sotero (2006) developed the historical trauma theory that provides a framework for examining how the life course of a trauma-exposed population compares to that of unexposed populations. This theory is based on four main assumptions: (1) mass trauma is deliberately and systematically inflicted upon a population by a subjugating, dominant population; (2) trauma is not limited to a single event, but continues over a prolonged period; (3) traumatic events reverberate throughout the population and create a universal trauma experience; and (4) the magnitude of the trauma experience hinders the affected population from its natural, historical course, resulting in a legacy of physical, psychological, and economic disparities persisting across generations (Sotero, 2006). Although most historical trauma research has focused on Holocaust survivors and Indigenous Americans, other scholars have posited that African Americans have also been exposed to historical trauma due to their history of intercontinental slavery, significant barriers to upward socioeconomic mobility, and continuing marginalization and vulnerability (Ruef & Fletcher, 2003).

The first assumption of historical trauma theory deals with a dominant group inflicting subjugation on a target group and identifies at least four elements necessary for sufficient subjugation: (1) overwhelming physical and psychological violence; (2) segregation and/or displacement; (3) economic deprivation; and (4) cultural dispossession. As a response to the drastic reduction in the American Indian population during the early 16th century, the "African Holocaust" was initiated by European colonizers and aimed to forcefully capture Africans to help cultivate their plantations (Burnside, 1997; Worth, 2001). Following their capture, Africans were chained together and subsequently transported across the Atlantic Ocean to the Americas (i.e., the Middle Passage) while routinely being exposed to inhumane conditions including starvation, human waste, decaying bodies, and several forms of abuse at the hands of crew members (Huggins, 1990; Leary, 2005). While the exact magnitude of deceased Africans is unknown, it is projected that close to two million deaths occurred during the Middle Passage due to unsanitary conditions, dehydration, and suicide (Eltis, 2007; Eltis & Richardson, 2010; Wolfe, 2013). Furthermore, many captives were led through a dehumanization process in which their flesh was branded with a hot iron and deemed as chattel while simultaneously stripping them of their identity.

After arrival to their destination in the Americas, Africans were then offloaded and subsequently sold into chattel slavery where they were often separated from their family members onto various plantations. Once on plantations, enslaved Africans were responsible for intensive labor that included farming various crops, but were not compensated as they had no rights, voice, or suffrage (Franklin & Moss, 2000; Worth, 2001). As a result of a Northern victory in the North American Civil War in 1861, the 13th Amendment legally abolished slavery and Africans were soon given full legal citizenship as well as the right to vote (men only) with

the 14th and 15th Amendments to the U.S. Constitution. However, these amendments were not honored or upheld by many southern states and African Americans still faced injustice despite the measures put in place to protect them (Rollins & Hicks, 2010). From 1877 through the 1970's, Jim Crow laws were enforced with the intentions of maintaining the racial divide by assigning a "less than European status" to African Americans (George, 2000). Consequently, African Americans during this period experienced extremely limited economic and political progress, widespread racial terrorism at the hands of white supremacist organizations, racial segregation in nearly every domain of daily life, and unethical policing practices that sparked the revival of slavery via convict leasing in the prison system (Christian, 1999; George, 2000; Smith, 1996).

Although the aforementioned overt acts of subjugation were annulled over time, their legacy remains in the form of discrimination and contributes to further disparities in stress and trauma exposure in the modern era. With respect to African Americans, discrimination has been widely studied and is generally defined as "the beliefs, attitudes, institutional arrangements, and acts that tend to denigrate individuals or groups because of their phenotypic characteristics or ethnic group affiliation" (Clark, Anderson, Clark, & Williams, 1999, p. 805). Discrimination is an adverse experience because it can encompass both acute and chronic events, occur on multiple levels, ultimately undermine positive views of the self, diminish social relationships, and interfere with overall quality of life. In addition to being an acute, interpersonal occurrence (e.g., being called a racial slur on the street), discrimination can also be a chronic stressor when there are recurring instances of mistreatment over prolonged periods, the discriminatory conditions do not change, and the discrimination produces other stress exposures (APA, 2017).

Lastly, discrimination can be a persistent stressor due to the limited resources that are available to address it.

In one of its most pervasive forms, cultural-level discrimination refers to the dissemination of attitudes regarding the relative privileges, rights, and status that should be granted to different groups (Harrell, 2000). Historically, these attitudes have been strongly influenced by mass media (e.g., newspapers, film, television, Internet) and have served as an influential way of establishing stereotypes about group members. For example, past work has suggested that ongoing negative portrayals of racial minority group members as lazier, more violent, and less intelligent contributes to the desire for distance from members of these groups (Brondolo, Libretti, Rivera, & Walsemann, 2012; Dixon, 2008). Consequently, this desire helps fuel the formation of policies at the institutional level that effectively exclude these groups across several domains and ultimately result in their unequal treatment. Institutional-level discrimination refers to the specific policies and procedures of institutions (e.g., education, government) that consistently result in unequal treatment for certain groups, including African Americans (Brondolo et al., 2012).

One notable example of institutional discrimination is residential segregation in which racial minorities are prevented from occupying spaces with ample resources, thus limiting their socioeconomic attainment and contributing to greater exposure to acute and chronic stressors (Williams, 2012; Williams et al., 2010). For example, one study demonstrated that white children with low-income backgrounds were significantly more likely than African-American children to live in middle-class neighborhoods with greater material and social resources (Drake & Jonson-Reid, 2014). In contrast, a substantial proportion of African-American children from low-income families were shown to live in areas where 40% or more of families were at the poverty level

(Drake & Jonson-Reid, 2014). Within high poverty areas, the local infrastructure generally cannot provide additional resources to help minimize gaps in individual income, assets, and education. Serving as another form of institutional discrimination and being partially a function of residential segregation, school segregation also further prevents African-American students access to the necessary educational and social resources that are readily available in other areas (Lankford & Wyckoff, 2006). Along with income, education is a key indicator of SES and has direct associations with health across several studies. For example, studies have found that adults without a high school diploma were nearly twice as likely to die over a five-year period compared to those with a professional degree, while between 1990 and 2008, life expectancy at age 25 among men and women with less than 12 years of education fell by more than three and five years, respectively (Olshansky et al., 2012; Ross, Masters, & Hummer, 2012).

Disparities in the experience of interpersonal-level, discrimination-based stress and trauma for African Americans have been well-documented and encompass the directly perceived discriminatory interactions between people occurring in their institutional roles or as private and public individuals (Krieger, 1999). A common domain in which these disparities can be observed is law enforcement. Evidence suggests that compared to whites, African Americans are more likely to be interrogated by the police, more likely to be arrested or incarcerated, and more likely to receive harsher sentences (Doerner & Demuth, 2010; Smith & Holmes, 2014). In addition to law enforcement, discriminatory practices have similarly been identified in the context of the labor market and have been shown to be a significant barrier to upward socioeconomic mobility. When compared to whites, African Americans have historically had a higher unemployment rate (Bureau of Labor Statistics, 2016) as demonstrated by several audit studies showing that they are less likely to be called for interviews and less likely to receive employment (Pager & Western,

2012; Pager, Western, & Pedulla, 2009). Finally, African Americans also experience discriminatory job loss in the form of layoffs and termination at significantly higher rates than their white counterparts who have the same or similar qualifications (Bell, Berry, Marquardt, & Galvin Green, 2013; Couch & Fairlie, 2010; Elvira & Zatzick, 2002).

Ultimately, the disparities in general, as well as historical trauma and stress exposure for African Americans, may also contribute to further inequality in bereavement experiences. For example, a study using two large national data sets investigated differences in the experience of losing a family member in the United States and reported that African Americans were significantly more likely to experience the death of a mother, father, sibling, spouse, and a child when compared to non-Hispanic whites (Umberson et al., 2017). They were also more likely to experience multiple family member deaths. Findings demonstrated that these differences in death exposure appeared early in childhood and remained significant into early and mid-adulthood. More recently, the novel coronavirus disease 2019 (COVID-19) has emerged as another significant contributing factor to the disproportionate rates at which African Americans experience mortality, and ultimately, bereavement. A systematic review highlighted that COVID-19 mortality was 105% higher in African Americans than in whites (Mude et al., 2021). Another study showed that death rates were nearly six times higher for more than 100 predominantly African-American counties when compared to mostly white counties (Alcendor, 2020). Similar rates have been found at the state level with places like Michigan reporting that mortality rates were nearly seven times higher for African Americans compared to whites (Zelner et al., 2021). This is important because bereavement is a well-known risk factor for adverse mental and physical health outcomes for affected family members (Carey et al., 2014; Rosenberg, Baker, Syrjala, & Wolfe, 2012; Schoenfelder, Sandler, Wolchik, MacKinnon, 2011)

and childhood through early adulthood is a critical time when during which this experience may have enduring health consequences.

How stress and trauma exposure affect health

One of the most heavily studied pathways linking stress and trauma exposure to health is the hypothalamus-pituitary-adrenal (HPA) axis, or the body's major stress system. Through the adaptive process of allostasis, the HPA axis attempts to address a stressor by producing hormones like cortisol in order to return to homeostasis. In contrast to normal stress, toxic stress occurs when there is frequent or sustained activation of the body's stress system that prevents a return to a healthy state of homeostasis (McEwen & McEwen, 2017). This unbalanced physiological state is characterized as allostatic load (McEwen, 1998), and when allostatic load is high, it can negatively affect brain architecture and several organ systems (Lupien, McEwen, Gunnar, & Heim, 2009; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Furthermore, it can contribute to stress systems that have relatively lower thresholds for perceived threats and ultimately increase the risk of cognitive impairment, as well as stress-related disease throughout the lifespan (Shonkoff, Boyce, & McEwen, 2009). Past work has also highlighted several physiological consequences of stress and trauma on the brain, including interruptions in the formation of connections between brain cells and subsequent changes in the function and structure of brain circuitry (Teicher et al., 2016). For example, childhood maltreatment has been shown to increase the amygdala's reactivity to threat, as well as decrease the size and density of brain areas involved in working memory, executive function, and self-awareness (Baker et al., 2013; Dannlowski et al., 2012; Heim & Binder, 2012; Saleh et al., 2017; Teicher & Samson, 2016). More generally, stress and trauma have also been linked to other

changes in brain circuitry that may intensify responses to new stressors as well as interfere with stress recovery (Blair & Raver, 2012; Tyrka, Ridout, & Parade, 2016).

The cardiovascular system is highly susceptible to the effects of stress and trauma exposure. For example, exaggerated and prolonged stress system responses have been linked to increased risks for heart attack, hypertension, and metabolic syndrome (Brody, Yu, Miller, Ehrlich & Chen, 2018; Mujahid, James, Kaplan, & Salonen, 2017; Subramanyam et al., 2013). One mechanism that may explain this occurrence is heart rate variability (HRV), which is an index of parasympathetic cardiac influence measured by the continuous intervals in time from one heartbeat to the next (Hill et al., 2017). When exposed to stress and trauma, higher HRV is typically cardio-protective and indicates better physical and mental health (Kemp & Quintana, 2013). In contrast, lower HRV has been linked to several risk factors for cardiovascular disease, the onset of hypertension, and all-cause mortality (Schroeder et al., 2003; ; Thayer & Lane, 2007; Thayer, Yamamoto, & Brosschot, 2010). However, it is important to note that while some work suggests that African Americans generally have higher HRV compared to whites (Hill et al., 2015), they are still at a greater risk for poor cardiovascular health (Benjamin et al., 2017; Carnethon et al., 2017; Howard et al., 2017).

The considerable link between mental and physical health has been highlighted in the high rates of comorbidity between depression and cardiovascular disease, with some scholars suggesting that HRV may be an important component in explaining this link (Larsen & Christenfeld, 2009). Lower HRV has been identified as an indicator of psychopathology (Beauchaine & Thayer, 2015), showing associations with poorer mental health outcomes including anxiety (Chalmers, Quintana, Abbot, & Kemp, 2014; Tully, Cosh, & Baune, 2013), borderline personality disorder (Koenig, Kemp, Feeling, Thayer, & Kaess, 2016), and

schizophrenia (Clamor, Lincoln, Thayer, & Koenig, 2016). Additionally, previous work has shown that several forms of psychopathology can have significant, negative consequences for a range of physical health outcomes. A meta-analysis of 62 empirical studies addressing the physical health consequences of post-traumatic stress disorder (PTSD) and PTSD symptoms showed significantly greater cardio-respiratory symptoms (e.g., asthma, heart disease), gastro-intestinal complaints (e.g., diarrhea, ulcers), and greater frequency and severity of pain (Pacella, Hruska, & Delahanty, 2013). Another meta-analysis discovered links between atherosclerotic cardiovascular disease (e.g., heart attack, stroke) and the reporting of anxiety symptoms and disorders (Batelaan, Seldenrijk, Bot, van Balkom, & Penninx, 2016).

High allostatic load and chronic stress may also suppress or dysregulate various immune and neuroendocrine system functions, resulting in increased susceptibility to inflammatory and autoimmune diseases (Dhabhar, 2014; Marsland, Walsh, Lockwood, & John-Henderson, 2017; Rohleder, 2014). Furthermore, stress and trauma have been linked to the shortening of leukocyte telomeres, which is a cellular marker of biological aging (Lopizzo et al., 2017; Oliveira et al., 2016; Tyrka et al., 2010; Verhoeven, van Oppen, Puterman, Elzinga, & Penninx, 2015). Importantly, shorter leukocyte telomere length has been associated with increased risk of all-cause mortality, coronary heart disease, stroke, heart attack, and type 2 diabetes (D'Mello et al., 2015; Haycock et al., 2014; Needham et al., 2015; Wang, Zhan, Pedersen, Fang, & Hägg, 2018). Shorter leukocyte telomere length has also been found among patients with several forms of psychopathology, including anxiety disorders, depressive disorders, and PTSD (Darrow et al., 2016).

When it comes to the impact of stress and trauma involving ethnic and racial discrimination specifically, empirical research has identified several negative physical health

consequences (Benner et al., 2018; Carter et al., 2019; Carter, Lau, Johnson, & Kirkinis, 2017; Williams, Lawrence, Davis, & Vu, 2019). For example, prior work has demonstrated that many adverse cardiovascular outcomes, including hypertension and lower HRV, as well as sleep problems like insomnia and poor sleep quality, are associated with discriminatory experiences (Bethea et al., 2019; Couto, Goto, & Bastos, 2012; Dolezsar, McGrath, Herzig, & Miller, 2014; Fuller-Rowell et al., 2017; Hill et al., 2017; Panza et al., 2019; Slopen, Lewis, & Williams, 2016). Furthermore, links have been found between discrimination and likelihood of asthma, shorter telomere length, and greater allostatic load (Brody et al., 2014; Chae et al., 2014; Coogan et al., 2014; Pantesco et al., 2018; Thakur et al., 2017). Past research also suggests that poor mental health outcomes can occur in response to discrimination, including anxiety disorders, mood disorders, suicide and death ideation, depressive symptoms, and poorer psychological well-being (Arshanapally, Werner, Sartor, & Bucholz, 2018; Assari, Moazen-Zadeh, Caldwell, & Zimmerman, 2017; Mouzon, Taylor, Keith, Nicklett, & Chatters, 2017; Schmitt, Branscombe, Postmes, & Garcia, 2014; Walker et al., 2017).

Health behaviors are another important pathway that may link stress and trauma exposure to health, as well as connect mental and physical health outcomes. For example, general stress exposure, including childhood maltreatment and financial strain, has been implicated in several subsequent health-impairing behaviors, including the onset and maintenance of smoking, unhealthy eating, substance use, and greater odds of insufficient physical activity (Advani et al., 2014; Jackson, Knight, & Rafferty, 2010; Moore-Greene, Gross, Silver, & Perrino, 2012). Health behaviors also highlight a domain in which mental and physical health may interact. Studies have shown that anxious and stressed individuals are more likely to engage in binge eating behavior, which may contribute to unhealthy weight gain (Rosenbaum & White, 2015), while

people dealing with depression are more likely to be alcohol dependent, which may increase their risk for physical ailments such as liver disease (McKay et al., 2016). With respect to ethnic and racial discrimination, those who report experiencing higher levels report a greater number of health risk behaviors, including engaging in fights, a higher number of sexual partners, and the use of alcohol and illicit drugs such as marijuana (Desalu, Goodhines, & Park, 2019; Flores et al., 2010; Hunte & Barry, 2012; Kogan, Yu, Allen, Pocock, & Brody, 2015; Kulis, Marsiglia, & Nieri, 2009). Based on this evidence, multiple explanations have been established as to how stress and trauma exposure impacts an individual, can lead to diminished mental and physical health status, and how mental and physical health impacts may be linked.

Intergenerational transmission of stress and trauma and health outcomes

In addition to investigating how stress and trauma exposure are linked to health, research efforts have addressed how these adverse experiences may be transmitted across generations and affect subsequent health. One line of work within this research area deals with stress and trauma exposure during pregnancy. For example, maternal psychological stress during pregnancy has been associated with several negative health outcomes for offspring, including adverse neurodevelopment, low birth weight, and preterm birth (Chan, Nugent, & Bale, 2018; Coussons-Read et al., 2012; Davis & Sandman, 2010; Diego et al., 2006; Glover, 2015). Another body of work focuses specifically on stress and trauma experienced prior to pregnancy in one generation and its association with the health of the subsequent generation. Studies have linked maternal early life stress and trauma to several offspring outcomes, including low birth weight, maladaptive infant socioemotional development, and child physical growth (Choi et al., 2017; Gavin, Hill, Hawkins, & Maas, 2011; McDonnell & Valentino, 2016).

In addition to general stress and trauma exposure, historical trauma theory (Sotero, 2006) posits that a collective trauma experience also contributes to significant health disparities for the affected population that linger across generations. Past work has shown that individuals from historically-traumatized populations may be particularly susceptible to poor psychological well-being in the form of greater anger, paranoia, prolonged grief, and self-hatred (Danieli, 1998; Danzer, Rieger, Schubmehl, & Cort, 2016). Consequently, the children and grandchildren of these populations who have not been directly traumatized may consciously and subconsciously absorb these trauma responses from their parents and show increased risk for impaired health (Sotero, 2006). The historical trauma literature has tended to focus primarily on the psychosocial and psychobiological consequences of mass trauma experiences. For example, historical trauma for the Native-American and Jewish populations has been associated with an increased prevalence of neuropsychiatric disorders, depressive symptoms, substance use problems, and suicidal ideation, as well as reduced cortisol levels and glucocorticoid receptor (GR) sensitivity in the offspring of survivors (Lehrner et al., 2014; McQuaid et al., 2017; Walls & Whitbeck, 2012; Yehuda et al., 2016; Yehuda et al., 2014). Some empirical work has explored the transgenerational, physical effects of collective trauma exposure and shown associations between famine exposure and poor neonatal physical health outcomes such as birthweight and ponderal index, as well as adulthood body size, coronary heart disease, diabetes, and obstructive airways disease (Lumey, Stein, & Susser, 2011; Painter et al., 2008; Painter, Roseboom, & Bleker, 2005; Roseboom, de Rooij, & Painter, 2006). However, a gap in the literature persists regarding how historical and ongoing stress and trauma exposure in African Americans is linked to physical health outcomes in subsequent familial generations beyond birth and early life outcomes.

Plan for the Dissertation

Past work has demonstrated considerable differences in health outcomes, as well as the greater prevalence of adverse events that are experienced by African Americans compared to whites. Moreover, the impact of adversity exposure may be transmitted across generations to influence the health outcomes of subsequent familial generations. This dissertation explores these issues in greater detail across two studies. The first study, a systematic review of the literature (Chapter 2), describes the current empirical literature that investigates intergenerational links between parental adversity experienced prior to pregnancy and physical health outcomes in African-American families. Chapter 3 reports an empirical study of African-American parents and their adult, biological children that addresses several of the limitations uncovered in the previous chapter. Collectively, both studies illustrate the importance of accounting for several factors that are integral to a more meticulous examination of the intergenerational health impacts of adversity in the African-American community. The findings from these studies and recommendations for future research directions are discussed in the Epilogue (Chapter 4).

References

Adler, N. E. (2009). Health disparities through a psychological lens. *American Psychologist, 64*, 663-673. doi: 10.1037/0003-066X.64.8.663

Advani, P. S., Reitzel, L. R., Nguyen, N. T., Fisher, F. D., Savoy, E. J., Cuevas, A. G., Wetter, D. W., & McNeill, L. H. (2014). Financial strain and cancer risk behaviors among African Americans. *Cancer Epidemiology, Biomarkers, and Prevention, 23*, 967-975. doi: 10.1158/1055-9965.EPI-14-0016

Alcendor, D. J. (2020). Racial disparities-associated COVID-19 mortality among minority populations in the US. *Journal of Clinical Medicine, 9(8)*, 2442. doi: 10.3390/jcm9082442

Allgood, K. L., Hunt, B., & Rucker, M. G. (2016). Black: White disparities in HIV mortality in the United States: 1990–2009. *Journal of Racial and Ethnic Health Disparities*, *3*, 168-175. doi: 10.1007/s40615-015-0141-8

American Cancer Society (2019). *Cancer Facts & Figures 2019*. Atlanta, GA: American Cancer Society. Retrieved from https://www.cancer.org/content/dam/cancer-org/research/cancer-facts-and-statistics/annual-cancer-facts-and-figures/2019/cancer-facts-and-figures-2019.pdf

American Psychological Association, APA Working Group on Stress and Health Disparities. (2017). Stress and health disparities: Contexts, mechanisms, and interventions among racial/ethnic minority and low-socioeconomic status populations. Retrieved from http://www.apa.org/pi/health-disparities/resources/stress-report.aspx

Arshanapally, S., Werner, K. B., Sartor, C. E., & Bucholz, K. K. (2018). The association

between racial discrimination and suicidality among African-American adolescents and young adults. *Archives of Suicide Research, 22*, 584-595. doi: 10.1080/13811118.2017.1387207

Assari, S., Moazen-Zadeh, E., Caldwell, C. H., & Zimmerman, M. A. (2017). Racial discrimination during adolescence predicts mental health deterioration in adulthood: Gender differences among Blacks. *Frontiers in Public Health, 5*, 104. doi: 10.3389/fpubh.2017.00104

Baker, L. M., Williams, L. M., Korgaonkar, M. S. Cohen, R. A., Heaps, J. M., & Paul, R. H. (2013). Impact of early vs. late childhood early life stress on brain morphometrics. *Brain Imaging and Behavior, 7*, 196-203. doi: 10.1007/s11682-012-9215-y

Batelaan, N. M., Seldenrijk, A., Bot, M., van Balkom, A. J., & Penninx, B. W. (2016). Anxiety and new onset of cardiovascular disease: Critical review and meta-analysis. *The British Journal of Psychiatry, 208*, 223-231. doi: 10.1192/bjp.bp.114.156554

Beauchaine, T. P., & Thayer, J. F. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology, 98*, 338-350. doi: 10.1016/j.ijpsycho.2015.08.004

Bell, M. P., Berry, D. P., Marquardt, D. J., & Galvin Green, T. (2013). Introducing discriminatory job loss: Antecedents, consequences, and complexities. *Journal of Managerial Psychology, 28*, 584-605. doi: 10.1108/JMP-10-2012-0319

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., de Ferranti, S. D., Floyd, J., Fornage, M., Gillespie, C., Isasi, C. R., Jimenez, M. C., Jordan, L. C., Judd, S. E., Lackland, D., Lichtman, J. H., Lisabeth, L., Liu, S., Longenecker, C. T., Mackey, R. H., Matsushita, K., Mozaffarian, D., Mussolino, M. E., Nasir, K., Neumar, R. W.,

Palaniappan, L., Pandey, D. K., Thiagarajan, R. R., Reeves, M. J., Ritchey, M., Rodriguez, C. J., Roth, G. A., Rosamond, W. D., Sasson, C., Towfighi, A., Tsao, C. W., Turner, M. B., Virani, S. S., Voeks, J. H., Willey, J. Z., Wilkins, J. T., Wu, J. H., Alger, H. M., Wong, S. S., & Muntner P (2017). Heart disease and stroke statistics—2017 update: A report from the American Heart Association. *Circulation, 135*, e146-e603. doi: 10.1161/CIR.0000000000000485

Benner, A. D., Wang, Y., Shen, Y., Boyle, A. E., Polk, R., & Cheng, Y. P. (2018). Racial/ethnic discrimination and well-being during adolescence: A meta-analytic review. *American Psychologist, 73,* 855-883. doi: 10.1037/amp0000204

Bethea, T. N., Zhou, E. S., Schernhammer, E. S., Castro-Webb, N., Cozier, Y. C., & Rosenberg, L. (2019). Perceived racial discrimination and risk of insomnia among middle-aged and elderly Black women. *Sleep, 2019,* zsz208. doi: 10.1093/sleep/zsz208

Blair, C., & Raver, C. C. (2012). Child development in the context of adversity: Experiential canalization of brain and behavior. *American Psychologist, 67,* 309-318. doi: 10.1037/a0027493

Boardman, J. D., & Alexander, K. B. (2011). Stress trajectories, health behaviors, and the mental health of Black and White young adults. *Social Science & Medicine, 72,* 1659-1666. doi: 10.1016/j.socscimed.2011.03.024

Brave Heart, M. Y. H. (1998). The return to the sacred path: Healing the historical trauma and historical unresolved grief response among the Lakota through a psychoeducational group intervention. *Smith College Studies in Social Work, 68,* 287-305. doi: 10.1080/00377319809517532

Brave Heart, M. Y. H., & DeBruyn, L. M. (1998). The American Indian holocaust: Healing

historical unresolved grief. *American Indian and Alaska Native Mental Health Research, 8,* 60-82. doi: 10.5820/aian.0802.1998.60

Brody, G. H., Yu, T., Miller, G. E., Ehrlich, K. B., & Chen, E. (2018). John Henryism coping and metabolic syndrome among young black adults. *Psychosomatic Medicine, 80*, 216-221. doi: 10.1097/PSY.0000000000000540

Brody, G. H., Lei, M. K., Chae, D. H., Yu, T., Kogan, S. M., & Beach, S. R. (2014). Perceived discrimination among African American adolescents and allostatic load: A longitudinal analysis with buffering effects. *Child Development, 85,* 989-1002. doi: 10.1111/cdev.12213

Brondolo, E., Libretti, M., Rivera, L., & Walsemann, K. M. (2012). Racism and social capital: The implications for social and physical well-being. *Journal of Social Issues, 68*, 358-384. doi: 10.1111/j.1540-4560.2012.01752.x

Bureau of Labor Statistics. (2016). *National Census of Fatal Occupational Injuries in 2015* [Press release]. Retrieved from https://www.bls.gov/news.release/pdf/cfoi.pdf

Burnside, M. S. (1997). *Spirits of the passage: The transatlantic slave trade in the seventeenth century*. New York, NY: Simon & Schuster.

Carey, I. M., Shah, S. M., DeWilde, S., Harris, T., Victor, C. R., & Cook, D. G. (2014). Increased risk of acute cardiovascular events after partner bereavement: A matched cohort study. *JAMA Internal Medicine, 174,* 598-605. doi: 10.1001/jamainternmed.2013.14558

Carnethon, M. R., Pu, J., Howard, G., Albert, M. A., Anderson, C. A., Bertoni, A. G., Mujahid,

M. S., Palaniappan, L., Taylor Jr., H. A., Willis, M. & Yancy, C. W. (2017). Cardiovascular health in African Americans: A scientific statement from the American Heart Association. *Circulation, 136,* e393-e423. doi: 10.1161/CIR.0000000000000534

Carter, R. T., Johnson, V. E., Kirkinis, K., Roberson, K., Muchow, C., & Galgay, C. (2019). A meta-analytic review of racial discrimination: Relationships to health and culture. *Race and Social Problems, 11*, 15-32. doi: 10.1007/s12552-018-9256-y

Carter, R. T., Lau, M. Y., Johnson, V., & Kirkinis, K. (2017). Racial discrimination and health outcomes among racial/ethnic minorities: A meta-analytic review. *Journal of Multicultural Counseling and Development, 45*, 232-259. doi: 10.1002/jmcd.12076

Caughy, M. O. B., Franzini, L., Windle, M., Dittus, P., Cuccaro, P., Elliott, M. N., & Schuster, M. A. (2012). Social competence in late elementary school: Relationships to parenting and neighborhood context. *Journal of Youth and Adolescence, 41*, 1613-1627. doi: 10.1007/s10964-012-9779-2

Chae, D. H., Nuru-Jeter, A. M., Adler, N. E., Brody, G. H., Lin, J., Blackburn, E. H., & Epel, E. S. (2014). Discrimination, racial bias, and telomere length in African-American men. *American Journal of Preventive Medicine, 46,* 103-111. doi: 10.1016/j.amepre.2013.10.020

Chalmers, J. A., Quintana, D. S., Abbott, M. J., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: A meta-analysis. *Frontiers in Psychiatry*, *5*, 80. doi: 10.3389/fpsyt.2014.00080

Chan, J. C., Nugent, B. M., & Bale, T. L. (2018). Parental advisory: Maternal and paternal stress can impact offspring neurodevelopment. *Biological Psychiatry, 83,* 886-894. doi: 10.1016/j.biopsych.2017.10.005

Chen, H. Y., & Panegyres, P. K. (2017). Ethnic differences in early onset Alzheimer's disease. *Journal of Alzheimer's Disease & Parkinsonism, 7*, 346. doi: 10.4172/2161-0460.1000346

Christian, C. M. (1999). *Black saga: The African American experience*: *A chronology*. Washington, DC: Civitas Counterpoint.

Clamor, A., Lincoln, T. M., Thayer, J. F., & Koenig, J. (2016). Resting vagal activity in schizophrenia: Meta-analysis of heart rate variability as a potential endophenotype. *The British Journal of Psychiatry, 208*, 9-16. doi: 10.1192/bjp.bp.114.160762

Clark, R., Anderson, N. B., Clark, V. R., & Williams, D. R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist, 54*, 805-816. doi: 10.1037/0003-066X.54.10 .805

Cohen, S., & Janicki-Deverts, D. (2012). Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006, and 2009. *Journal of Applied Social Psychology, 42*, 1320-1334. doi: 10.1111/j.1559-1816.2012.00900.x

Coogan, P. F., Yu, J., O'Connor, G. T., Brown, T. A., Cozier, Y. C., Palmer, J. R., & Rosenberg, L. (2014). Experiences of racism and the incidence of adult-onset asthma in the Black Women's Health Study. *Chest, 145*, 480-485. doi: 10.1378/chest.13-0665

Couch, K. A., & Fairlie, R. (2010). Last hired, first fired? Black-white unemployment and the business cycle. *Demography, 47*, 227-247. doi: 10.1353/dem.0.0086

Coussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C., & Cole, S. (2012). The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behavior, and Immunity, 26*, 650-659. doi: 10.1016/j.bbi.2012.02.009

Couto, P. F., Goto, J. B., & Bastos, J. L. (2012). Blood pressure and interpersonal discrimination: Systematic review of epidemiologic studies. *Arquivos Brasileiros de Cardiologia, 99,* 956-963. doi: 10.1590/S0066-782X2012005000090

Cunningham, T. J., Croft, J. B., Liu, Y., Lu, H., Eke, P. I., & Giles, W. H. (2017). Vital signs: Racial disparities in age-specific mortality among blacks or African Americans—United States, 1999–2015. *MMWR. Morbidity and Mortality Weekly Report, 66,* 444-456. doi: 10.15585/mmwr.mm6617e1

Danieli, Y. (1998). *International handbook of multigenerational legacies of trauma*. New York, NY: Plenum.

Dannlowski, U., Stuhrmann, A., Beutelmann, V., Zwanzger, P., Lenzen, T., Grotegerd, D., Domschke, K., Hohoff, C., Ohrmann, P., Bauer, J., Lindner, C., Postert, C., Konrad, C., Arolt, V., Heindel, W., Suslow, T., & Kugel, H. (2012). Limbic scars: Long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry, 71,* 286-293. doi: 10.1016/j.biopsych.2011.10.021

Danzer, G., Rieger, S. M., Schubmehl, S., & Cort, D. (2016). White psychologists and African Americans' historical trauma: Implications for practice. *Journal of Aggression, Maltreatment & Trauma, 25,* 351-370. doi: 10.1080/10926771.2016.1153550

Darrow, S. M., Verhoeven, J. E., Révész, D., Lindqvist, D., Penninx, B. W., Delucchi, K. L., Wolkowitz, O. M., & Mathews, C. A. (2016). The association between psychiatric disorders and telomere length: A meta-analysis involving 14,827 persons. *Psychosomatic Medicine, 78,* 776-787. doi: 10.1097/PSY.0000000000000356

Davis, E. P., & Sandman, C. A. (2010). The timing of prenatal exposure to maternal cortisol and

psychosocial stress is associated with human infant cognitive development. *Child Development, 81,* 131-148. doi: 10.1111/j.1467-8624.2009.01385.x

Desalu, J. M., Goodhines, P. A., & Park, A. (2019). Racial discrimination and alcohol use and negative drinking consequences among Black Americans: A meta-analytical review. *Addiction, 114,* 957-967. doi: 10.1111/add.14578

Devenish, B., Hooley, M., & Mellor, D. (2017). The pathways between socioeconomic status and adolescent outcomes: A systematic review. *American Journal of Community Psychology, 59,* 219-238. doi: 10.1002/ajcp.12115

Dhabhar, F. S. (2014). Effects of stress on immune function: The good, the bad, and the beautiful. *Immunologic Research, 58,* 193-210. doi: 10.1007/s12026-014-8517-0

Dixon, T. L. (2008). Network news and racial beliefs: Exploring the connection between national television news exposure and stereotypical perceptions of African Americans. *Journal of Communication, 58,* 321-337. doi: 10.1111/j.1460-2466.2008.00387.x

D'Mello, M. J., Ross, S. A., Briel, M., Anand, S. S., Gerstein, H., & Paré, G. (2015). Association between shortened leukocyte telomere length and cardiometabolic outcomes: Systematic review and meta-analysis. *Circulation: Cardiovascular Genetics, 8,* 82-90. doi: 10.1161/CIRCGENETICS.113.000485

Doerner, J. K., & Demuth, S. (2010). The independent and joint effects of race/ethnicity, gender, and age on sentencing outcomes in U.S. federal courts. *Justice Quarterly, 27,* 1-27. doi: 10.1080/07418820902926197

Dolezsar, C. M., McGrath, J. J., Herzig, A. J., & Miller, S. B. (2014). Perceived racial discrimination and hypertension: A comprehensive systematic review. *Health Psychology, 33,* 20-34. doi: 10.1037/a0033718

Drake, B., & Jonson-Reid, M. (2014). Poverty and child maltreatment. In J. Korbin & H. Krugman (Eds.), *Handbook of child maltreatment* (pp. 131-148). Springer, Dordrecht.

Eltis, D., & Richardson, D. (2010). *Atlas of the transatlantic slave trade*. New Haven, CT: Yale University Press.

Eltis, D. (2007). A brief overview of the transatlantic slave trade. *Voyages: The trans-Atlantic slave trade database*. Retrieved from https://slavevoyages.org/voyage/essays

Elvira, M. M., & Zatzick, C. D. (2002). Who's displaced first? The role of race in layoff decisions. *Industrial Relations: A Journal of Economy and Society, 41*, 329-361. doi: 10.1111/1468-232X.00248

Evans, G. W., & Kim, P. (2013). Childhood poverty, chronic stress, self-regulation, and coping. *Child Development Perspectives, 7*, 43-48. doi: 10.1111/cdep.12013

Evans-Campbell, T. (2008). Historical trauma in American Indian/Native Alaska communities: A multilevel framework for exploring impacts on individuals, families, and communities. *Journal of Interpersonal Violence, 23*, 316-338. doi: 10.1177/0886260507312290

Flores, E., Tschann, J. M., Dimas, J. M., Pasch, L. A., & de Groat, C. L. (2010). Perceived racial/ethnic discrimination, posttraumatic stress symptoms, and health risk behaviors among Mexican American adolescents. *Journal of Counseling Psychology, 57*, 264-273. doi: 10.1037/a0020026

Franklin, J. H., & Moss, A. A. (2000). *A history of African Americans: From slavery to freedom (8th ed)*. New York, NY: Alfred A. Knopf.

Fuller-Rowell, T. E., Curtis, D. S., El-Sheikh, M., Duke, A. M., Ryff, C. D., & Zgierska, A. E.

(2017). Racial discrimination mediates race differences in sleep problems: A longitudinal analysis. *Cultural Diversity & Ethnic Minority Psychology, 23*, 165-173. doi: 10.1037/cdp0000104

George, C. (2000). *Life under the Jim Crow laws*. San Diego, CA: Lucent Books.

Harrell, S. P. (2000). A multidimensional conceptualization of racism-related stress: Implications for the well-being of people of color. *American Journal of Orthopsychiatry, 70*, 42-57. doi: 10.1037/h0087722

Hall, E. W., Rosenberg, E. S., & Sullivan, P. S. (2018). Estimates of state-level chronic hepatitis C virus infection, stratified by race and sex, United States, 2010. *BMC Infectious Diseases, 18*, 224. doi: 10.1186/s12879-018-3133-6

Haycock, P. C., Heydon, E. E., Kaptoge, S., Butterworth, A. S., Thompson, A., & Willeit, P. (2014). Leucocyte telomere length and risk of cardiovascular disease: Systematic review and meta-analysis. *BMJ, 349*, g4227. doi: 10.1136/bmj.g4227

Hayes, B. H., Haberling, D. L., Kennedy, J. L., Varma, J. K., Fry, A. M., & Vora, N. M. (2018). Burden of pneumonia-associated hospitalizations: United States, 2001-2014. *Chest, 153*, 427-437. doi: 10.1016/j.chest.2017.09.041

Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression: Review of human studies on sensitive periods, gene–environment interactions, and epigenetics. *Experimental Neurology, 233*, 102-111. doi: 10.1016/j.expneurol.2011.10.032

Hill, L. K., Richmond, A. S., Hoggard, L. S., Gray, D. L., Williams, D. P., & Thayer, J. F.

(2017). Examining the association between perceived discrimination and heart rate variability in African Americans. *Cultural Diversity and Ethnic Minority Psychology, 23*, 5-14. doi: 10.1037/cdp0000076

Hill, L. K., Hu, D. D., Koenig, J., Sollers III, J. J., Kapuku, G., Wang, X., Snieder, H., & Thayer, J. F. (2015). Ethnic differences in resting heart rate variability: A systematic review and meta-analysis. *Psychosomatic Medicine*, *77*, 16-25. doi: 10.1097/PSY.0000000000000133

Hornby, G., & Lafaele, R. (2011). Barriers to parental involvement in education: An explanatory model. *Educational Review, 63*, 37-52. doi: 10.1080/00131911.2010.488049

Howard, G., Safford, M. M., Moy, C. S., Howard, V. J., Kleindorfer, D. O., Unverzagt, F. W., Soliman, E. Z., Flaherty, M. L., McClure, L. A., Lackland, D. T., Wadley, V. G., Pulley, L., & Cushman, M. (2017). Racial differences in the incidence of cardiovascular risk factors in older black and white adults. *Journal of the American Geriatrics Society*, *65*, 83-90. doi: 10.1111/jgs.14472

Huggins, N. I. (1990). *Black odyssey: The African American ordeal in slavery*. New York, NY: Vintage Books.

Hunte, H. E., & Barry, A. E. (2012). Perceived discrimination and DSM-IV-based alcohol and illicit drug use disorders. *American Journal of Public Health, 102*, e111-e117. doi: 10.2105/AJPH.2012.300780

Jackson, J. S., Knight, K. M., & Rafferty, J. A. (2010). Race and unhealthy behaviors: Chronic stress, the HPA axis, and physical and mental health disparities over the life course. *American Journal of Public Health, 100,* 933-939. doi: 10.2105/AJPH.2008.143446

Jonson-Reid, M., Drake, B., & Kohl, P. L. (2009). Is the overrepresentation of the poor in child

welfare caseloads due to bias or need? *Children and Youth Services Review, 31,* 422-427. doi: 10.1016/j.childyouth.2008.09.009

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: Insights from the study of heart rate variability. *International Journal of Psychophysiology, 89,* 288-296. doi: j.ijpsycho.2013.06.018

Kim, H. S., Rotundo, L., Yang, J. D., Kim, D., Kothari, N., Feurdean, M., Ruhl, C., & Unalp-arida, A. (2017). Racial/ethnic disparities in the prevalence and awareness of Hepatitis B virus infection and immunity in the United States. *Journal of Viral Hepatitis, 24,* 1052-1066. doi: 10.1111/jvh.12735

Koenig, J., Kemp, A. H., Feeling, N. R., Thayer, J. F., & Kaess, M. (2016). Resting state vagal tone in borderline personality disorder: A meta-analysis. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *64,* 18-26. doi: 10.1016/j.pnpbp.2015.07.002

Kogan, S. M., Yu, T., Allen, K. A., Pocock, A. M., & Brody, G. H. (2015). Pathways from racial discrimination to multiple sexual partners among male African American adolescents. *Psychology of Men & Masculinity*, *16,* 218-228. doi:10.1037/a0037453

Krieger, N. (1999). Embodying inequality: A review of concepts, measures, and methods for studying health consequences of discrimination. *International Journal of Health Services, 29,* 295-352. doi: 10.2190/M11W-VWXE-KQM9-G97Q

Kulis, S., Marsiglia, F. F., & Nieri, T. (2009). Perceived ethnic discrimination versus acculturation stress: Influences on substance use among Latino youth in the Southwest. *Journal of Health and Social Behavior, 50,* 443-459. doi: 10.1177/002214650905000405

Lankford, H., & Wyckoff, J. (2006). The effect of school choice and residential location on the

racial segregation of students. In *Improving school accountability* (pp. 185-239). Emerald Group Publishing Limited.

Larsen, B. A., & Christenfeld, N. J. (2009). Cardiovascular disease and psychiatric comorbidity: The potential role of perseverative cognition. *Cardiovascular Psychiatry and Neurology, 2009,* 1-8. doi: 10.1155/2009/791017

Leary, J. D. (2005). *Post traumatic slave syndrome: America's legacy of enduring injury and healing*. Milwaukee, Oregon: Uptone Press.

Lee, C., Coe, C. L., & Ryff, C. D. (2017). Social disadvantage, severe child abuse, and biological profiles in adulthood. *Journal of Health and Social Behavior, 58,* 371-386. doi: 10.1177/0022146516685370

Lehrner, A., Bierer, L. M., Passarelli, V., Pratchett, L. C., Flory, J. D., Bader, H. N., Harris, I. R., Bedi, A., Daskalakis, N. P., Makotkine, I., & Yehuda, R. (2014). Maternal PTSD associates with greater glucocorticoid sensitivity in offspring of Holocaust survivors. *Psychoneuroendocrinology, 40*, 213-220. doi: 10.1016/j.psyneuen.2013.11.019

Lopizzo, N., Tosato, S., Begni, V., Tomassi, S., Cattane, N., Barcella, M., Turco, G., Ruggieri, M., Riva, M. A., Pariante, C. M., & Cattaneo, A. (2017). Transcriptomic analyses and leukocyte telomere length measurement in subjects exposed to severe recent stressful life events. *Translational Psychiatry, 7*, e1042. doi: 10.1038/tp.2017.5

Lumey, L. H., Stein, A. D., & Susser, E. (2011). Prenatal famine and adult health. *Annual Review of Public Health*, *32*, 237-262. doi: 10.1146/annurev-publhealth-031210-101230

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*, 434-445. doi: 10.1038/nrn2639

Marsland, A. L., Walsh, C., Lockwood, K., & John-Henderson, N. A. (2017). The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. *Brain, Behavior, and Immunity, 64*, 208-219. doi: 10.1016/j.bbi.2017.01.011

McDonnell, C. G., & Valentino, K. (2016). Intergenerational effects of childhood trauma: Evaluating pathways among maternal ACEs, perinatal depressive symptoms, and infant outcomes. *Child Maltreatment, 21*, 317-326. doi: 10.1177/1077559516659556

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*, 171-179. doi: 10.1056/NEJM199801153380307

McEwen, C. A., & McEwen, B. S. (2017). Social structure, adversity, toxic stress, and intergenerational poverty: An early childhood model. *Annual Review of Sociology, 43*, 445-472. doi: 10.1146/annurev-soc-060116-053252

McKay, K. A., Tremlett, H., Fisk, J. D., Patten, S. B., Fiest, K., Berrigan, L., & Marie, R. A. for the CIHR Team in the Epidemiology and Impact of Comorbidity on Multiple Sclerosis (2016). Adverse health behaviours are associated with depression and anxiety in multiple sclerosis: A prospective multisite study. *Multiple Sclerosis Journal, 22*, 685-693. doi: 10.1177/1352458515599073

McQuaid, R. J., Bombay, A., McInnis, O. A., Humeny, C., Matheson, K., & Anisman, H. (2017). Suicide ideation and attempts among First Nations peoples living on-reserve in Canada: The intergenerational and cumulative effects of Indian residential schools. *The Canadian Journal of Psychiatry, 62*, 422-430. doi: 10.1177/0706743717702075

Mehta, K. M., & Yeo, G. W. (2017). Systematic review of dementia prevalence and incidence in

United States race/ethnic populations. *Alzheimer's & Dementia, 13*, 72-83. doi: 10.1016/j.jalz.2016.06.2360

Moore-Greene, G. M., Gross, S. M., Silver, K. D., & Perrino, C. S. (2012). Chronic stress and decreased physical exercise: Impact on weight for African American women. *Ethnicity & Disease, 22,* 185-191.

Mouzon, D. M., Taylor, R. J., Keith, V. M., Nicklett, E. J., & Chatters, L. M. (2017). Discrimination and psychiatric disorders among older African Americans. *International Journal of Geriatric Psychiatry, 32*, 175-182. doi: 10.1002/gps.4454

Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., . . . Howard, V. J. (2016). Executive summary: Heart disease and stroke statistics—2016 update: A report from the American Heart Association. *Circulation, 133*, 447-454. doi: 10.1161/CIR.0000000000000366

Mujahid, M. S., James, S. A., Kaplan, G. A., & Salonen, J. T. (2017). Socioeconomic position, John Henryism, and incidence of acute myocardial infarction in Finnish men. *Social Science & Medicine*, 173, 54-62. doi: 10.1016/j.socscimed.2016.11.034

Mude, W., Oguoma, V. M., Nyanhanda, T., Mwanri, L., & Njue, C. (2021). Racial disparities in COVID-19 pandemic cases, hospitalisations, and deaths: A systematic review and meta-analysis. *Journal of Global Health, 11*, 05015. doi: 10.7189/jogh.11.05015

Needham, B. L., Rehkopf, D., Adler, N., Gregorich, S., Lin, J., Blackburn, E. H., & Epel, E. S. (2015). Leukocyte telomere length and mortality in the National Health and Nutrition Examination Survey, 1999–2002. *Epidemiology, 26*, 528-535. doi: 10.1097/EDE.0000000000000299

Ogden, C. L., Carroll, M. D., Lawman, H. G., Fryar, C. D., Kruszon-Moran, D., Kit, B. K., &

Flegal, K. M. (2016). Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA, 315,* 2292-2299. doi: 10.1001/jama.2016.6361

Ogden, C. L., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2014). Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA, 311,* 806-814. doi: 10.1001/jama.2014.732

Oliveira, B. S., Zunzunegui, M. V., Quinlan, J., Fahmi, H., Tu, M. T., & Guerra, R. O. (2016). Systematic review of the association between chronic social stress and telomere length: A life course perspective. *Ageing Research Reviews, 26,* 37-52. doi: 10.1016/j.arr.2015.12.006

Olshansky, S. J., Antonucci, T., Berkman, L., Binstock, R. H., Boersch-Supan, A., Cacioppo, J. T., Carnes, B. A., Carstensen, L. L., Fried, L. P., Goldman, D. P., Jackson, J., Kohli, M., Rother, J., Zheng, Y., & Rowe, J. (2012). Differences in life expectancy due to race and educational differences are widening, and many may not catch up. *Health Affairs, 31*, 1803-1813. doi: 10.1377/hlthaff.2011.0746

Pacella, M. L., Hruska, B., & Delahanty, D. L. (2013). The physical health consequences of PTSD and PTSD symptoms: A meta-analytic review. *Journal of Anxiety Disorders, 27,* 33-46. doi: 10.1016/j.janxdis.2012.08.004

Pager, D., Western, B., & Pedulla, D. (2009). Employment discrimination and the changing landscape of low-wage labor markets. *University of Chicago Legal Forum, 1*, 317-344.

Painter, R. C., Osmond, C., Gluckman, P., Hanson, M., Phillips, D. I., & Roseboom, T. J. (2008).

Transgenerational effects of prenatal exposure to the Dutch famine on neonatal adiposity and health in later life. *BJOG: An International Journal of Obstetrics & Gynaecology,* 115, 1243-1249. doi: 10.1111/j.1471-0528.2008.01822.x

Painter, R. C., Roseboom, T. J., & Bleker, O. P. (2005). Prenatal exposure to the Dutch famine and disease in later life: An overview. *Reproductive Toxicology*, *20,* 345-352. doi: 10.1016/j.reprotox.2005.04.005

Pantesco, E. J., Leibel, D. K., Ashe, J. J., Waldstein, S. R., Katzel, L. I., Liu, H. B., Weng, N. P., Evans, M. K., Zonderman, A. B., & Beatty Moody, D. L. (2018). Multiple forms of discrimination, social status, and telomere length: Interactions within race. *Psychoneuroendocrinology*, *98*, 119-126. doi: 10.1016/j.psyneuen.2018.08.012

Panza, G. A., Puhl, R. M., Taylor, B. A., Zaleski, A. L., Livingston, J., & Pescatello, L. S. (2019). Links between discrimination and cardiovascular health among socially stigmatized groups: A systematic review. *PloS One, 14,* e0217623. doi: 10.1371/journal.pone.0217623

Prather, C., Fuller, T. R., Jeffries IV, W. L., Marshall, K. J., Howell, A. V., Belyue-Umole, A., & King, W. (2018). Racism, African American women, and their sexual and reproductive health: A review of historical and contemporary evidence and implications for health equity. *Health Equity*, *2,* 249-259. doi: 10.1089/heq.2017.0045

Reiss, F. (2013). Socioeconomic inequalities and mental health problems in children and adolescents: A systematic review. *Social Science & Medicine*, *90,* 24-31. doi: 10.1016/j.socscimed.2013.04.026

Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic

differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine, 41*, 71-83. doi: 10.1017/S0033291710000401

Rohleder, N. (2014). Stimulation of systemic low-grade inflammation by psychosocial stress. *Psychosomatic Medicine, 76*, 181-189. doi: 10.1097/PSY.0000000000000049

Rollins, J. C., & Hicks, B. M. (2010). *From Moses to the Joshua generation*. Fort Washington, MD: Lulu.com.

Roseboom, T., de Rooij, S., & Painter, R. (2006). The Dutch famine and its long-term consequences for adult health. *Early Human Development*, *82*, 485-491. doi: 10.1016/j.earlhumdev.2006.07.001

Rosenbaum, D. L., & White, K. S. (2015). The relation of anxiety, depression, and stress to binge eating behavior. *Journal of Health Psychology*, *20*, 887-898. doi: 10.1177/1359105315580212

Rosenberg, A. R., Baker, K. S., Syrjala, K., & Wolfe, J. (2012). Systematic review of psychosocial morbidities among bereaved parents of children with cancer. *Pediatric Blood & Cancer*, *58*, 503-512. doi: 10.1002/pbc.23386

Ross, C. E., Masters, R. K., & Hummer, R. A. (2012). Education and the gender gaps in health and mortality. *Demography, 49*, 1157-1183. doi: 10.1007/s13524-012-0130-z

Ruef, M., & Fletcher, B. (2003). Legacies of American slavery: Status attainment among southern blacks after emancipation. *Social Forces*, *82*, 445-480. doi: 10.1353/sof.2004.0024

Ruiter, M. E., DeCoster, J., Jacobs, L., & Lichstein, K. L. (2010). Sleep disorders in African

Americans and Caucasian Americans: A meta-analysis. *Behavioral Sleep Medicine, 8,* 246-259. doi: 10.1080/15402002.2010.509251

Saleh, A., Potter, G. G., McQuoid, D. R., Boyd, B., Turner, R., MacFall, J. R., & Taylor, W. D. (2017). Effects of early life stress on depression, cognitive performance and brain morphology. *Psychological Medicine, 47,* 171-181. doi: 10.1017/S0033291716002403

Schilling, E. A., Aseltine, R. H., & Gore, S. (2007). Adverse childhood experiences and mental health in young adults: A longitudinal survey. *BMC Public Health, 7,* 30. doi: 10.1186/1471-2458-7-30

Schmitt, M. T., Branscombe, N. R., Postmes, T., & Garcia, A. (2014). The consequences of perceived discrimination for psychological well-being: A meta-analytic review. *Psychological Bulletin, 140,* 921-948. doi: 10.1037/a0035754

Schoenfelder, E. N., Sandler, I. N., Wolchik, S., & MacKinnon, D. (2011). Quality of social relationships and the development of depression in parentally-bereaved youth. J*ournal of Youth and Adolescence, 40,* 85-96. doi: 10.1007/s10964-009-9503-z

Schroeder, E. B., Liao, D., Chambless, L. E., Prineas, R. J., Evans, G. W., & Heiss, G. (2003). Hypertension, blood pressure, and heart rate variability: The Atherosclerosis Risk in Communities (ARIC) study. *Hypertension, 42,* 1106-1111. doi: 10.1161/01.HYP.0000100444.71069.73

Seeman, T., Epel, E., Gruenewald, T., Karlamangla, A., & McEwen, B. S. (2010). Socio-economic differentials in peripheral biology: Cumulative allostatic load. *Annals of the New York Academy of Sciences, 1186,* 223-239. doi: 10.1111/j.1749-6632.2009.05341.x

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and

the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *JAMA, 301*, 2252-2259. doi: 10.1001/jama.2009.754

Siddiqi, A. E., Hu, X., Hall, H. I., & Centers for Disease Control and Prevention (CDC) (2015). Mortality among blacks or African Americans with HIV infection —United States, 2008-2012. *MMWR. Morbidity and Mortality Weekly Report, 64*, 81-86.

Slopen, N., Lewis, T. T., & Williams, D. R. (2016). Discrimination and sleep: A systematic review. *Sleep Medicine, 18*, 88-95. doi: 10.1016/j.sleep.2015.01.012

Smith, B. W., & Holmes, M. D. (2014). Police use of excessive force in minority communities: A test of the minority threat, place, and community accountability hypotheses. *Social Problems, 61*, 83-104. doi: 10.1525/sp.2013.12056

Smith, J. D. (1996). *Black voices from reconstruction: 1865–1877*. Brookfield, CT: The Millbrook Press.

Sotero, M. (2006). A conceptual model of historical trauma: Implications for public health practice and research. *Journal of Health Disparities Research and Practice, 1*, 93-108.

Steenland, K., Goldstein, F. C., Levey, A., & Wharton, W. (2016). A meta-analysis of Alzheimer's disease incidence and prevalence comparing African-Americans and Caucasians. *Journal of Alzheimer's Disease, 50*, 71-76. doi: 10.3233/JAD-150778

Sternthal, M. J., Slopen, N., & Williams, D. R. (2011). Racial disparities in health: How much does stress really matter? *Du Bois Review: Social Science Research on Race, 8*, 95-113. doi: 10.1017/S1742058X11000087

Subramanyam, M. A., James, S. A., Diez-Roux, A. V., Hickson, D. A., Sarpong, D., Sims, M.,

Taylor, H. A., & Wyatt, S. B. (2013). Socioeconomic status, John Henryism and blood pressure among African-Americans in the Jackson heart study. *Social Science & Medicine, 93*, 139-146. doi: 10.1016/j.socscimed.2013.06.016

Sweeting, J. A., Garfin, D. R., Holman, E. A., & Silver, R. C. (2020). Associations between exposure to childhood bullying and abuse and adulthood outcomes in a representative national U.S. sample. *Child Abuse & Neglect, 101*, 104048. doi: 10.1016/j.chiabu.2019.104048

Teicher, M. H., & Samson, J. A. (2016). Annual research review: Enduring neurobiological effects of childhood abuse and neglect. *Journal of Child Psychology and Psychiatry, 57*, 241-266. doi: 10.1111/jcpp.12507

Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience, 17*, 652-666. doi: 10.1038/nrn.2016.111

Thakur, N., Barcelo, N.E., Borrell, L. N., Singh, S., Eng, C., Davis, A., Meade, K., LeNoir, M. A., Avila, P. C., Farber, H. J., Serebrisky, D., Brigino-Buenaventura, E., Rodriguez-Cintron, W., Thyne, S., Rodriguez-Santana, J. R., Sen, S., Bibbins-Domingo, K., & Burchard, E. G. (2017) Perceived discrimination associated with asthma and related outcomes in minority youth: The GALA II and SAGE II studies. *Chest, 151*, 804-812. doi: 10.1016/j.chest.2016.11.027

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology, 141*, 122-131. doi: 10.1016/j.ijcard.2009.09.543

Thayer, J. F., & Lane, R. D. (2007). The role of vagal function in the risk for cardiovascular

disease and mortality. *Biological Psychology, 74,* 224-242. doi: 10.1016/j.biopsycho.2005.11.013

Topitzes, J., Pate, D. J., Berman, N. D., & Medina-Kirchner, C. (2016). Adverse childhood experiences, health, and employment: A study of men seeking job services. *Child Abuse & Neglect, 61,* 23-34. doi: 10.1016/j.chiabu.2016.09.012

Truman, J., & Langdon, L. (2015). *Criminal Victimization*, 2014. Retrieved from the Bureau of Justice Statistics website: https://www.bjs.gov/content/pub/pdf/cv14.pdf

Tully, P. J., Cosh, S. M., & Baune, B. T. (2013). A review of the affects of worry and generalized anxiety disorder upon cardiovascular health and coronary heart disease. *Psychology, Health & Medicine, 18,* 627-644. doi: 10.1080/13548506.2012.749355

Tyrka, A. R., Ridout, K. K., & Parade, S. H. (2016). Childhood adversity and epigenetic regulation of glucocorticoid signaling genes: Associations in children and adults. *Development and Psychopathology, 28,* 1319-1331. doi: 10.1017/S0954579416000870

Tyrka, A. R., Price, L. H., Kao, H. T., Porton, B., Marsella, S. A., & Carpenter, L. L. (2010). Childhood maltreatment and telomere shortening: Preliminary support for an effect of early stress on cellular aging. *Biological Psychiatry, 67,* 531-534. doi: 10.1016/j.biopsych.2009.08.014

Ulmer, J. T., Harris, C. T., & Steffensmeier, D. (2012). Racial and ethnic disparities in structural disadvantage and crime: White, Black, and Hispanic comparisons. *Social Science Quarterly,* 93, 799-819. doi: 10.1111/j.1540-6237.2012.00868.x

Umberson, D., Olson, J. S., Crosnoe, R., Liu, H., Pudrovska, T., & Donnelly, R. (2017). Death of

family members as an overlooked source of racial disadvantage in the United States. *Proceedings of the National Academy of Sciences, 114*, 915-920. doi: 10.1073/pnas.1605599114

US Census Bureau. (2019a). *Income and Poverty in the United States: 2019*. Retrieved from https://www.census.gov/library/publications/2020/demo/p60-270.html

US Census Bureau. (2019b). *Wealth, Asset Ownership, & Debt of Households Detailed Tables: 2019* Retrieved from https://www.census.gov/data/tables/2019/demo/wealth/wealth-asset-ownership.html

Valdez, Z., & Golash-Boza, T. (2017). US racial and ethnic relations in the twenty-first century. *Ethnic and Racial Studies, 40*, 2181-2209. doi: 10.1080/01419870.2016.1262052

Verhoeven, J. E., van Oppen, P., Puterman, E., Elzinga, B., & Penninx, B. W. (2015). The association of early and recent psychosocial life stress with leukocyte telomere length. *Psychosomatic Medicine, 77*, 882-891. doi: 10.1097/PSY.0000000000000226

Walls, M. L., & Whitbeck, L. B. (2012). The intergenerational effects of relocation policies on indigenous families. *Journal of Family Issues, 33*, 1272-1293. doi: 10.1177/0192513X12447178

Walker, R., Francis, D., Brody, G., Simons, R., Cutrona, C., & Gibbons, F. (2017). A longitudinal study of racial discrimination and risk for death ideation in African American youth. *Suicide and Life-Threatening Behavior, 47*, 86-102. doi: 10.1111/sltb.12251

Wang, Q., Zhan, Y., Pedersen, N. L., Fang, F., & Hägg, S. (2018). Telomere length and all-cause

mortality: A meta-analysis. *Ageing Research Reviews, 48,* 11-20. doi: 10.1016/j.arr.2018.09.002

Watson, T., & McLanahan, S. (2011). Marriage meets the joneses relative income, identity, and marital status. *Journal of Human Resources, 46,* 482-517. doi: 10.3368/jhr.46.3.482

Williams, D. R., Lawrence, J. A., Davis, B. A., & Vu, C. (2019). Understanding how discrimination can affect health. *Health Services Research, 54,* 1374-1388. doi: 10.1111/1475-6773.13222

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

Williams, D. R. (2012). Miles to go before we sleep: Racial inequities in health. *Journal of Health and Social Behavior, 53,* 279-295. doi: 10.1177/0022146512455804

Williams, D. R., Mohammed, S. A., Leavell, J., & Collins, C. (2010). Race, socioeconomic status and health: Complexities, ongoing challenges and research opportunities. *Annals of the New York Academy of Sciences, 1186,* 69-101. doi: 10.1111/j.1749-6632.2009.05339.x

Wolfe, B. (2013). Slave ships and the middle passage. In *Encyclopedia Virginia*. Retrieved from https://www.encyclopediavirginia.org/Slave_Ships_and_the_Middle_Passage

Worth, R. (2001). *Cinque of the Amistad and the slave trade in world history.* Berkeley Heights, NJ: Enslow Publishers.

Yehuda, R., Daskalakis, N. P., Bierer, L. M., Bader, H. N., Klengel, T., Holsboer, F., & Binder, E. B. (2016). Holocaust exposure induced intergenerational effects on FKBP5 methylation. *Biological Psychiatry, 80,* 372-380. doi: 10.1016/j.biopsych.2015.08.005

Yehuda, R., Daskalakis, N. P., Lehrner, A., Desarnaud, F., Bader, H. N., Makotkine, I., Flory, J. D., Bierer, L. M., & Meaney, M. J. (2014). Influences of maternal and paternal PTSD on epigenetic regulation of the glucocorticoid receptor gene in Holocaust survivor offspring. *American Journal of Psychiatry, 171*, 872-880. doi: 10.1176/appi.ajp.2014.13121571

Yuen, C. M., Kammerer, J. S., Marks, K., Navin, T. R., & France, A. M. (2016). Recent transmission of tuberculosis—United States, 2011–2014. *PloS One*, *11*, e0153728. doi: 10.1371/journal.pone.0153728

Zelner, J., Trangucci, R., Naraharisetti, R., Cao, A., Malosh, R., Broen, K., Masters, N., & Delamater, P. (2021). Racial disparities in coronavirus disease 2019 (COVID-19) mortality are driven by unequal infection risks. *Clinical Infectious Diseases, 72(5)*, e88-e95. doi: 10.1093/cid/ciaa1723

Zou, B., Yeo, Y. H., Le, M. H., Henry, L., Chang, E. T., Lok, A. S., Cheung, R., & Nguyen, M. H. (2019). Prevalence of viremic Hepatitis C virus infection by age, race/ethnicity, and birthplace and disease awareness among viremic persons in the United States, 1999–2016. *The Journal of Infectious Diseases*, *3*, 408-418. doi: 10.1093/infdis/jiz479

Chapter 2: Parental preconception adversity and offspring health in African Americans: A systematic review of intergenerational studies[1]

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Abstract

This systematic review explores the empirical literature addressing the association between parental preconception adversity and offspring physical health in African-American families. We conducted a literature search in PubMed, Web of Science, PsycINFO, CINAHL, and Scopus through June 2021. Articles were included if they: reported data about at least two generations of African-American participants from the same family; measured parental preconception adversity at the individual level; measured at least one offspring physical health outcome; and examined associations between parental adversity and child health. We identified 701 unique articles; thirty-eight articles representing 30 independent studies met inclusion criteria. Twenty-five studies (83%) reported that parental preconception adversity was associated with child health; six studies (20%) reported that parental preconception adversity was not associated with at least one offspring outcome; several studies reported both. Only six studies (20%) reported an association specific to African Americans. Empirical evidence linking parental preconception adversity with offspring physical health in African Americans is limited and mixed. In the current literature, very few studies report evidence addressing intergenerational associations between parental preconception adversity and offspring physical health in the African-American population, specifically, and even fewer investigate forms of parental preconception adversity that have been shown to disproportionately affect African Americans (e.g., racism). To better understand root causes of racial health disparities, more rigorous systematic research is needed to address how intergenerational transmission of historical and ongoing race-based trauma may impact offspring health among African Americans.

Introduction

African Americans (AAs) are more likely than whites to experience poor health throughout the lifespan (Carnethon et al., 2017; Mehta et al., 2013). Historical trauma theory (Sotero, 2006) suggests that this is due to the unique history of race-based adversity experienced by AAs: slavery, economic marginalization, ongoing systemic violence, and discrimination. AAs also experience increased prevalence of adversity that is common across all races (e.g., domestic violence; Boardman & Alexander, 2011; Roberts et al., 2011), suggesting that AAs experience multiple forms of significant adversity (i.e., stress, trauma) with effects that may have rippled across generations and contributed to the widespread health inequalities seen today.

Historical trauma theory (Sotero, 2006) posits that affected groups experience physical, psychological, and economic disparities that persist across generations. These disparities also contribute to AAs being at greater risk for adversities experienced across all races, such as adverse childhood experiences (ACEs), environmental exposures, and stress across several domains (e.g., financial, relationship; Boardman & Alexander, 2011; Roberts et al., 2011). Moreover, race-specific adversity (e.g., institutional racism, interpersonal discrimination) permeates multiple domains of life for AA families (DeGue et al., 2016; Williams & Collins, 2001). Importantly, these experiences have been linked to several negative physiological consequences and physical health outcomes.

For populations experiencing historical trauma, research suggests that the adversity they disproportionately experience (e.g., ACEs, discrimination, low socioeconomic status or SES; Pager & Western, 2012; Sacks & Murphey, 2018) is more likely to result in epigenetic alterations (Conching & Thayer, 2019) that can affect gene expression and produce biological dysfunction. Such changes have been identified in several domains including the immune

(Dhabhar, 2014), neuroendocrine (Marsland et al., 2017), and cardiovascular (Hill et al., 2017) systems, epigenetic aging (Brody et al., 2016), and the methylation of genes involved in immune responses and threat-related amygdala reactivity (Houtepen et al., 2016). Specific examples include exposure to racism and discrimination being associated with lower parasympathetic cardiac modulation as measured by heart-rate variability (HRV; Hill et al., 2017) and several other indicators of poor health (Lewis et al., 2015). These physiologic correlates of discrimination and racism likely increase risk for cardiovascular disease (Barber et al., 2016) and other chronic health problems (Mouzon et al., 2017). Beyond negatively impacting individuals directly exposed to adversity, a growing body of empirical work has illustrated how these health consequences can also be observed across generations and how they may occur.

Understanding intergenerational transmission

Several mechanisms are thought to link adversity experienced in one generation with a future generation's physical health (Choi et al., 2017). Investigators have mainly explored pregnant mothers and how negative exposures *during the prenatal period* are associated with increased risk of poor offspring health (Thayer & Kuzawa, 2011). The developmental origins of health and disease (DOHaD) hypothesis describes a period of great epigenetic elasticity during fetal development occurring simultaneously with the transfer of hormones and other information between the mother and child (Kuzawa & Quinn, 2009). Consequently, the intrauterine environment plays an instrumental role in shaping the offspring epigenome. Maternal mood and stress during pregnancy are associated with DNA methylation in offspring tissues which was associated with greater offspring central adiposity and body mass index (Cao-Lei et al., 2015) among several other negative health outcomes. Ultimately, this work suggests that prenatal

maternal adversity can cause harmful epigenetic patterns in offspring through intrauterine signaling with serious long-term health repercussions.

Two other research literatures also address potential mechanisms by which maternal adversity experienced *before pregnancy* (henceforth preconception) may contribute to behaviors that impact the health and epigenome of future generations. In the first, early-life stress (e.g., ACEs) is associated with greater risk of early pregnancy during adolescence (Madigan et al., 2014); in the second, teen pregnancies are linked to increased risk of intrauterine growth restriction (Malabarey et al., 2012), low birth weight (LBW), and preterm birth (PTB; Torvie et al., 2015). Importantly, these neonatal outcomes have implications for subsequent offspring physical health, including greater body fat percentage and insulin resistance (Crume et al., 2014) and metabolic syndrome (Parkinson et al., 2013). However, direct associations between parental ACEs in one generation and physical health outcomes in subsequent generations are infrequently studied and focused almost exclusively on maternal, as opposed to paternal, adversity. Consequently, limited work has explored across generations to determine whether parental preconception adversity is directly linked to children's health, with even less work accounting for how fathers' adversity experiences may play a role in this potential link.

Intergenerational transmission of historical trauma and health

The intergenerational health consequences of historical trauma experienced by specific populations have been studied primarily among Holocaust survivors and Indigenous populations. Holocaust survivors' children often experience reduced cortisol excretion, lower overall cortisol levels (Bierer et al., 2014), and changes in DNA methylation of stress regulatory genes (Yehuda et al., 2016). For Indigenous populations, studies have highlighted the intergenerational impact of Indian Residential Schools documenting that children from families with at least one parent or

grandparent attendee report poorer self-rated health and higher rates of chronic and infectious diseases (Wilk et al., 2017). When it comes to exploring similar issues in the AA community, empirical work has shown links between several forms of exposure to racism and adverse offspring outcomes (Bower et al., 2018; Dominguez, 2011; Slaughter-Acey et al., 2016), but overwhelmingly focuses on prenatal exposure to these specific forms of adversity. As a result, there is a need to examine closely the evidence for intergenerational health associations with respect to distinct experiences of historical trauma (e.g., discrimination, racism) in this population prior to conception.

Overview of the present review

Research has documented that *prenatal* maternal stress is associated with offspring health, and that parental preconception adversity has potential behavioral repercussions (e.g., teen pregnancy), which may have consequences for offspring physical health. However, researchers less often explore direct links between parental *preconception* adversity and their *offspring's* physical health, especially in AAs. Furthermore, while recent research has explored the intergenerational health impacts of historical and ongoing adversity in Holocaust survivors and Indigenous populations, less is known about the empirical work addressing how the unique, preconception adversity experiences of the AA population may affect their offspring's physical health outcomes across generations. Consequently, this review examines this literature with the goal of providing a synopsis and potential roadmap for future work in this important area of research.

Method

We conducted a computerized, systematic search of five electronic databases (CINAHL, PsycINFO, PubMed, Scopus, Web of Science) through June 2021 to identify empirical studies

addressing the intergenerational links between parental preconception adversity and offspring physical health outcomes. This review is registered in PROSPERO under protocol CRD42018105369. Studies consistent with the following inclusion criteria were reviewed:

1. Reports data from AAs living in the U.S.
2. Includes participants from at least 2 separate generations of the same family (e.g., mother/father and daughter/son)
3. Measures at least 1 form of parental adversity that:
    a. Is measured at the individual level for the parent and not reported by the offspring
    b. Occurred prior to the conception of the specific offspring in the study
4. Includes a measure of at least 1 physical health outcome in the offspring gathered via independent information sources (e.g., medical records), offspring self-report, or parent report
5. Examines the association between parental preconception adversity and the index child's physical health outcome.

Justification for inclusion criteria

Inclusion criteria were partially established through the identification of several related systematic reviews (Alhusen et al., 2017; Gone et al., 2019), but were further adapted to address the specific aims of the current review. Due to the unique historical and ongoing adverse experiences of AAs in the U.S. (Alexander, 2010; Anderson, 2016), this review included only studies focusing on individuals and families residing in the U.S. Given the research documenting the offspring health consequences of historical trauma in Indigenous populations and Holocaust survivors (e.g., depressive symptoms, epigenetic changes; Walls & Whitbeck, 2012; Yehuda et al., 2016), and the historical experience of AAs in the U.S., examining similar intergenerational

processes in AAs is needed. Furthermore, we only included studies reporting data from at least two separate generations of AAs from the same family as this is essential for exploring the intergenerational effects of parental adversity on offspring physical health. Studies must have clearly assessed parental *preconception* adversity to understand the intergenerational health impacts of parental adversity beyond what has already been established in the prenatal stress literature.

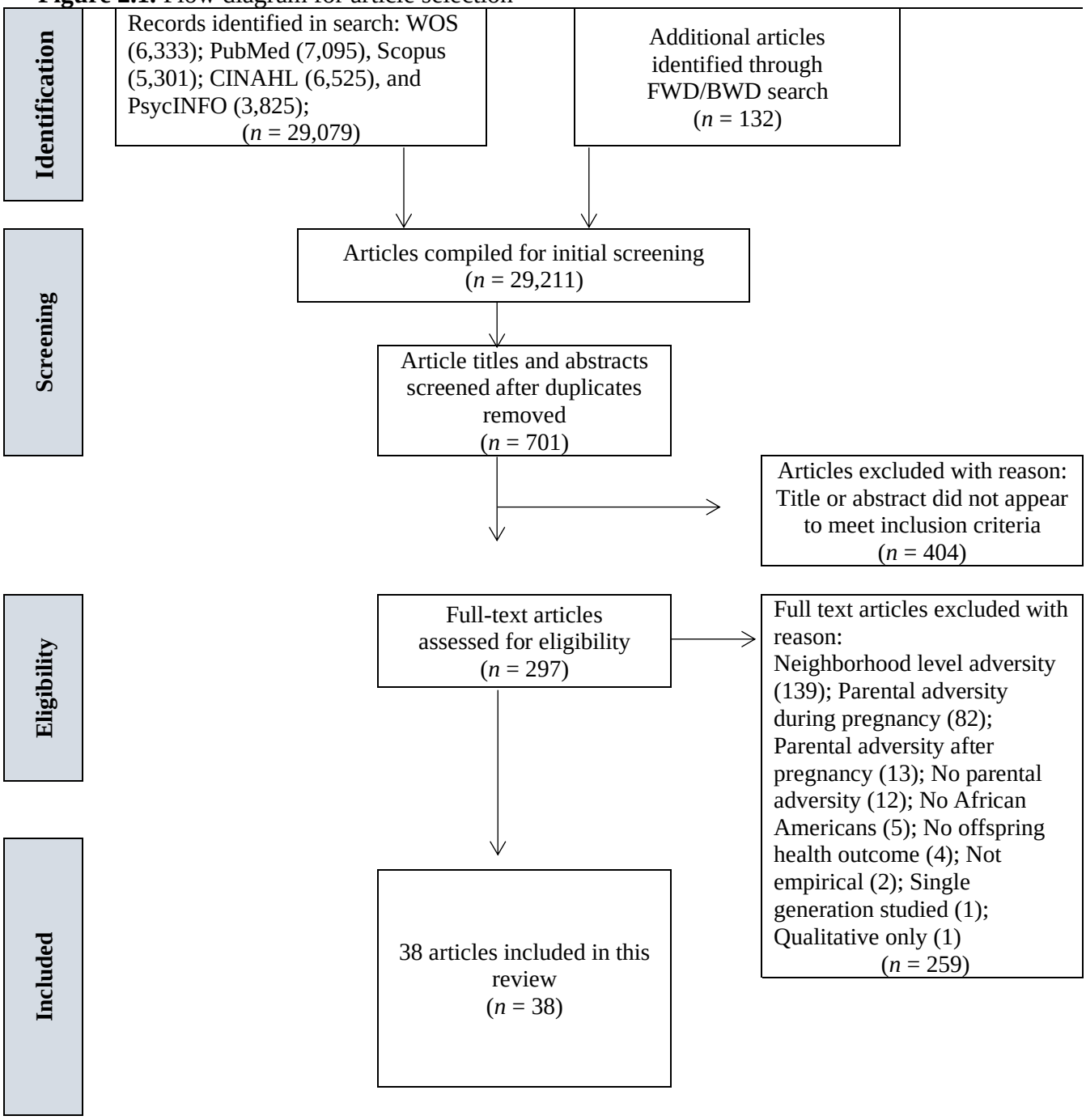
We included studies examining only individual-level parental adversity due to the difficulty in gauging the direct impact of neighborhood-level experiences on individuals and their families, and its possible confounding the link between parental adversity and child health. Additionally, we only included studies that captured adversity exposure directly reported by the parent; studies in which offspring reported on their parent's adverse experiences were omitted due to concerns about the accuracy of these accounts as offspring may not be fully aware of their parents' lifetime exposures. Finally, we included studies reporting at least one measure of offspring physical health with a particular focus on those that captured these outcomes through independent information sources (e.g., medical charts) and offspring self-report as these present the most objective and least biased measures. Although parent report of offspring physical health is subject to considerable bias as parents may be reluctant to disclose their offspring's physical health status candidly or may unknowingly report health issues incorrectly, we also included these studies in the review to capture how they compare to studies using independent and less biased measures.

Procedure

Keyword, controlled vocabulary, or MeSH term combinations were constructed to represent each component of the review topic (see Appendix A). Searches were restricted to

English-language journal articles and dissertations. Two independent reviewers conducted all searches separately and performed an initial screening of articles by title. Next, the abstracts of relevant articles identified by title were reviewed and those appearing to meet inclusion criteria were further assessed for eligibility by examining the full text of the article. All results were compared at each step and any discrepancies were resolved by the two independent reviewers (J.S. and A.A.) and an advisor (E.A.H.) through a consensual, iterative process. The two coders agreed on 78% and negotiated 22% of the articles when reviewing article titles. After reviewing article abstracts, the coders agreed on 89% and negotiated 11% of the articles for full review. Following full-text review, coders demonstrated 95% agreement and negotiated 5% of the final collection of articles. The two independent reviewers also conducted a forward and backward search of the included articles (i.e., they screened articles that were cited by or cited these articles) to identify and add any additional articles meeting inclusion criteria to the final collection (see Figure 2.1). Lastly, the grey literature was assessed using list-servs of American Psychological Association's (APA) Division 38 (Society for Health Psychology) and 56 (Trauma Psychology), as well as the International Society for Traumatic Stress Studies' (ISTSS) Intergenerational Transmission of Trauma and Resilience special interest group (SIG), asking for any relevant studies that met inclusion criteria.

Figure 2.1. Flow diagram for article selection



Data synthesis

Two authors (J.S. and A.A.) reviewed all articles that met the full inclusion criteria and extracted data to create a table of evidence (see Appendices B-E). All authors then reviewed and

discussed findings to identify patterns in associations reported between parental preconception adversity and child physical health. A.A. and J.S. also conducted quality assessments of all included articles using the Newcastle-Ottawa Scale criteria (NOS; Wells et al., 2000; see Appendices F, H, J).

Quality assessment

A.A. and J.S. conducted quality assessments of each article from the 30 studies reviewed using the NOS (Wells et al., 2000; see Appendices F-K). Most studies (n = 23, 77%) were assessed with an adapted version of the NOS for cohort studies based on Kansagara et al. (2017); six studies (20%) were analyzed with a NOS adaptation for cross-sectional studies based on Herzog et al. (2013) while one study was assessed using an adaptation for case-control studies. A large majority of studies reviewed (n = 27, 90%) were classified as having either a moderate or high risk of bias for several reasons including having inadequate or incomplete participant response rate information, non-representative or small samples, retrospective measures of parental preconception adversity, parental report of offspring health, and not accounting for important confounders (e.g., prenatal adversity, current stress levels, offspring exposure to adversity, race/ethnicity). As a result, the quality of the current studies significantly limits our ability to provide a comprehensive assessment of the association between parental preconception adversity and offspring health in AA families.

Results

Search results

Results from the three-step process used to determine eligibility for inclusion is depicted in Figure 2.1. The initial search results returned 6,333 articles in Web of Science (WOS), 7,095 articles in PubMed, 6,525 articles in CINAHL, 5,301 articles in Scopus, 3,825 articles in

PsycInfo, and 132 articles identified through the backward and forward search for a total of 29,211 results. After deleting duplicates, 701 articles remained; after reviewing article titles, 404 articles were dropped because the titles did not mention relevant topics. Next, the abstracts of the remaining 297 articles were assessed for eligibility. Of these articles, 259 were excluded because: parental adversity was assessed at the neighborhood level (n = 139), not explicitly measured during the preconception period (e.g., during pregnancy; n = 82), or after pregnancy (n = 13); no parental adversity was measured (n = 12); the sample did not include AAs or did not give the percentage of the sample that was AA (n = 5); no offspring physical health outcome was reported (n = 4); the study was not empirical (n = 2); only a single generation was studied (n = 1); and results were qualitative (n = 1). Ultimately, 38 articles representing 30 unique studies were included in the review.

Study characteristics

Appendices B-E present the characteristics and key findings of the 38 papers from these 30 studies. Each appendix covers one of four categories: studies with entirely AA study samples (n = 5; see Appendix B); studies with partial AA study samples that examine the role of race in the association between parental preconception adversity and offspring physical health (n = 5; see Appendix C); studies with partial AA study samples that do not examine the role of race in the association between parental preconception adversity and offspring physical health (n = 10; see Appendix D); and studies with parent-reported offspring health outcomes (n = 10; see Appendix E). Most of the studies were published after 2010 (n = 25, 83%); most studies used a cohort design (n = 24, 80%); and six studies used a single group cross-sectional design (n = 6, 20%). Nineteen studies used retrospective (63%), and 11 used prospective (37%) approaches. It

is also important to note that three separate studies produced eleven articles, resulting in more than 30 total entries in the appendices.

Sample characteristics

Sample sizes ranged greatly with the smallest including 31 participants and the largest including 9,350; the median was 493. Three studies produced eleven articles that were included in this review; one study produced five articles (Cheng et al., 2016; Witt et al., 2014a, 2014b, 2015, 2016), one published four articles (Cammack et al., 2019; Flagg et al., 2014; Ihongbe 2018; Strutz et al., 2014), a third study yielded two articles (Brunst et al., 2017; Sternthal et al., 2011). This left 30 unique study samples, six of which (20%) included only AAs, six (20%) had >50% AA participants, and the remaining 18 (60%) had <50% AA respondents. Most of the 30 unique studies used convenience samples (n = 25, 83%), two had clinical samples (7%), and three utilized nationally representative samples (10%).

Parental adversity measures

All 30 studies focused on *maternal* adversity. Most studies measured maternal childhood adversity (n = 21, 70%) or general lifetime adversity (n = 4, 13%); two studies reported both in separate articles; three other studies (10%) explored race-specific adversity. Of the 21 studies measuring childhood adversity, ten (48%) captured ACEs in general; four (19%) focused on childhood SES; three (14%) focused specifically on childhood abuse; two (10%) assessed general childhood stress (e.g., assault, loss, physical danger); one measured early-life neighborhood conditions (e.g., disorder, social control, violence); and another measured both ACEs and childhood SES. Four studies included general lifetime adversity measures (e.g., bereavement, economic strain, adulthood abuse, relationship problems) and traumatic events (e.g., disasters, interpersonal trauma). Two of these studies (10%) reported both childhood

adversity (e.g., abuse, early-life neighborhood conditions, SES; Cammack et al., 2019; Sternhal et al.,2011) and general lifetime adversity (e.g., stressful events, trauma; Brunst et al., 2017; Strutz et al., 2014). Lastly, three studies (14%) measured race-specific adversity including exposure to several forms of racism and racial discrimination in childhood and adolescence (e.g., direct, indirect, vicarious).

Offspring health outcomes

Eighteen studies (60%) included independent reports of offspring health (e.g., biological data, medical records, offspring report) exclusively while nine studies (30%) only included parent-reported offspring health measures; three studies (10%) included both independent and parent-reported offspring health. Most studies reported health outcomes that were captured at birth (n = 24, 80%) while two (7%) measured outcomes at four months of age; the remaining four studies' (13%) outcomes were measured between birth and seventeen years of age. Because many studies reported more than one offspring health outcome (n = 12, 40%), the outcome numbers reported below may not add up to exactly 30. The most common health outcomes measured were infant birth weight (n = 13, 43%), birth timing or gestational age (n = 10, 33%), and premature or PTB status (n = 9, 30%). Other infant-specific health outcomes included stillbirth (n = 3, 10%), fetal growth measures (n = 2, 7%), respiratory sinus arrhythmia (RSA; n = 2, 7%), and miscarriage (n = 2, 7%). Admission to special care nursery and the length of hospital stay were each captured only once across the studies. Finally, several child health outcomes related to asthma (e.g., control, cytokine production, diagnosis; n = 3, 10%), cord blood immunoglobulin E (IgE) levels (n = 1, 3%), obesity status (n = 1, 3%), overall health status (n = 1, 3%), startle response (n = 1, 3%), and HRV (n = 1, 3%) were measured.

Parental adversity & independently reported offspring health

Studies using 100% AA samples. Table 2.1 reflects a brief tally of all results of the review; Appendix B provides a detailed summary of the five studies that had 100% AA samples and compared independently reported health of children whose mothers reported preconception adversity to children whose mothers did not. Four studies captured offspring birth outcomes (e.g., birth timing, birth weight, fetal growth) and one study explored adolescent outcomes (e.g., child HRV, startle response; see Appendix B). Parental preconception adversity was significantly associated with poor offspring physical health in four of these studies. Specifically, maternal early-life adversity (e.g., cumulative stress, neighborhood disorder) was significantly associated with birth timing in two studies (Gillespie et al., 2017; Sealy-Jefferson et al., 2019); maternal childhood abuse (e.g., emotional, physical) was significantly associated with heightened offspring startle response and HRV ratio (Jovanovic et al., 2011), a physiologic measure previously linked to greater cardiovascular disease risk and all-cause mortality (Fang et al., 2020). Another study linked *indirect* maternal exposure to racism in childhood with offspring LBW (Hilmert et al., 2014).

However, four of these studies also reported non-significant associations between maternal preconception adversity and offspring physical health in AA families (Hilmert et al., 2014; Jovanovic et al., 2011; Rowell, 2020; Sealy-Jefferson et al., 2019). General, maternal childhood adversity (e.g., cumulative ACEs, neighborhood disorder, physical and sexual abuse) was not associated with several birth or early-life offspring outcomes (e.g., birth weight, gestational age; Jovanovic et al., 2011; Rowell, 2020; Sealy-Jefferson et al., 2019), and *direct* maternal exposure to racism in childhood was not associated with fetal growth (Hilmert et al., 2014). Thus, of the five studies with all AA samples, the results were mixed and inconclusive:

four reported that parental adversity was associated with some offspring health outcomes, but four also reported some non-significant findings.

Table 2.1. Results from studies addressing intergenerational transmission of adversity in African-American families

Author	Maternal preconception adversity associated with child health?		Racial differences identified in association between maternal preconception adversity and child health?	
Studies using all African-American samples				
Gillespie et al.	YES		NA	
Hilmert et al.	YES		NA	
Jovanovic et al.	YES		NA	
Rowell	NO		NA	
Sealy-Jefferson et al.	YES		NA	
TOTAL for all AA Samples	YES=4	NO=1		
Studies testing racial differences in link between parental preconception adversity and child health				
Dominguez et al.	YES		YES	
Gray et al.	YES		NO	
Margerison-Zilko et al.	YES		NO	
Masho et al.	NO		NO	
Seng et al.	NO		NO	
TOTAL for testing racial differences	YES=3	NO=2	YES=1	NO=4
Studies not testing racial differences in link between parental preconception adversity and child health				
Blackmore et al.	YES		NA	
Chen et al.	YES		NA	
Cheng et al.[a]	YES		NA	
Cowell et al.	NO		NA	
Freedman et al.	YES		NA	
Jones et al.	YES		NA	
Mersky et al.	YES		NA	
Miller et al.	YES		NA	
Noll et al.	YES		NA	
Smith et al.	YES		NA	
Sternthal et al.[c]	YES		NA	
Witt et al. 2014a[a]	YES		NA	
Witt et al. 2014b[a]	YES		NA	
Witt et al. 2015[a]	YES		NA	

Witt et al. 2016[a]		YES		NA
TOTAL for not testing racial differences	YES=14	NO=1		

Studies with parent-reported offspring health

Astone et al.		YES		NA
Brunst et al.[c]		YES		NA
Cammack et al.[b]		YES		NO
Daniels et al.		YES		NA
Flagg et al.[b]		NO		NO
Freeman et al.		NO		NO
Gavin et al.		YES		NO
Hillis et al.		YES		NO
Ihongbe[b]		YES		NO
Kerkar et al.		YES		NO
Lê-Scherban et al.		YES		NO
Stein et al.		YES		NO
Strutz et al.[b]		YES		NO
TOTAL for parent-reported offspring health	YES=11	NO=2	YES=0	NO=10
GRAND TOTAL	YES=32	NO=6	YES=1	NO=14

Table note. NA refers to not applicable.
[a]These five papers report data from the same study.
[b]These four papers report data from the same study.
[c]These two papers report data from the same study.

Studies testing for racial differences. Five studies that had partial AA samples tested for racial differences in the association between parental preconception adversity and offspring health (see Appendix C). Four of these studies measured offspring birth outcomes (e.g., birth timing, birth weight, gestational age) and one study explored early-life outcomes (e.g., infant RSA). In two studies, maternal childhood adversity (e.g., ACEs, abuse or violence) was associated with PTB (Margerison-Zilko et al., 2017) and infant RSA (Gray et al., 2017) -- an index of parasympathetic nervous system activity (Beauchaine, 2001) that heightens risk for chronic disease (Masi et al., 2007) -- but no differences were found between AA and white mothers in either study. Another study reported that vicarious maternal exposure to racism in

childhood was significantly associated with offspring birth outcomes in AA families but not in white families (Dominguez et al., 2008).

Four of these studies also reported non-significant associations between preconception adversity and offspring physical health (Dominguez et al., 2008; Margerison-Zilko et al., 2017; Masho et al., 2015; Seng et al., 2011). In Dominguez et al., (2008), *direct* maternal exposure to racism in childhood was not linked to offspring birth weight, and three studies found no association between economic strain, loss, child maltreatment, or substance use and offspring birth outcomes in any racial group (Margerison-Zilko et al., 2017; Masho et al., 2015; Seng et al., 2011). Thus, of five studies addressing racial differences in the association between maternal preconception adversity and child health, three studies reported significant associations, but only one of them documented a stronger association in AAs than whites, while four studies also reported non-significant race-specific findings (see Table 2.1).

Studies not testing racial differences. Fifteen articles, representing 11 unique studies, used partial AA samples without testing for racial differences in the association between parental preconception adversity and independently reported offspring health (see Appendix D). Most studies (n = 8, 73%) captured offspring birth outcomes (e.g., admission to special care nursery, birth timing, birth weight, fetal death, fetal growth, length of hospital stay, PTB status) while two (18%) explored early-life outcomes (e.g., cord blood IgE levels, infant RSA) and one (9%) measured adolescent outcomes (e.g., asthma control, cytokine production). Ten of the 11 studies (91%) reported at least one significant association between preconception maternal adversity and offspring physical health outcomes (Blackmore et al., 2016; Chen et al., 2017; Cheng et al., 2016; Freedman et al., 2017; Jones et al., 2019; Mersky & Lee, 2019; Miller et al., 2017; Noll et al., 2007; Smith et al., 2016; Sternthal et al., 2011; Witt et al., 2014a, 2014b; Witt et al., 2015;

Witt et al., 2016). Six of these (55%) examined maternal childhood adversity (e.g., ACEs, sexual abuse) and identified significant links with birth and other early-life outcomes (Blackmore et al., 2016; Freedman et al., 2017; Jones et al., 2019; Mersky & Lee, 2019; Noll et al., 2007; Smith et al., 2016).

Three studies (Chen et al., 2017; Miller et al., 2017; Sternthal et al., 2011) reported significant associations between maternal early life disadvantage (e.g., low childhood SES, childhood family economic hardship) and birth outcomes (Miller et al., 2017) and other early-life and adolescent outcomes (Chen et al., 2017; Sternthal et al., 2011). One study (represented in five articles) – the Early Childhood Longitudinal Study-Birth Cohort – used a nationally representative sample of 9,350 mother-child dyads, and reported significant associations between maternal preconception stressful life events (PSLEs; e.g., bereavement, divorce) and birth outcomes such as very LBW (Cheng et al., 2016; Witt et al., 2014a; Witt et al., 2015; Witt et al., 2016) and PTB (Witt et al., 2014b), but not LBW (Witt et al., 2014a). Finally, one study reported no significant link between maternal ACE exposure and infant birth timing (Cowell et al., 2021). In sum, ten independent studies with multiracial samples reported significant links between maternal preconception adversity and offspring physical health but did not examine racial differences in the strength of these associations while one study reported a non-significant finding (see Table 2.1).

Parental adversity & parent-reported offspring health

Thirteen articles, representing ten unique studies, examined associations between maternal preconception adversity and parent-reported offspring health outcomes (see Appendix E). Of these ten studies, six (60%) had <50% AA respondents, three (30%) had >50% AA participants, and only one included only AAs. Most studies (n = 8, 80%) measured offspring

birth outcomes (e.g., birth timing, birth weight, fetal death) while the remaining two (20%) explored early life outcomes (e.g., asthma diagnosis, obesity status, overall health status). All but one study (n = 9, 90%) reported at least one significant association between preconception maternal adversity and poor offspring physical health (Astone et al., 2007; Brunst et al., 2017; Cammack et al., 2019; Daniels et al., 2020; Gavin et al., 2011; Hillis et al., 2004; Kerkar et al., 2021; Lê-Scherban et al., 2018; Stein et al., 2000). Seven studies (70%) examined maternal childhood adversity (e.g., ACEs, neighborhood social control and disorder, SES), with six identifying at least one significant association with birth outcomes (e.g., timing, weight, fetal death; Astone et al., 2007; Gavin et al., 2011; Hillis et al., 2004; Kerkar et al., 2021; Stein et al., 2000) and other early-life outcomes (e.g., asthma diagnosis, obesity status, overall health; Lê-Scherban et al., 2018). One study reported that AA mothers exposed to vicarious childhood (≤ age 12) racial discrimination and direct adolescent (ages 13-19) racial discrimination had significantly higher PTB risk than AA mothers who were not exposed to such discrimination (Daniels et al., 2020).

Three articles (Cammack et al., 2019; Flagg et al., 2014; Strutz, 2014) and one dissertation (Ihongbe, 2018) reported data from the same National Longitudinal Study of Adolescent to Adult Health ("Add Health"), a large nationally representative sample comprised of over 90,000 adolescents. Findings from this study were mixed suggesting that while maternal preconception adversity (e.g., childhood abuse, chronic stressors, neighborhood violence exposure) was significantly associated with birth outcomes (e.g., birth weight, PTB, very LBW; Cammack et al., 2019; Ihongbe, 2018; Strutz et al., 2014), grandparental exposure to neighborhood disorder was not associated with their grandchild's birth weight (Flagg et al., 2014). Finally, Freeman et al. (2014) found no significant link between maternal early life

poverty and risk of infant LBW. To summarize, ten independent studies investigated associations between maternal preconception adversity and parent-reported offspring physical health outcomes with all but one study reporting significant findings (see Table 2.1).

Mechanisms for intergenerational transmission of adversity

Only seven studies (23%) identified and measured potential mechanisms linking parental preconception adversity with offspring health. Most studies explored how maternal preconception adversity affected various prenatal physiological processes including changes to immune function and inflammation, cortisol levels, hemodynamic factors related to blood pressure (BP), and placental tissue telomere length (TL). In Miller et al. (2017), a panel of maternal inflammatory biomarkers was investigated (interferon-γ; interleukins, or IL- 6, 8, 10, and 13; tumor necrosis factor-α), and IL-6 levels mediated links between maternal childhood disadvantage and several infant outcomes including birth weight, PTB, small for gestational age, length of hospital stay, and admission to special care nursery. Gillespie et al. (2017) showed that maternal cortisol mediated the association between a mother's childhood stress and her offspring's birth timing, but only in women giving birth after spontaneous labor. In contrast, Noll et al. (2007) found that maternal cortisol did not mediate the association between the mother's childhood sexual abuse and her baby's PTB status. Hilmert et al. (2014) reported that greater maternal exposure to indirect racism in childhood interacted with prenatal increases in diastolic BP (DBP) to predict lower infant birth weight. Finally, Jones et al. (2019) demonstrated that placental tissue TL moderated the association between a mother's ACE exposure and infant stress responsivity.

Maternal preconception adversity also demonstrated associations with behavioral and lifestyle factors that have been previously linked to adverse outcomes for newborns. In Smith et

al. (2016), prenatal smoking and substance use accounted for most of the differential impact of maternal ACE exposure on infant birth weight; prenatal smoking was also the strongest mediator of the link between maternal ACEs and her infant's gestational age. Similarly, maternal childhood maltreatment (e.g., childhood sexual abuse) was linked to adolescent substance use and prenatal tobacco and alcohol use, ultimately affecting infant birth weight (Gavin et al., 2011) and PTB status (Noll et al., 2007). Prenatal alcohol use also partially mediated the link between maternal childhood sexual abuse and PTB status (Noll et al., 2007).

Discussion

The literature reviewed provides mixed and inconclusive evidence about the association between maternal preconception adversity and offspring physical health in AA families (see Table 2.2 and Appendices B-E). We reviewed 38 articles, representing 30 unique studies; 25 (83%) of these studies documented that maternal preconception adversity was associated with poor health outcomes in their offspring (e.g., LBW, PTB, RSA). Six (20%) also reported at least one non-significant association, with some studies reporting both. This literature suggests that several types of maternal preconception adversity (e.g., ACEs, overall lifetime adversity, neighborhood disadvantage) may impact a range of birth and early life offspring physical health outcomes in diverse samples. However, findings specifically addressing whether these associations are stronger in AA samples were both limited and quite mixed. Five of the 25 studies reporting a significant association between preconception adversity and poor child health found this association was more likely in AAs who experienced preconception adversity than in AAs who did not (Daniels et al., 2020; Gillespie et al., 2017; Hilmert et al., 2014; Jovanovic et al., 2011; Sealy-Jefferson et al., 2019), one documented a stronger association in AAs than in whites (Dominguez et al., 2008), two found no role for race in the strength of this association

(Gray et al., 2017; Margerison-Zilko et al., 2017), and 19 studies did not examine racial differences (See Appendix D & E). Similarly, preconception exposure to racism in AA moms was also shown to significantly impact offspring health, but these links appear dependent on the type (e.g., direct, indirect/vicarious racism) and timing (e.g., childhood vs. adulthood) of exposure (Dominguez et al., 2008; Hilmert et al., 2014). Thus, while the literature generally suggests that preconception maternal adversity is a risk factor for poor offspring health across demographically diverse samples, the heterogenous nature of adversity and outcome assessments, and control variables used in the different analyses make drawing firm conclusions impossible.

Table 2.2 Summary table of critical findings

Critical Findings
• Literature provides limited, mixed evidence about associations between parental preconception adversity and offspring physical health in AA families • 25 out of 30 unique studies reported significant associations between parental preconception adversity and offspring health; 6 out of 30 reported non-significant associations • Only six studies reported significant associations between parental preconception adversity and offspring physical health that was specific to AAs: 5 compared AAs who reported preconception adversity to AAs who did not, 1 compared AAs who reported preconception adversity to whites • Several studies reported both significant and nonsignificant associations across different offspring health outcomes

Several potential mechanisms linking maternal adversity with offspring health were also suggested. In samples with only AAs, maternal preconception adversity was linked to both prenatal cortisol levels as well as changes in prenatal DBP that were ultimately associated with birth timing and birth weight (Gillespie et al., 2017; Hilmert et al., 2014). Studies including AAs, but not reporting findings exclusive to this group, identified multiple biomarkers (e.g., IL-6,

placental tissue TL) as key mechanisms in the impact of maternal preconception adversity on several infant outcomes (e.g., admission to special care nursery, birth weight, length of hospital stay, PTB, small for gestational age). Lastly, some studies demonstrated significant links between maternal preconception adversity and prenatal behavioral and lifestyle mechanisms (e.g., smoking, substance use) that have been shown in previous work to partially explain negative outcomes for infants.

Limitations of the literature

The literature reviewed herein has several weaknesses that limit our ability to clearly address whether there is an association between parental preconception adversity and offspring health in AAs comprehensively (see Table 2.3). First, 25 (83%) of the 30 unique studies reviewed used relatively small convenience samples, introducing sampling and selection bias which limits the causal interpretation of significant associations identified and renders the findings ungeneralizable to the broader AA population. These biases are further compounded by the fact that most studies did not adequately address important potential confounding variables (e.g., current parental mental/physical health status, child exposure to adversity) that may account for any significant associations identified.

Table 2.3. Implications for research, practice, and policy

Implications
Future research should: o Account for paternal preconception adversity experiences when exploring intergenerational links to offspring health o Capture both general and race-specific parental preconception adversity (e.g., racism) disproportionately affecting AAs using a diverse range of measures simultaneously o Measure offspring health beyond birth/early-life outcomes to examine longer-term repercussions of preconception adversity and identify mechanisms responsible for health repercussions o Conduct prospective, longitudinal studies that assess adversity and outcomes as they occur, not retrospectively
Practitioners need to: o Assess adversity to identify families at greatest risk for potential health impacts of adversity across generations in AA community o Conduct research to develop and test interventions that target the mechanisms linking parental preconception adversity with offspring health in the AA community
Policy o To address health disparities that affect AAs, funding is needed for rigorous longitudinal research examining the impact of parental preconception adversity on offspring health across the lifespan

Moreover, only 20% (n = 6) used an all-AA sample (comparing AAs with vs. without preconception adversity), and four of these studies had fewer than 100 participants (Gillespie et al., 2017; Hilmert et al., 2014; Jovanovic et al., 2011; Rowell, 2020); 60% of studies with multiracial samples had <50% AA respondents, further compounding concerns about sampling bias. Studies using these relatively small, convenience samples also lack statistical power which, when combined with sampling/selection biases, limits the applicability of the findings. An additional 17% (n = 5) of studies used multiracial samples and tested for racial differences, but most studies with multiracial samples (n = 19) did not test for racial differences. This is important because when interpreting both significant and non-significant findings from these

studies, the degree to which these associations apply to AAs specifically and whether divergent findings for AAs are being obscured by larger racial groups within the samples is not clear. Finally, multiple articles reported data from the same nationally representative studies including the Asthma Coalition on Community Environment and Social Stress project (ACCESS; Brunst et al., 2017; Sternthal et al., 2011); the Early Childhood Longitudinal Study-Birth Cohort (Cheng et al. 2016; Witt et al., 2014a, 2014b; Witt et al., 2015; Witt et al., 2016); and the National Longitudinal Study of Adolescent to Adult Health (Add Health; Cammack et al., 2019; Flagg et al., 2014; Ihongbe, 2018; Strutz et al., 2014). Although the use of these nationally representative samples makes the reported article findings more generalizable, they each represent just one study with evidence linking different forms of parental adversity with different offspring outcomes because they come from the same sample.

Moreover, the overwhelming majority of the 30 independent studies focused on birth and early-life outcomes (n = 26, 87%) providing limited evidence for the longer-term repercussions of parental adversity on offspring physical health. Although adverse birth outcomes may initiate a lifetime of poor health (Crume et al., 2014; Parkinson et al., 2013), it is not clear from these studies what role parental adversity plays in this process or what mechanisms might explain subsequent poor health. Knowing more about the root causes of offspring health outcomes and the mechanisms linking them with parental preconception adversity in AA families could inform the development of public health interventions seeking to interrupt the intergenerational transmission of trauma's negative health effects.

All but one of the studies included in this review (Noll et al., 2007) relied on retrospective parental reports of preconception adversity, which introduces substantial retrospective recall bias. Reports of distressing events from one's past are subject to recall bias

because respondents may not remember previous events accurately, may omit details or entire events, or unknowingly revise past memories, especially when the events being asked about happened several years before (Widom, 2019). Inaccurate reporting of past life events may prevent researchers from correctly identifying the specific parental adverse experiences associated with offspring health. Furthermore, social-desirability bias may result in underreporting these events despite being assured that their responses are anonymous or confidential due to a desire for their responses to be viewed favorably by others. Such underreporting may compromise the ability to detect potential associations with offspring outcomes. Assessments of parental preconception adversity were also quite disparate and this lack of consistency in measurement further limits our ability to draw conclusions about the types of parental adversity that may be more detrimental to child health. Finally, this literature currently suffers from sex-based, gender-role biases regarding the health impact of parental adversity as all included studies exclusively measured maternal (not paternal) adversity. This is a significant omission because recent work suggests that paternal preconception adversity can impact offspring health through genetic and epigenetic changes to sperm (Braun et al., 2017).

It is also important to note that 90% of studies in this review assessed universal forms of adversity (e.g., ACEs, overall lifetime adversity, neighborhood disadvantage) commonly experienced across all racial groups, while only three addressed race-based adversity. That is, very few studies addressed the link between parental preconception exposure to race-specific adversity (e.g., discrimination, racism) and offspring health. Past work documents that AAs experience these specific adversities in several life domains at disproportionate rates (DeGue et al., 2016; Williams & Collins, 2001) and they can be particularly damaging due to their complex nature. These adversities can occur on multiple levels (e.g., cultural, institutional, interpersonal),

ultimately undermine positive views of the self, diminish social relationships and the sense of belonging, and interfere with overall quality of life (Brondolo et al., 2017). Furthermore, they include acute events that can also become persistent stressors when recurring instances occur over prolonged periods or when they produce additional adversity exposures and there are limited resources available to address them. Importantly, empirical work has suggested that the health impacts of these specific experiences may be transmitted across generations (Hill et al., 2017; Lewis et al., 2015). Given that AAs have historically experienced race-based adversity unlike that of most groups in the U.S. (except Indigenous Americans; e.g., slavery, segregation), there remains a need to address the impact of the unique adversities experienced by AA parents (e.g., anti-Black racism) on their offspring's health if we are to fully interrogate the roots of racial health disparities seen today.

Future directions

This body of literature is underdeveloped in several ways, making it challenging to draw any strong conclusions. Further studies that include larger, nationally, or regionally representative AA samples are needed to increase the generalizability of findings. Alternatively, the use of nationally representative multiracial, multiethnic samples could be used if researchers examine racial and ethnic differences in the associations between parental preconception adversity and offspring health. Beyond the use of retrospective methods, identifying populations as early in life as possible before conception takes place and following them longitudinally would be a more accurate way to measure adverse experiences and their impact on offspring health. In addition, more consistent efforts should be made to intentionally capture a diverse range of parental adversity during several distinct time periods (e.g., childhood, adulthood, preconception, prenatal) within the same study and emphasize statistical analyses that provide

opportunities to disentangle the intergenerational health impacts of adversity experienced at specific time periods. For example, being able to account for the presence of prenatal adversity when exploring associations between preconception adversity and offspring health can help more accurately characterize the impact of preconception adversity and highlight potential mediating factors. This body of literature may also benefit from studies that employ a diverse range of measures (e.g., surveys or interviews, biological data) and utilize them simultaneously to capture the impact of adversity more comprehensively.

When it comes to offspring health, future studies should examine a wider array of outcomes to better understand the impact of parental preconception adversity. The current literature overwhelmingly addresses birth and infancy outcomes (e.g., weight, development); while they are important indicators of early life health, a more comprehensive assessment of health outcomes as children progress into adolescence and adulthood is needed to identify the long-term repercussions of parental preconception adversity. By including health data that encompass the child's developmental trajectory, investigators can access a greater assortment of physical health measures (e.g., biological, observational, survey) gathered directly from offspring, that are more accurate than parental reports, and may reflect intergenerational adversity's health impact across the lifespan. Furthermore, it may provide measures that are more proximal to physical health abnormalities that can ultimately serve as indicators for some of the ailments and chronic diseases that disproportionately affect AA adults (Carnethon et al., 2017; Mehta et al., 2013).

Future research should also examine how paternal experiences of preconception adversity may affect offspring health and the unique mechanisms that are responsible for this transmission from fathers to children. While some evidence suggests that maternal preconception adversity

may be associated with offspring health trajectories (e.g., Mahrer et al., 2020), it is also important to explore how paternal, preconception adversity may affect offspring health. Focusing on fathers provides the advantage of also accounting for the potential impact of parental experiences on offspring health beyond the direct biological repercussions of maternal experiences through the uterine environment (Braun et al., 2017). Identifying and measuring potential mechanisms responsible for intergenerational transmission of health impacts by capturing biological measures (e.g., epigenetic changes, inflammatory biomarkers, cortisol, telomere length), behavioral (e.g., parental substance use), and other factors simultaneously should also be a strong focus, as well as how these factors may interact with maternal mechanisms to affect future generations' health. Such work is essential to beginning to understand the intergenerational health impacts of paternal preconception adversity for AAs. More specifically, it may help us better understand how race-based adversities experienced disproportionately by AA boys and men (e.g., police encounters, incarceration) may be associated with offspring health relative to other, more general adversity (e.g., poverty, violence). Indeed, it is crucial for future studies to tease apart the unique impacts of different types of preconception adversity on offspring health so that the specific impact of racialized trauma on the intergenerational transmission of health disparities in AA families can be identified. Finally, it would allow us to address the unique impact of paternal adversity relative to maternal adversity, and how they interact to shape offspring health.

Conclusion

This review provides mixed evidence about the intergenerational impacts of parental preconception adversity on offspring physical health in AA families. Most studies investigated general adversity (e.g., ACEs, early-life disadvantage) and birth-related outcomes rather than

race-specific adversity (e.g., racism) and chronic diseases known to disproportionately affect AAs. Several potential mechanisms responsible for these intergenerational health impacts were also identified and measured. Most studies used multiracial samples without addressing racial differences or reporting findings exclusive to the AA population. Given the historical and ongoing adversity (e.g., racism, systemic violence) and health disparities experienced by AAs, exploring how preconception adversity may affect health across generations is essential. Doing so may help explain the many health disparities observed among the AA population.

References

Alexander, M. (2010). *The new Jim Crow: Mass incarceration in the age of colorblindness*. New York: The New Press.

Alhusen, J. L., Bower, K. M., Epstein, E., & Sharps, P. (2016). Racial discrimination and adverse birth outcomes: An integrative review. *Journal of Midwifery & Women's Health, 61(6),* 707-720. doi:10.1111/jmwh.12 490

Anderson, C. (2016). *White Rage*: *The unspoken truth of our racial divide.* Bloomsbury Press, U.S.A.

Astone, N. M., Misra, D., & Lynch, C. (2007). The effect of maternal socio-economic status throughout the lifespan on infant birthweight. *Paediatric and Perinatal Epidemiology, 21(4),* 310-318. doi:10.1111/j.1365-3016.2007.00821.x

Barber, S., Hickson, D. A., Wang, X., Sims, M., Nelson, C., & Diez-Roux, A. V. (2016). Neighborhood disadvantage, poor social conditions, and cardiovascular disease incidence among African American adults in the Jackson Heart Study. *American Journal of Public Health, 106(12),* 2219-2226. doi:10.2105/AJPH.2016.303471

Beauchaine, T. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13(2),* 183-214. doi:10.1017/S0954579401002012

Bierer, L. M., Bader, H. N., Daskalakis, N. P., Lehrner, A., Makotkine, I., Seckl, J. R., & Yehuda, R. (2014). Elevation of 11β-hydroxysteroid dehydrogenase type 2 activity in Holocaust survivor offspring: Evidence for an intergenerational effect of maternal trauma exposure. *Psychoneuroendocrinology, 48,* 1-10. doi:10.1016/j.psyneuen.2014.06.001

Blackmore, E. R., Putnam, F. W., Pressman, E. K., Rubinow, D. R., Putnam, K. T., Matthieu, M.

M., Gilchrist, M. A., Jones, I., & O'Connor, T. G. (2016). The effects of trauma history and prenatal affective symptoms on obstetric outcomes. *Journal of Traumatic Stress, 29(3),* 245-252. doi:10.1002/jts.22095

Boardman, J. D., & Alexander, K. B. (2011). Stress trajectories, health behaviors, and the mental health of black and white young adults. *Social Science & Medicine, 72(10),* 1659-1666. doi:10.1016/j.socscimed.2011.03.024

Bower, K. M., Geller, R. J., Perrin, N. A., & Alhusen, J. (2018). Experiences of racism and preterm birth: Findings from a pregnancy risk assessment monitoring system, 2004 through 2012. *Women's Health Issues, 28(6),* 495-501. doi:10.1016/j.whi.2018.06.002

Braun, J. M., Messerlian, C., & Hauser, R. (2017). Fathers matter: Why it's time to consider the impact of paternal environmental exposures on children's health. *Current Epidemiology Reports, 4(1),* 46-55. doi:10.1007/s40471-017-0098-8

Brody, G. H., Miller, G. E., Yu, T., Beach, S. R., & Chen, E. (2016). Supportive family environments ameliorate the link between racial discrimination and epigenetic aging: A replication across two longitudinal cohorts. *Psychological Science, 27(4),* 530-541. doi:10.1177/0956797615626703

Brondolo, E., Byer, K., Gianaros, P. J., Liu, C., Prather, A. A., Thomas, K., Keita, G., & Woods-Giscombé, C. L. (2017). Stress and health disparities: Contexts, mechanisms, and interventions among racial/ethnic minority and low socioeconomic status populations. *American Psychological Association (APA) Working Group Report*. Retrieved from http://www.apa.org/pi/health-disparities/resources/stress-report.aspx

Brunst, K. J., José Rosa, M., Jara, C., Lipton, L. R., Lee, A., Coull, B. A., & Wright, R. J.

(2017). Impact of maternal lifetime interpersonal trauma on children's asthma: Mediation through maternal active asthma during pregnancy. *Psychosomatic Medicine, 79(1),* 91-100. doi:10.1097/PSY.0000000000000354.

Cammack, A. L., Hogue, C. J., Drews-Botsch, C. D., Kramer, M. R., & Pearce, B. D. (2019). Associations between maternal exposure to child abuse, preterm birth, and very preterm birth in young, nulliparous women. *Maternal and Child Health Journal, 23(6),* 847-857. doi:10.1007/s10995-018-02709-w

Cao-Lei, L., Dancause, K. N., Elgbeili, G., Massart, R., Szyf, M., Liu, A., Laplante, D. P., & King, S. (2015). DNA methylation mediates the impact of exposure to prenatal maternal stress on BMI and central adiposity in children at age 13½ years: Project Ice Storm. *Epigenetics, 10(8),* 749-761. doi:10.1080/15592294.2015.1063771

Carnethon, M. R., Pu, J., Howard, G., Albert, M. A., Anderson, C. A., Bertoni, A. G., Mujahid, M. S., Palaniappan, L., Taylor Jr, H. A., Willis, M., & Yancy, C. W. (2017). Cardiovascular health in African Americans: A scientific statement from the American Heart Association. *Circulation, 136(21),* e393-e423. doi:10.1161/CIR.0000000000000534

Chen, E., Shalowitz, M. U., Story, R. E., Ehrlich, K. B., Manczak, E. M., Ham, P. J., Van Le, B. S., & Miller, G. E. (2017). Parents' childhood socioeconomic circumstances are associated with their children's asthma outcomes. *Journal of Allergy and Clinical Immunology, 140(3),* 828-835. doi:10.1016/j.jaci.2016.11.040

Cheng, E. R., Park, H., Wisk, L. E., Mandell, K. C., Wakeel, F., Litzelman, K., Chatterjee, D., &

Witt, W. P. (2016). Examining the link between women's exposure to stressful life events prior to conception and infant and toddler health: The role of birth weight. *Journal of Epidemiology and Community Health, 70(3),* 245-252. doi:10.1136/jech-2015-205848

Choi, K. W., Sikkema, K. J., Vythilingum, B., Geerts, L., Faure, S. C., Watt, M. H., Roos, A., & Stein, D. J. (2017). Maternal childhood trauma, postpartum depression, and infant outcomes: Avoidant affective processing as a potential mechanism. *Journal of Affective Disorders, 211,* 107-115. doi:10.1016/j.jad.2017.01.004

Conching, A. K. S., & Thayer, Z. (2019). Biological pathways for historical trauma to affect health: A conceptual model focusing on epigenetic modifications. *Social Science & Medicine, 230,* 74-82. doi:10.1016/j.socscimed.2019.04.001

Cowell, W., Taing, L., Askowitz, T., Bosquet Enlow, M., Hacker, M. R., & Wright, R. J. (2021). Associations of maternal trait anger expression and lifetime traumatic and non-traumatic experiences with preterm birth. *Maternal and Child Health Journal, 25(4),* 635-644. doi:10.1007/s10995-020-03026-x

Crume, T. L., Scherzinger, A., Stamm, E., McDuffie, R., Bischoff, K. J., Hamman, R. F., & Dabelea, D. (2014). The Long-term impact of intrauterine growth restriction in a diverse US cohort of children: The EPOCH study. *Obesity, 22(2),* 608-615. doi:10.1002/oby.20565

Daniels, K. P., Valdez, Z., Chae, D. H., & Allen, A. M. (2020). Direct and vicarious racial discrimination at three life stages and preterm labor: Results from the African American Women's Heart & Health Study. *Maternal and Child Health Journal, 24(11),* 1387-1395. doi:10.1007/s10995-020-03003-4

DeGue, S., Fowler, K. A., & Calkins, C. (2016). Deaths due to use of lethal force by law

enforcement: Findings from the national violent death reporting system, 17 US states, 2009–2012. *American Journal of Preventive Medicine, 51(5),* S173-S187. doi:10.1016/j.amepre.2016.08.027

Dhabhar, F. S. (2014). Effects of stress on immune function: The good, the bad, and the beautiful. *Immunologic Research, 58(2),* 193-210. doi:10.1007/s12026-014-8517-0

Dominguez, T. P. (2011). Adverse birth outcomes in African American women: The social context of persistent reproductive disadvantage. *Social Work in Public Health, 26(1),* 3-16. doi:10.1080/10911350902986880

Dominguez, T. P., Dunkel-Schetter, C., Glynn, L. M., Hobel, C., & Sandman, C. A. (2008). Racial differences in birth outcomes: The role of general, pregnancy, and racism stress. *Health Psychology, 27(2),* 194-203. doi:10.1037/0278-6133.27.2.194

Fang, S. C., Wu, Y. L., & Tsai, P. S. (2020). Heart rate variability and risk of all-cause death and cardiovascular events in patients with cardiovascular disease: A meta-analysis of cohort studies. *Biological Research for Nursing, 22(1),* 45-56. doi:10.1177/1099800419877442

Flagg, L. A., Needham, B. L., & Locher, J. L. (2014). Neighborhood disadvantage, preconception health behaviors and infant birthweight: A preliminary study. *International Journal of Contemporary Sociology, 51(1),* 7-25.

Freedman, A. A., Cammack, A. L., Temple, J. R., Silver, R. M., Dudley, D. J., Stoll, B. J., Varner, M. W., Saade, G. R., Conway, D., Goldenberg, R. L., & Hogue, C. J. (2017). Maternal exposure to childhood maltreatment and risk of stillbirth. *Annals of Epidemiology, 27(8),* 459-465. doi:10.1016/j.annepidem.2017.07.005

Freeman, L. L. (2014). *Cumulative inequality and race/ethnic disparities in low birthweight: Differences by early life SES* [Unpublished doctoral dissertation]. Rice University.

Gavin, A. R., Hill, K. G., David Hawkins, J., & Maas, C. (2011). The role of maternal early life and later-life risk factors on offspring low birth weight: Findings from a three-generational study. *Journal of Adolescent Health, 49(2),* 166-171. doi:10.1016/j.jadohealth.2010.11.246

Gillespie, S. L., Christian, L. M., Alston, A. D., & Salsberry, P. J. (2017). Childhood stress and birth timing among African American women: Cortisol as biological mediator. *Psychoneuroendocrinology, 84*, 32-41. doi:10.1016/j.psyneuen.2017.06.009

Gone, J. P., Hartmann, W. E., Pomerville, A., Wendt, D. C., Klem, S. H., & Burrage, R. L. (2019). The impact of historical trauma on health outcomes for indigenous populations in the USA and Canada: A systematic review. *American Psychologist, 74(1)*, 20-35. doi:10.1037/amp0000338

Gray, S. A., Jones, C. W., Theall, K. P., Glackin, E., & Drury, S. S. (2017). Thinking across generations: Unique contributions of maternal early life and prenatal stress to infant physiology. *Journal of the American Academy of Child & Adolescent Psychiatry, 56(11),* 922-929. doi:10.1016/j.jaac.2017.09.001

Herzog, R., Álvarez-Pasquin, M. J., Díaz, C., Del Barrio, J. L., Estrada, J. M., & Gil, Á. (2013). Are healthcare workers' intentions to vaccinate related to their knowledge, beliefs and attitudes? A systematic review. *BMC Public Health, 13,* 154. doi:10.1186/1471-2458-13-154

Hill, L. K., Richmond, A. S., Hoggard, L. S., Gray, D. L., Williams, D. P., & Thayer, J. F. (2017). Examining the association between perceived discrimination and heart rate variability in African Americans. *Cultural Diversity and Ethnic Minority Psychology, 23(1),* 5-14. doi:10.1037/cdp0000076.

Hillis, S. D., Anda, R. F., Dube, S. R., Felitti, V. J., Marchbanks, P. A., & Marks, J. S. (2004). The association between adverse childhood experiences and adolescent pregnancy, long-term psychosocial consequences, and fetal death. *Pediatrics, 113(2),* 320-327. doi:10.1542/peds.113.2.320

Hilmert, C. J., Dominguez, T. P., Dunkel Schetter, C., Srinivas, S. K., Glynn, L. M., Hobel, C. J., & Sandman, C. A. (2014). Lifetime racism and blood pressure changes during pregnancy: Implications for fetal growth. *Health Psychology, 33(1),* 43-51. doi:10.1037/a0031160

Houtepen, L. C., Vinkers, C. H., Carrillo-Roa, T., Hiemstra, M., van Lier, P. A., Meeus, W., Branje, S., Heim, C. M., Nemeroff, C. B., Mill, J., Schalkwyk, L. C., Creyghton, M. P., Kahn, R. S., Joëls, M., Binder, E. B., & Boks, M. P. (2016). Genome-wide DNA methylation levels and altered cortisol stress reactivity following childhood trauma in humans. *Nature Communications, 7(1),* 1-10. doi:10.1038/ncomms10967

Ihongbe, T. O. (2018). *The impact of mother–father relationship, social support and neighborhood context on preterm birth* [Unpublished doctoral dissertation]. Virginia Commonwealth University.

Jones, C. W., Esteves, K. C., Gray, S. A., Clarke, T. N., Callerame, K., Theall, K. P., & Drury, S. S. (2019). The transgenerational transmission of maternal adverse childhood experiences (ACEs): Insights from placental aging and infant autonomic nervous system reactivity. *Psychoneuroendocrinology, 106,* 20-27. doi:10.1016/j.psyneuen.2019.03.022

Jovanovic, T., Smith, A., Kamkwalala, A., Poole, J., Samples, T., Norrholm, S. D., Ressler, K. J., & Bradley, B. (2011). Physiological markers of anxiety are increased in children of abused mothers. *Journal of Child Psychology and Psychiatry, 52(8),* 844-852. doi:10.1111/j.1469-7610.2011.02410.x

Kansagara, D., O'Neil, M., Nugent, S., Freeman, M., Low, A., Kondo, K., Elven, C., Zahker, B., Motu'apuaka, M., Paynter, R., & Morasco, B. J. (2017). Benefits and harms of cannabis in chronic pain or post-traumatic stress disorder: A systematic review. Evidence-based Synthesis Program Report. Washington (DC): Department of Veterans Affairs.

Kerkar, S., Shankar, A., Boynton-Jarrett, R., & Harville, E. W. (2021). Adverse childhood experiences are associated with miscarriage in adulthood: The GROWH study. *Maternal and Child Health Journal, 25(3),* 479-486. doi:10.1007/s10995-020-03079-y

Kuzawa, C. W., & Quinn, E. A. (2009). Developmental origins of adult function and health: Evolutionary hypotheses. *Annual Review of Anthropology, 38,* 131-147. doi:10.1146/annurev-anthro-091908-164350

Lê-Scherban, F., Wang, X., Boyle-Steed, K. H., & Pachter, L. M. (2018). Intergenerational associations of parent adverse childhood experiences and child health outcomes. *Pediatrics, 141(6),* e20174274. doi:10.1542/peds.2017-4274

Lewis, T. T., Cogburn, C. D., & Williams, D. R. (2015). Self-reported experiences of discrimination and health: Scientific advances, ongoing controversies, and emerging issues. *Annual Review of Clinical Psychology, 11,* 407-440. doi:10.1146/annurev-clinpsy-032814-112728

Madigan, S., Wade, M., Tarabulsy, G., Jenkins, J. M., & Shouldice, M. (2014). Association between abuse history and adolescent pregnancy: A meta-analysis. *Journal of Adolescent Health, 55(2),* 151-159. doi:10.1016/j.jadohealth.2014.05.002

Mahrer, N. E., Guardino, C. M., Hobel, C., & Dunkel Schetter, C. (2020). Maternal stress before conception is associated with shorter gestation. *Annals of Behavioral Medicine, 55(3),* 242-252. doi:10.1093/abm/kaaa047

Malabarey, O. T., Balayla, J., Klam, S. L., Shrim, A., & Abenhaim, H. A. (2012). Pregnancies in young adolescent mothers: A population-based study on 37 million births. *Journal of Pediatric and Adolescent Gynecology, 25(2),* 98-102. doi:10.1016/j.jpag.2011.09.004

Margerison-Zilko, C. E., Strutz, K. L., Li, Y., & Holzman, C. (2017). Stressors across the life-course and preterm delivery: Evidence from a pregnancy cohort. *Maternal and Child Health Journal, 21(3),* 648-658. doi:10.1007/s10995-016-2151-5

Marsland, A. L., Walsh, C., Lockwood, K., & John-Henderson, N. A. (2017). The effects of acute psychological stress on circulating and stimulated inflammatory markers: A systematic review and meta-analysis. *Brain, Behavior, and Immunity, 64,* 208-219. doi:10.1016/j.bbi.2017.01.011

Masho, S. W., Price, S. K., Kinser, P. A., & Jallo, N. (2015). Racial disparities in the association between stress and preterm birth. *Journal of Health Disparities Research and Practice, 8(4),* 80-92.

Masi, C. M., Hawkley, L. C., Rickett, E. M., & Cacioppo, J. T. (2007). Respiratory sinus arrhythmia and diseases of aging: Obesity, diabetes mellitus, and hypertension. *Biological Psychology, 74(2),* 212-223. doi:10.1016/j.biopsycho.2006.07.006

Mehta, N. K., Lee, H., & Ylitalo, K. R. (2013). Child health in the United States: Recent trends in racial/ethnic disparities. *Social Science & Medicine, 95,* 6-15. doi:10.1016/j.socscimed.2012.09.011

Mersky, J. P., & Lee, C. P. (2019). Adverse childhood experiences and poor birth outcomes in a diverse, low-income sample. *BMC Pregnancy and Childbirth, 19(1),* 387. doi:10.1186/s12884-019-2560-8

Miller, G. E., Culhane, J., Grobman, W., Simhan, H., Williamson, D., Adam, E. K., Buss, C.,

Entringer, S., Kim, K., Garcia-Espana, J. F., Keenan-Devlin, L., McDade, T. W., Wadhwa, P. D., & Borders, A. (2017). Mothers' childhood hardship forecasts adverse pregnancy outcomes: Role of inflammatory, lifestyle, and psychosocial pathways. *Brain, Behavior, and Immunity, 65*, 11-19. doi:10.1016/j.bbi.2017.04.018

Mouzon, D. M., Taylor, R. J., Woodward, A. T., & Chatters, L. M. (2017). Everyday racial discrimination, everyday non-racial discrimination, and physical health among African-Americans. *Journal of Ethnic & Cultural Diversity in Social Work, 26(1-2)*, 68-80. doi:10.1080/15313204.2016.1187103

Noll, J. G., Schulkin, J., Trickett, P. K., Susman, E. J., Breech, L., & Putnam, F. W. (2007). Differential pathways to preterm delivery for sexually abused and comparison women. *Journal of Pediatric Psychology, 32(10)*, 1238-1248. doi:10.1093/jpepsy/jsm046

Pager, D., & Western, B. (2012). Identifying discrimination at work: The use of field experiments. *Journal of Social Issues, 68(2)*, 221-237. doi:10.1111/j.1540-4560.2012.01746.x

Parkinson, J. R., Hyde, M. J., Gale, C., Santhakumaran, S., & Modi, N. (2013). Preterm birth and the metabolic syndrome in adult life: A systematic review and meta-analysis. *Pediatrics, 131(4)*, e1240-e1263. doi:10.1542/peds.2012-2177

Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine, 41(1)*, 71-83. doi:10.1017/S0033291710000401

Rowell, T. A. (2020). *Examining the impact of pregnant black women's adverse childhood*

experiences through maternal health and birth outcomes [Unpublished doctoral dissertation]. Kent State University.

Sacks V., & Murphey, D. (2018). The prevalence of adverse childhood experiences, nationally, by state, and by race or ethnicity. *Center for Victim Research Repository*. https://ncvc.dspacedirect.org/handle/20.500.11990/1142.

Sealy-Jefferson, S., Mustafaa, F. N., & Misra, D. P. (2019). Early-life neighborhood context, perceived stress, and preterm birth in African American women. *SSM-Population Health, 7*, 100362. doi:10.1016/j.ssmph.2019.100362

Seng, J. S., Low, L. K., Sperlich, M., Ronis, D. L., & Liberzon, I. (2011). Post-traumatic stress disorder, child abuse history, birthweight and gestational age: A prospective cohort study. *BJOG: An International Journal of Obstetrics & Gynaecology, 118(11)*, 1329-1339. doi:10.1111/j.1471-0528.2011.03071.x

Slaughter-Acey, J. C., Sealy-Jefferson, S., Helmkamp, L., Caldwell, C. H., Osypuk, T. L., Platt, R. W., Straughen, J. K., Dailey-Okezie, R. K., Abeysekara, P., & Misra, D. P. (2016). Racism in the form of micro aggressions and the risk of preterm birth among black women. *Annals of Epidemiology, 26(1)*, 7-13. doi:10.1016/j.annepidem.2015.10.005

Smith, M. V., Gotman, N., & Yonkers, K. A. (2016). Early childhood adversity and pregnancy outcomes. *Maternal and Child Health Journal, 20(4)*, 790-798. doi:10.1007/s10995-015-1909-5

Sotero, M. (2006). A conceptual model of historical trauma: Implications for public health practice and research. *Journal of Health Disparities Research and Practice, 1(1)*, 93-108.

Stein, J. A., Lu, M. C., & Gelberg, L. (2000). Severity of homelessness and adverse birth outcomes. *Health Psychology, 19(6)*, 524-534. doi:10.1037/0278-6133.19.6.524

Sternthal, M. J., Coull, B. A., Chiu, Y. H., Cohen, S., & Wright, R. J. (2011). Associations among maternal childhood socioeconomic status, cord blood IgE levels, and repeated wheeze in urban children. *Journal of Allergy and Clinical Immunology, 128(2)*, 337-345.E1. doi:10.1016/j.jaci.2011.05.008

Strutz, K. L., Hogan, V. K., Siega-Riz, A. M., Suchindran, C. M., Halpern, C. T., & Hussey, J. M. (2014). Preconception stress, birth weight, and birth weight disparities among US women. *American Journal of Public Health, 104(8)*, e125-e132. doi:10.2105/AJPH.2014.301904

Thayer, Z. M., & Kuzawa, C. W. (2011). Biological memories of past environments: Epigenetic pathways to health disparities. *Epigenetics, 6(7)*, 798-803. doi:10.4161/epi.6.7.16222

Torvie, A. J., Callegari, L. S., Schiff, M. A., & Debiec, K. E. (2015). Labor and delivery outcomes among young adolescents. *American Journal of Obstetrics and Gynecology, 213(1)*, 95.e1-95.e8. doi:10.1016/j.ajog.2015.04.024

Walls, M. L., & Whitbeck, L. B. (2012). The intergenerational effects of relocation policies on indigenous families. *Journal of Family Issues, 33(9)*, 1272-1293. doi:10.1177/0192513X12447178

Wells, G. A., Shea, B., O'Connell, D., Peterson, J., Welch, V., Losos, M., & Tugwell, P. (2000). The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Accessed August 31, 2021. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp

Widom, C. S. (2019). Are retrospective self-reports accurate representations or existential recollections? *JAMA Psychiatry, 76(6)*, 567-568. doi:10.1001/jamapsychiatry.2018.4599

Wilk, P., Maltby, A., & Cooke, M. (2017). Residential schools and the effects on Indigenous

health and well-being in Canada—A scoping review. *Public Health Reviews, 38(8)*, 1-23. doi:10.1186/s40985-017-0055-6

Williams, D. R., & Collins, C. (2001). Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Reports, 116*, 404-416. doi:10.1093/phr/116.5.404

Witt, W. P., Cheng, E. R., Wisk, L. E., Litzelman, K., Chatterjee, D., Mandell, K., & Wakeel, F. (2014a). Maternal stressful life events prior to conception and the impact on infant birth weight in the United States. *American Journal of Public Health, 104(S1)*, S81-S89. doi:10.2105/AJPH.2013.301544

Witt, W. P., Cheng, E. R., Wisk, L. E., Litzelman, K., Chatterjee, D., Mandell, K., & Wakeel, F. (2014b). Preterm birth in the United States: The impact of stressful life events prior to conception and maternal age. *American Journal of Public Health, 104(S1)*, S73-S80. doi:10.2105/AJPH.2013.301688

Witt, W. P., Mandell, K. C., Wisk, L. E., Cheng, E. R., Chatterjee, D., Wakeel, F., Park, H., & Zarak, D. (2016). Infant birthweight in the US: The role of preconception stressful life events and substance use. *Archives of Women's Mental Health, 19(3)*, 529-542. doi:10.1007/s00737-015-0595-z

Witt, W. P., Park, H., Wisk, L. E., Cheng, E. R., Mandell, K., Chatterjee, D., & Zarak, D. (2015). Neighborhood disadvantage, preconception stressful life events, and infant birth weight. *American Journal of Public Health, 105(5)*, 1044-1052. doi:10.2105/AJPH.2015.302566

Yehuda, R., Daskalakis, N. P., Bierer, L. M., Bader, H. N., Klengel, T., Holsboer, F., & Binder, E. B. (2016). Holocaust exposure induced intergenerational effects on FKBP5 methylation. *Biological Psychiatry, 80(5)*, 372-380. doi:10.1016/j.biopsych.2015.08.005

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Chapter 3: Associations between adversity and health outcomes within and across generations of African-American families

Abstract

Adversity throughout the lifespan has been associated with negative health not only in the individual experiencing it, but also in their offspring. Although these associations have been explored in populations exposed to collective trauma due to their group affiliation (e.g., Holocaust survivors, Indigenous Americans), less is known about these associations in the African-American community. Furthermore, little is known about the potential differences that timing and type of adversity have on intergenerational health impacts in African-American families. A dyadic sample of African-American mothers and their adult children (N = 57 dyads) was used to investigate whether several types of maternal adversity were related to their child's health and whether the specific timing of adversity was associated with offspring health outcomes. Utilizing generalized estimating equations for dyadic analysis, findings showed that maternal reports of preconception general adversity were associated with a higher number of offspring-reported, doctor-diagnosed health ailments after controlling for adversity reported during other time periods and offspring adversity (IRR, 1.05; 95% CI: 1.00-1.11). Maternal reports of post-conception law enforcement-related adversity were associated with better self-rated health in their offspring (unstandardized b = -.22, SE = .07, z = -3.08, p = .002). Findings highlight the importance of both timing and type of maternal adversity when exploring links to offspring health. Findings also demonstrate how maternal adversity can be linked to adult offspring health while controlling for offspring's own adversity exposure. Findings highlight the importance of accounting for the specific timing and type of maternal adversity when exploring intergenerational health impacts in African Americans.

Introduction

As we have observed in prior chapters, the evidence exploring intergenerational associations between parental adversity and offspring health in African Americans (AAs) is currently limited in several ways (Sweeting, Akinyemi, & Holman, 2022). First, the empirical literature has primarily focused on intergenerational health impacts of *either* general adversity *or* race-specific forms of adversity (e.g., discrimination, racism). Moreover, there are essentially no studies that juxtapose multiple types of adversity to explore whether differences exist in the relative impact of specific types of parental adversity on offspring health. Next, many studies have failed to account for the specific timing of parental adversity, and none have attempted to disentangle how adversity experienced at a certain time may be related to offspring outcomes, while controlling for adversity experienced at other times. This is important because there is a large literature documenting the link between prenatal adversity and child outcomes (Cao-Lei et al., 2015; Chan, Nugent, & Bale, 2018; Eberle, Fasig, Brüseke, & Stichling, 2021), but knowledge of how parental adversity experienced before conception (e.g., childhood, adulthood prior to child's conception; henceforth preconception) is linked to offspring health outcomes is limited. As a result, more rigorous studies are needed to address these gaps by examining the association between parental adversity and offspring health in AA families more comprehensively.

Importance of adversity type

Numerous single-generation studies have identified how certain forms of adversity may differ in their health impact relative to others (Friedman, Montez, Sheehan, Guenewald, & Seeman, 2015; Negriff, 2020; Nelson, Bhutta, Harris, Danese, & Samara, 2020). Despite this evidence and the higher rates at which AAs report experiencing multiple forms of adversity,

most intergenerational health studies of AAs have captured universal forms of adversity only (Gillespie, Christian, Alston, & Salsberry, 2017; Margerison-Zilko, Strutz, Li, & Holzman, 2017), with few addressing race-based adversity specifically (Daniels, Valdez, Chae, & Allen, 2020; Hilmert et al., 2014). Furthermore, there are essentially no studies that capture multiple adversity types from parents and attempt to identify their relative impact on offspring health. Thus, prior research fails to ascertain whether disparities in the magnitude of health impacts depend on the type of adversity experienced.

General adversity. The bulk of intergenerational health studies with samples including AAs have captured general parental adversity in numerous forms, including adverse childhood experiences (ACEs), lifetime adversity, neighborhood conditions, socioeconomic status (SES), stressful life events, and violence exposure. In relation to these forms of adversity, links have been made to several birth and early-life offspring outcomes such as birth timing, birth weight, and stress reactivity (Cammack et al., 2019; Cheng et al., 2016; Gray et al., 2017; Margerison-Zilko et al., 2017). This evidence suggests that the impacts of general adversity remain robust regardless of race and that these may not occur in ways that are unique to AA families.

Racial discrimination and racism. The limited attention to race-specific adversity is a significant oversight as AAs have been exposed to persistent discrimination, racism, and violence on multiple levels (e.g., interpersonal, institutional) since the abolishment of slavery in the 1860's. Discrimination and racism are thought to be uniquely harmful forms of adversity due to their pervasive nature, their occurring across life domains on several levels, extending beyond single events into reoccurring experiences, and having distinct health consequences (Brondolo et al., 2017). Importantly, experiencing these adversities has been linked to a range of negative health outcomes including lower parasympathetic cardiac modulation (Hill et al., 2017) and

several other indicators of poor health (Lewis et al., 2015) that have ultimately been connected to greater risk for cardiovascular disease (Barber et al., 2016) and other chronic health problems (Mouzon et al., 2017).

Racial discrimination can occur for AAs in a variety of domains (e.g., education, employment, housing) and one important contributing factor is SES. SES often affects access to different resources and is impacted considerably by one's education and income (Williams et al., 2016). Prior work documenting discrimination in educational contexts has shown that Black students are more likely to be expelled and suspended, receive out-of-school suspensions for minor behavior, and experience severe punishment through court action or notification of the police than their white peers (Rocque, 2010; Skiba et al., 2011; Welch & Payne, 2010). This is significant because these experiences can impede academic progress as well as student achievement (Arcia, 2006; Perry & Morris, 2014) and negatively impact one's ability to improve their SES. With regard to the income component of SES, similar discriminatory trends have been highlighted in employment outcomes for AAs as they are treated worse than people of other races (Chavez, Ornelas, Lyles, & Williams, 2015), are more likely to be contacted about jobs with lower starting salaries and less prestige (Gaddis, 2015), and are significantly more likely to be laid off compared to their white peers (Elvira & Zatzick, 2002; Park & Sandefur, 2003). Together, these forms of discrimination are interrelated in shaping one's SES and can affect other outcomes that contribute to health status for families.

Residential segregation is another type of discrimination that appears in the context of housing, is disproportionately experienced by AAs, and can be further exacerbated by SES by limiting where families are able to live. AAs residing in heavily segregated areas tend to have access to poorer quality housing, lower quality education, fewer employment opportunities,

fewer food sources, fewer recreational facilities, limited health care options, and more sources of environmental toxins (Williams & Collins, 2016). Furthermore, segregation has been shown to have a negative impact on health outcomes for AAs in the form of increased odds of having low birth weight babies, chronic inflammation, and cardiovascular disease (Barber et al., 2016; Simons et al., 2018; Walton, 2009) which can ultimately impact the health of subsequent generations. Importantly, this is a rather unique association for AAs as other groups such as Asians and Hispanics living in ethnic enclaves have been shown to experience protective effects of segregation through buffers for acculturative stress, sources of social support, and help in coping with race-related stressors (Walton, 2009). An additional way in which residential segregation can negatively impact health outcomes has been highlighted in the health care realm. Discrimination and racism have been uncovered through implicit bias towards AAs, or the unconscious influence of stereotypes towards a group that contributes to judgment of and behavior toward people from this group (Devine, 1989). This has been demonstrated in the form of lower referral rates for thrombolysis, a reduced likelihood of providing opioids for Black children, greater perceptions of physician verbal dominance, and less positive perceptions of physician interactions by AA patients compared to their white counterparts (Cooper et al., 2012; Green et al., 2007; Sabin & Greenwald, 2012). Ultimately, having limited access to high quality health care as a function of SES can affect not only the health of parents, but their children as well.

Racial discrimination in law enforcement. The unequal experiences of racial discrimination and racism have been widely and often publicly observed in the context of U.S. law enforcement, making it a rather unique form of race-based adversity. People of African descent encounter law enforcement officials (e.g., police) at disproportionate rates and

experience more detrimental outcomes during these encounters. A 2015 Bureau of Justice Statistics Special Report concluded that AAs were more likely to experience street stops and more likely to be the driver in a traffic stop compared to white and Hispanic Americans (Davis, Whyde, & Langdon, 2018). In addition to an increased likelihood of being stopped by police, research has also shown that Black Americans are three times more likely than white Americans to report the use of force or being threatened by police (Davis, Whyde, & Langdon, 2018). This is significant because law enforcement encounters in general can involve several stressful components such as fear, humiliation, and violations of one's sense of personal freedom, resulting in feelings of disrespect and helplessness (Brunson & Miller, 2006; Friedman, Lurigio, Greenleaf, & Albertson, 2004). Importantly, these stress responses can be further heightened when encounters are violent or result in physical injury (Jackson, Fahmy, Vaughn, & Testa, 2019). A developing body of literature demonstrates that law enforcement encounters among AAs may be associated with a wide range of adverse repercussions for health and well-being including asthma, diabetes, financial strain, greater body weight, lower academic engagement and performance, sleep deprivation and poor sleep quality, poorer mental health, and fatal injuries (Alang, McAlpine, McCreedy, & Hardeman, 2017; Jackson, Testa, Vaughn, & Semenza, 2020; McLeod, Heller, Manze, & Echeverria, 2020; Sewell & Jefferson, 2016; Zeiders, Umaña-Taylor, Carbajal, & Pech, 2021).

With respect to law enforcement encounters that end fatally, evidence suggests that AAs account for nearly 25% of people shot and killed by police ("Fatal Fore: 2018 police shootings database," 2018) despite only accounting for roughly 13% of the U.S. population ("U.S. Census Bureau QuickFacts," 2021). Furthermore, they are killed by police at a rate that is more than twice the rate of white Americans with many of them being unarmed. Apart from the damaging

consequences for victims and their loved ones, police killings can also affect the health and well-being of AAs not directly connected to the killings due to both the national media and social media's ability to transmit news instantly and universally (Bor, Venkataramani, Williams, & Tsai, 2018). Traumatic events like racism in the form of police killings can be experienced vicariously (Harrell, 2000) and contribute to diminished well-being in numerous ways, including elevated perceptions of systemic racism and lack of fairness (Harrell, Hall, & Taliaferro, 2003), increased fear of victimization and higher mortality expectations, activation of earlier traumas, communal bereavement, and feelings of anger (Williams & Williams-Morris, 2000). Regardless of direct familial connections to victims of fatal police violence, AAs may be susceptible to the health outcomes commonly associated with bereavement, such as cardiovascular risk, chronic pain, inflammation, and risk of stroke (Aalbaek, Graff, & Vestergaard, 2017; Ennis & Majid, 2021).

Timing of parental adversity

Another factor that is important to consider when exploring intergenerational health impacts of parental adversity experiences concerns the timing of when adversity is experienced in one's life. As explained by the developmental origins of health and disease (DOHaD) hypothesis (Kuzawa & Quinn, 2009), a great deal of work has drawn links between parental adverse experiences during the prenatal period and an array of unfavorable offspring health outcomes, including impaired inflammation and respiratory outcomes, motor skills, and metabolic function, diminished cognitive development, greater mental health problems, and increased risk of obesity as well as infant mortality (Cao-Lei et al., 2020; Van den Bergh et al., 2020; Walsh et al., 2019).

Another body of research stemming from the seminal Adverse Childhood Experiences (ACE; Felitti et al., 1998) study explores how childhood adversity can be especially salient in shaping subsequent health outcomes (Borsini, Hepgul, Mondelli, Chalder, & Pariante, 2014; Carr, Martins, Stingel, Lemgruber, & Juruena, 2013; McKay et al., 2021; Sweeting, Garfin, Holman, & Silver, 2020). Building on these findings, other work has suggested that health consequences of parental childhood adversity can also be transmitted across generations and increase the risk of asthma symptoms, impaired nervous system function, low birth weight, and preterm birth in their offspring (Chen et al., 2017; Gray, Jones, Theall, Glackin, & Drury, 2017; Mersky & Lee, 2019).

A less studied time period in intergenerational health exploration deals with experiences occurring in adulthood prior to the conception of a child (i.e., after age 18, but before the prenatal period). Studies currently tend to capture "preconception" adversity in a manner that groups childhood events with those that may have happened in adulthood (see Cheng et al., 2016). By doing so, these studies are unable to establish whether significant links to offspring health are due to childhood or adulthood preconception adversity experiences and if there are differences in the impact that these experiences may have relative to each other. Consequently, no projects have captured parental adversity during childhood, adulthood before their child is conceived, and the prenatal period within the same sample and attempted to further characterize the intergenerational health impacts of parental adversity in specific phases of life while accounting for others (e.g., exploring links between childhood adversity and offspring health while controlling for prenatal adversity).

Other gaps in intergenerational health studies

Finally, the current literature addresses birth and early life outcomes (e.g., birth weight, infant development) frequently and while these outcomes are important indicators of long-term health, a more comprehensive picture of outcomes beyond birth and infancy is needed to better understand the long-term associations between parental exposure to adversity and the health of future generations. Capturing offspring outcomes later in life also provides the advantage of a more diverse range of health assessment tools (e.g., biological, survey) that can be collected from offspring directly and serve as more accurate gauges for the ailments and chronic diseases that are prevalent in AA adults. When doing so, it becomes necessary to account for the offspring's own exposure to adversity, along with their parents' adversity, to disentangle the impact that various sources and types of adversity may have. In the single study that examines offspring health beyond birth and early life in relation to parental adversity described in the previous chapter (see Chen et al., 2017), the offspring's own adversity exposure was not accounted for and thus represents an important area of focus in subsequent studies.

Overview of the Present Study

Recruiting a dyadic sample of AA parents and adult biological children, this study captured adversity experiences and health outcomes using confidential online surveys. The primary goals of this study were to explore whether different types and timing of parental adversity are associated with their child's health, while also controlling for the child's adversity exposure. This study also explored whether different types and timing of adversity were associated with health within each generation. Parents provided a detailed account of their lifetime adversity experiences across multiple domains, several measures of health, and a collection of demographic indicators. Similarly, offspring reported their experiences with

multiple types of adversity, several health measures, and demographic information. Through the collection of several health measures and a detailed account of adversity exposure, a comprehensive picture of health status was obtained and subsequently examined in relation to adversity exposure both within and across generations of AA families.

Research questions and hypotheses

RQ1: Is lifetime adversity associated with health outcomes within each generation of AA families? (i.e., Are parental and offspring adversity exposures linked to parental and offspring health outcomes, respectively?)

H1: Greater lifetime adversity exposure will be associated with poorer health.

RQ2: Are different types of adversity (e.g., general, law enforcement, racial discrimination) associated with health outcomes within each generation of parents and offspring in AA families?

H2: Law enforcement adversity and racial discrimination will be more strongly associated with health outcomes than will general adversity within each generation of AA families.

RQ3: Is the timing of the adversity (childhood before age 18, age 18 to before conception, post-conception) differentially associated with health outcomes in AA parents?

H3: Adversity experienced in childhood and from age 18 to before conception will be more strongly associated with health outcomes in parents than adversity experienced post-conception.

RQ4: Is the timing of the adversity (childhood before age 18, age 18 and after) differentially associated with health outcomes in AA offspring?

H4: Childhood adversity will have a greater association with health outcomes in offspring than adversity experienced after age 18.

RQ5: Is there an association between parental adversity exposure and offspring health outcomes?

H5: Greater parental adversity exposure will be associated with poorer offspring health.

RQ6: Are types of parental adversity differentially associated with offspring health?

H6: Law enforcement adversity and racial discrimination will have a greater association with offspring health than will general adversity.

RQ7: Is the timing of parental adversity (e.g., childhood, preconception, post-conception) differentially associated with offspring health?

H7: Parental adversity will be differentially associated with offspring health as a function of the timing.

Methods

Sample recruitment

A sample of 57 dyads (N = 114) comprised of African-American adults and one of their biological parents were recruited in several ways between September 26th, 2021 and March 31st, 2022. After completing the University of California, Irvine's Institutional Review Board (IRB) self-assessment tool for exempt research, contact was made with seven historically-Black

colleges and universities (HBCUs) across the United States describing the scope of the study. A total of seven different HBCUs shared recruitment materials with affiliated parents or students, comprised of roughly 9,800 people who received some form of study solicitation; the frequency of recruitment material distribution ranged from two single occasions to eleven consecutive, weekly disseminations across HBCUs.

Recruitment through parents. The first recruitment strategy involved getting in contact with parent-focused organizations affiliated with several HBCUs, including Florida Agricultural and Mechanical University (FAMU) and Spelman College. At FAMU, personnel from the Efferson Student Union and Activities group were contacted, sent the study recruitment materials, and the materials were shared with an e-mail list of approximately 200 parents of current FAMU students who signed up for FAMU's annual Parents and Family Weekend. Similarly, connections with personnel from the Spelman College Parents and Family Association were made and study recruitment materials were posted within the private Facebook group containing approximately 300 Spelman College parents. Recruitment materials shared with parents contained a description of the goals of the present study, the eligibility criteria, an explanation of how data collection for the study would be conducted, contact information for the Lead Researcher, and a link to start the survey. The eligibility criteria for parents included: at least 18 years old, have Internet access, identify as having African descent (e.g., African American, Black, African, Afro-Caribbean, etc.), and are the biological parent of a child who is at least 18 years old and willing to complete a separate, confidential online survey. Neither FAMU nor Spelman College required IRB approval for the distribution of study recruitment materials.

Recruitment through children. IRB approval was first obtained from several participating institutions including Jackson State University, Kentucky State University, Morehouse College, and North Carolina Central University. Upon receiving IRB approval, school officials sent recruitment e-mail messages to university-wide list-servs of current students at Jackson State University (approximately 7,000 students) and Kentucky State University (approximately 1,800 students). For Morehouse College, recruitment materials and messages were shared through the Psychology Department's Blackboard page as well as through emails to students in individual courses in the Biology and English departments reaching approximately 250 students. At North Carolina Central University, recruitment messages were sent by e-mail to students in a general psychology course containing approximately 250 students. A brief virtual presentation was also made during class to further encourage participation by describing the aims, benefits, and importance of the study. Recruitment materials were also distributed to approximately 30 students from a single general psychology course at Bennett College, but IRB was not required due to only sharing materials with a single class.

Beyond HBCUs, recruitment materials and messages were disseminated through several entities and organizations affiliated with UC Irvine and targeting students including the Black Student Union (BSU), the African American Studies Department, the Leadership Education to Advance Diversity: African, Black, and Caribbean (LEAD-ABC), Center for Black Cultures, Resources & Research (CBCRR), and Black Graduate Students at UC Irvine. Finally, recruitment materials were shared among several virtual, social media accounts and groups across Facebook and GroupMe comprised of predominantly AA students (e.g., Black Graduate Students in Psychology, HBCU Alumni, HBCU Connect, UC-HBCU Ph.D. students).

Recruitment materials shared with students included a description of the goals of the present study, the eligibility criteria (at least 18 years old, have Internet access, identify as having African descent, have a biological parent who is willing to complete a separate, confidential online survey), an explanation of how data collection for the study would be conducted, contact information for the Lead Researcher, and a link to a Study Information Page developed within UCI's Qualtrics platform. The Study Information Page reiterated the goals of the present study, the eligibility criteria, an explanation of how data collection for the study would be conducted, contact information for the Lead Researcher, and spaces for interested respondents to provide e-mail addresses for an adult child and biological parent. Upon submission of the completed Study Information page, e-mail addresses were checked for accuracy and then recorded into an Excel file. The page also explained to interested participants that once enrolled into the study, parents would first be asked to complete surveys and once they completed their surveys, children would then be sent their corresponding survey. If an identical e-mail address was given for both a child and parent, a follow-up message was sent to the e-mail address explaining that a unique e-mail address would need to be provided for the second dyad member in order to be enrolled into the study. After capturing valid e-mail addresses for both parties, messages were sent to the parent's e-mail address containing a brief description of the study, a flyer with the study information, and the link to the parent survey. Once the parent completed the survey, their offspring was contacted and invited to participate by taking their survey. Each respondent in the dyad would thus have their own unique link to their survey. Further survey completion methods are described in detail below.

Survey completion procedure

Regardless of recruitment method, parents always completed their survey first before their children (i.e., children were never sent a link to the child survey without having a completed parent survey recorded first). Upon navigating to the UC Irvine Qualtrics survey link, parent respondents were first shown an introduction page that provided a brief description of the study goals, the approximate time needed to complete the survey, how their survey data would be stored, protected, and used in the future, and contact information to UC Irvine's Institutional Review Board (IRB) for any concerns or questions as a research participant. To move past the introduction page and begin the survey, respondents were required to click the "Agree" button to acknowledge that they had reviewed the introduction page; they were also then asked to verify that they were at least 18 years old by clicking a "Yes" or "No" option. Parents were then instructed to answer a series of questions and at the conclusion of the survey, they were asked to provide an e-mail address for their biological child who would also be participating in the study. Reminder messages were sent to parents who did not complete the survey within three days of receiving the initial invitation e-mail and a total of five subsequent reminder messages were sent until the parent completed the survey; messages were simultaneously sent to offspring encouraging them to remind their parents to complete the survey. Unresponsive participants were no longer contacted after the fifth reminder message. Using the "Workflow" Qualtrics function, messages containing the link to the child survey were automatically sent to offspring at the e-mail addresses provided by their parents within the survey and this automatically linked the completed parent surveys with their child's e-mail address. Finally, parents indicated an e-mail address to which they wished to have their survey compensation sent. All contact information provided was recorded into an Excel file and a unique dyad ID was assigned.

Through the "Workflow" Qualtrics function, messages were automatically sent to adult children at the e-mail addresses given by their parents. These messages contained a brief description of the study, a flyer with study as well as contact information, confirmation that their parent had completed their portion of the survey and that it was now requested for them to complete their survey, and a personalized link to the child survey that connected their survey responses to their parent's. After reviewing the survey introduction page and verifying that they were at least 18 years old, children were asked to complete the survey and provide the e-mail address they wished to have their survey compensation sent to at the end. Reminder messages were first sent to child respondents who did not complete the survey within three days of receiving the initial invitation e-mail and a total of five subsequent reminder messages were sent until respondent completed the survey; messages were simultaneously sent to parents encouraging them to remind their offspring to complete their survey. Unresponsive participants were no longer contacted after the fifth reminder message. Once both the parent and child surveys were complete, electronic Amazon gift cards in the amount of $15 were sent to each member of the dyad as compensation using their designated e-mail addresses.

Parent measures

Life event timing. Parents were first asked to select the month and year of their birth and the year they turned 18 years old using a dropdown menu to establish the time period of their childhood. Next, they were asked to indicate the birth month and birth year of their biological child who would also be participating in the study. To capture an approximate indication of their child's conception month, respondents were shown a chart containing all twelve months along with a corresponding month that was approximately ten months prior. Parents were instructed to locate the month that their child was born and use the chart to identify the month that was

roughly ten months before as conception takes place approximately 9 months before a child is born. For example, if their child was born in June, the chart showed that their child's corresponding conception month would be August and they would select August from the dropdown menu.

Using the provided information, three specific time periods were identified in the instructions for each of the different types of adversity on which respondents were asked to report. The childhood period referred to events or experiences that happened before turning 18 years old and for added clarity, the month and year in which they indicated they turned 18 years old at the beginning of the survey was displayed (i.e., "piped in"). The preconception period referred to events that parents experienced between age 18 and before their child was conceived. For increased clarity, the month and year in which they indicated they turned 18 years old and the approximate month and year their child was conceived were displayed (i.e., "piped in"). Finally, the post-conception period referred to events or experiences that occurred after their child was conceived until the present. The approximate month and year their child was conceived was once again displayed to provide a reminder of the specific time period being asked about.

General adversity. Using 29 items adapted from the Lifetime Stress Exposure Inventory (Blum, Silver, & Poulin, 2014; Seery, Holman, & Silver, 2010), parents were asked to indicate their exposure to general adversity. This measure was originally modified from the Diagnostic Interview Schedule trauma section (Robins, Helzer, Croughan, & Ratcliff, 1981) and was broadened to include a wider array of events using primary care patients' reports of lifetime stress (Blum et al., 2014; Holman, Silver, & Waitzkin, 2000; Seery et al., 2010). This measure has produced rates of specific events comparable to those in other community samples (Breslau

et al., 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Several categories of adversity, such as ACEs (e.g., childhood abuse, neglect), financial stress (e.g., lost job, no money for food or shelter), interpersonal loss/bereavement (e.g., suffered loss in a natural disaster, lost loved one to homicide or suicide), and violence exposure (e.g., intimate partner violence, lived in dangerous neighborhood), were included in this measure. Responses were summed and cumulative scores were generated for childhood (i.e., before age 18), preconception (i.e., between age 18 and before their child was conceived), post-conception (i.e., after child was conceived), and total lifetime general adversity (i.e., childhood, preconception, and post-conception combined).

Law enforcement adversity. Respondents were asked about their lifetime experiences with law enforcement using 8 items adapted from the Police Practices Inventory (PPI; DeVylder et al., 2017). Items included: "Has a police officer ever:" 1) hit, punched, kicked, dragged, beat, or otherwise used physical force against you?; 2) hit, punched, kicked, dragged, beat, or otherwise used physical force against a close friend or family member?; 3) used a gun, baton, taser, or other weapon against you?; 4) used a gun, baton, taser, or other weapon against a close friend or family member?; 5) forced inappropriate sexual contact on you, including while conducting a body search in a public place?; 6) forced inappropriate sexual contact on a close friend or family member, including while conducting a body search in a public place?; 7) engaged in non-physical aggression towards you, including threatening, intimidating, stopping you without probable cause, or using slurs?; and 8) engaged in non-physical aggression towards a close friend or family member, including threatening, intimidating, stopping him or her without probable cause, or using slurs? Respondents were asked to indicate whether each item had happened (yes/no) and if so, the specific time period(s) in their life (e.g., childhood,

preconception, post-conception) it happened; they were able to indicate if it happened in more than one time period. In addition to the PPI, respondents were asked if any of following had ever happened: a) been arrested, convicted, or incarcerated; b) had a close friend or family member arrested, convicted, or incarcerated; c) a close friend or family member was killed by law enforcement. Responses from the eight adapted PPI items and three additional law enforcement questions were summed and cumulative scores were generated for childhood (i.e., before age 18), preconception (i.e., between age 18 and before their child was conceived), and post-conception (i.e., after child was conceived), and total lifetime law enforcement experiences (i.e., childhood, preconception, and post-conception combined).

Racial discrimination. Parents were asked to provide information regarding their lifetime experiences with racial discrimination using a modified version of the Brief Perceived Ethnic Discrimination Questionnaire-Community Version (PEDQ-CV; Brondolo et al., 2005). The Brief PEDQ-CV, a 17-item measure, is designed to measure lifetime experiences of racial discrimination and maltreatment in interpersonal and social contexts and has a coefficient alpha of .87 in college as well as community samples (Brondolo et al., 2005). This questionnaire is comprised of four subscales, each containing four items, that include social exclusion, stigmatization, discrimination at work or school, and threats or actual acts of harassment and/or harm. Each item is prompted by the phrase: "Because of your ethnicity/race, how often…," followed by statements from each domain. An additional item asks about exposure to discrimination from police, but this item was omitted due to potential overlap with the assessment of law enforcement adversity mentioned above, resulting in a total of 16 items. Respondents reported the frequency with which they experienced unfair treatment in the four domains on a scale from 1 (never) to 5 (very often) during their childhood (before age 18) and

preconception (between age 18 and before their child was conceived). Responses across all items were summed to create a cumulative racial discrimination score for childhood, preconception, and lifetime, with higher scores reflecting more frequent experiences with racial discrimination. The scale had excellent reliability (Cronbach's α=0.93 for childhood, 0.96 for preconception).

Physician-diagnosed health ailments. Health data using questions adapted from the Centers for Disease Control's National Center for Health Statistics annual National Health Interview Survey (U.S. Department of Health and Human Services, National Center for Health Statistics, 2000) were used to collect physical health data. Using prior work, this index was comprised of multiple physical health ailments that disproportionately affect African Americans. Respondents were asked, "Has a medical doctor *ever* diagnosed you as suffering from any of the following ailments?" with prompts for 22 ailments. Ailments included: heart problems, hypertension, stroke, coronary heart disease, heart attack, high cholesterol level, diabetes mellitus, obesity, ulcers, liver disease, kidney disease, Hepatitis B, Hepatitis C, Human Immunodeficiency Virus (HIV)/Acquired immunodeficiency syndrome (AIDS), tuberculosis, arthritis, chronic back pain, asthma, pneumonia, cancer (any type), sleep problems, and an "other" option that allowed respondents to indicate additional ailments not previously listed. The total number of reported physical health ailments was calculated for each respondent.

Self-rated health (SRH). Parents were asked to evaluate their health using the single-item, self-rated health (SRH; Mossey & Shapiro, 1982) measure. This SRH measure has been strongly correlated with a range of health outcomes, including chronic illness, major depressive symptoms, physical health, and mortality across several populations (Ambresin, Chondros, Dowrick, Herrman, & Gunn, 2014; Fayers & Sprangers, 2002; Singh-Manoux et al., 2007). Respondents rated their current health status as excellent, very good, good, fair, or poor. A

continuous measure of SRH was used with codes from 1 (excellent) to 5 (poor), with higher values indicative of poorer SRH. Due to the small numbers of responses for both the fair and poor options, these responses were combined to form a fair/poor category creating a condensed self-rated health measure. Results did not change based on the use of the original or condensed measure.

Waist-to-height ratio (WHtR). Parents were asked to indicate their approximate height and waist circumference size. To facilitate an approximation of waist circumference size, respondents were shown a chart containing a list of typical U.S. pants sizes for both men and women along with the corresponding waist size in inches for each pants size. Respondents were then instructed to select the waist size in inches that best reflected their size using a slider. A waist-to-height ratio (WHtR) was calculated for each respondent by dividing their self-reported waist circumference by their height. WHtR, an index of abdominal obesity, has been identified as a useful indicator for cardiometabolic conditions, cardiovascular disease, and years of life lost irrespective of age and sex (Kazlauskaite et al., 2017) with higher scores signaling greater overall risk for obesity-related mortality.

Positive affect. Respondents were asked to indicate their current level of positive affect using a modified version of the Positive and Negative Affect Schedule (PANAS-SF; Watson, Clark, & Tellegen, 1998). This measure was included to evoke positive emotions and counteract potential discomfort from answering questions regarding adverse life experiences. Ten items from the positive affect subscale of the PANAS-SF were included: interested, excited, strong, enthusiastic, proud, alert, inspired, determined, attentive and active. Respondents reported the extent to which they felt each of the ten emotions or feelings in the present moment on a scale from 1 (not at all) to 5 (very much) and cumulative scores were calculated with higher scores

representing higher levels of positive affect. The scale had excellent reliability (Cronbach's α=0.90).

Open-ended questions. Three open-ended questions were included in the survey aimed at reducing any potential discomfort associated with answering questions about life adversity by encouraging respondents to reflect on positive experiences and thoughts. The prompts included: 1) "Please describe something you felt good about or proud of doing in the last month. Write as little or as much as you want."; 2) "Please describe your favorite attribute or quality about yourself. Write as little or as much as you want."; 3) "Please share something that has brought you happiness or joy in spite of the COVID-19 pandemic. Write as little or as much as you want."

Demographics. Lastly, parents were asked to indicate several pieces of demographic information: relationship to child (biological mother or father); gender of child (daughter or son); race (African American/Black, African, African Caribbean/Afro-Caribbean, Multi-racial, other); approximate yearly household income during year child was born (Under $24,999; $25k-$49,999; $50k-$74,999; $75k-$99,999; $100k+); level of education at time child was born (less than high school diploma, high school diploma, some college, BA degree or higher); and whether they themselves were born in the U.S. (yes/no).

Child measures

General adversity. Using the same 29 items adapted from the Lifetime Stress Exposure Inventory (Blum et al., 2014; Seery et al., 2010) described previously, children were asked to indicate their exposure to general adversity. Responses were summed and cumulative scores were generated for childhood (before age 18), adulthood (age 18 & older), and general lifetime adversity (childhood and adulthood combined).

Law enforcement adversity. Children were asked about their experiences with law enforcement using the same eight adapted items from the PPI (DeVylder et al., 2017) and three additional law enforcement items described above. Respondents were asked to indicate whether each item had happened (yes/no) and if so, the specific time period(s) in their life (e.g., before age 18 only, age 18 & older only, both before and after age 18). Responses from the eight adapted PPI items and three additional law enforcement questions were summed and cumulative scores were generated for childhood (before age 18), adulthood (age 18 & older), and lifetime law enforcement adversity (childhood and adulthood combined).

Racial discrimination. Children were asked to report on their lifetime experiences with discrimination using similar methods described above with the PEDQ-CV. Respondents indicated the frequency of discrimination experiences on a scale from 1 (never) to 5 (very often). The item asking about exposure to discrimination from police was once again omitted due to potential overlap with the law enforcement adversity measure leaving a total of 16 PEDQ-CV items (Cronbach's α=0.90). A cumulative score of the responses to the 16 items was calculated to represent lifetime racial discrimination with higher scores indicating greater exposure.

Physician-diagnosed health ailments. Health data using questions adapted and modified from the American College Health Association's (ACHA) National College Health Assessment (NCHA) were gathered. The NCHA is a national survey that examines behaviors, attitudes, and health among U.S. college students. The NCHA has been administered twice annually since 2000 and has been established as reliable and valid among U.S. college students by the ACHA (American College Health Association, 2013). Respondents were asked, "Have you *ever* been diagnosed or treated by a healthcare professional for any of the following?" with prompts for 23 ailments. The ailments included: allergies; arthritis; asthma; cancer (any type); chronic pain;

coronary heart disease; diabetes; heart problems; Hepatitis B or C; hypertension; high cholesterol level; HIV)/AIDS; insomnia; kidney disease; liver disease; migraine headaches; obesity; pneumonia; stroke; and an "other" option that allowed respondents to indicate additional ailments not previously listed. The cumulative number of reported physical health ailments was calculated for each respondent.

Self-rated health (SRH). Children were asked to report their SRH status using the same single-item measure (Mossey & Shapiro, 1982) described above. A continuous measure of SRH was used with codes from 1 (excellent) to 5 (poor), with higher values indicative of poorer SRH. Due to the small numbers of responses for both the fair and poor options, these responses were combined to form a fair/poor category creating a condensed self-rated health measure. Results did not change based on the use of the original or condensed measure.

Waist-to-height ratio (WHtR). A WHtR was calculated through the same methods described above using the self-reported height and waist circumference provided by children with higher scores signaling greater overall risk for obesity-related mortality.

Positive affect. Children indicated their current level of positive affect using the same ten items from the PANAS-SF (Watson et al., 1998) to elicit positive emotions. Respondents were asked to indicate the extent to which they felt each of the ten emotions or feelings in the present moment on a scale from 1 (not at all) to 5 (very much) and cumulative scores were calculated, with higher values representing higher levels of positive affect. The scale had good reliability (Cronbach's α=0.90).

Open-ended questions. Children were also given the same three open-ended questions included in the parent survey described previously to minimize potential distress by prompting respondents to think about positive experiences and thoughts.

Demographics. Finally, students reported several demographic indicators including their age (in years), gender (male, female, transgender, non-binary/non-conforming), race (African American/Black, African, African Caribbean/Afro-Caribbean, Multi-racial, other), and whether they were born in the United States (yes/no).

Analytic strategy

Single generation analyses

Parents. Statistical analyses were conducted in Stata 14.2 (College Station, TX). Although the sample included some dyads with fathers (n = 4 dyads), there were not enough to properly control for potential gender differences in parents and as a result, fathers were dropped from the final analytic sample. All subsequent information and analyses pertains to mothers exclusively. Descriptive statistics for respondents' characteristics were summarized by use of means, standard deviations, and ranges for continuous data and proportions for categorical data and are reported in Table 3.1. All variable distributions were scrutinized to check for discrepancies, outliers, and to ensure that all variables were analyzed appropriately. There was a single outlier case in which a maternal respondent indicated that all general, law enforcement, and racial discrimination experiences happened to her in all time periods that was removed from the analysis, but results did not change with the exclusion of the case. To explore associations between maternal adversity exposure and their own health outcomes, several analyses were conducted with the use of generalized linear models (GLMs). GLMs are used as an alternative approach to ordinary least squares regression where data deviate from a normal distribution and allow for the specification of a nonnormal error distribution and a function that links the predictor to the outcome (Coxe, West, & Aiken, 2013; Myers & Montgomery, 1997). A process of exploring the most appropriate selection of family and link options for each outcome variable

was conducted and the configurations producing the best overall model fit were used. Overall model fit was evaluated with Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) values, log likelihood values, and the size of standard errors. AIC is an indicator of the quality or fit of a statistical model because it estimates the level of prediction error within a given model; similarly, BIC is an alternative marker of model fit and measures the trade-off between the fit of the model and the complexity of the model (Coxe et al., 2013). In both cases, smaller AIC and BIC values represent better model fit. Another helpful value that evaluates a model is the log-likelihood with higher values representing a better-fitting model of the data. Adversity exposures were categorized by timing (e.g., childhood before age 18, age 18 to before conception, post-conception) and type (e.g., general, law enforcement, racial discrimination).

For analyses involving the number of physician-diagnosed ailments as the outcome, GLMs with a log link identity and a Poisson family were used as Poisson distributions are specifically designed to examine count variable data that deviate from a normal distribution (Coxe, West, & Aiken, 2009). The first set of analyses explored links between maternal lifetime experiences of each adversity type and the number of physician-diagnosed health ailments in three separate models. Next, a hierarchical approach was used to analyze associations between the timing of each of the maternal adversity types and health outcomes with each subsequent model including adversity experienced in another time period to detect potential differences in their impact. For general and law enforcement adversity types, Model 1 started with childhood experiences; Model 2 added adulthood preconception experiences and Model 3 included post-conception experiences. For racial discrimination, only two models were run, with the first including childhood experiences and the second including adulthood preconception experiences. Due to relatively small sample size, only maternal age and income were included as covariates in

each model as these were the most theoretically relevant demographic indicators and no other demographics showed no significant associations with outcomes in bivariate analyses.

Identical approaches were taken for exploring associations between maternal adversity and self-rated health and waist-to-height ratio except for the models' link and family designations. When exploring self-rated health as the outcome, the identity link and gamma family distribution was used. Identity links and gamma distributions are generally used for variables that are continuous, non-negative, and positively skewed (Coxe, et al., 2013). Examining the distribution of the continuous self-rated health variable demonstrated that all of these conditions were met, as values ranged from one to four and very few values that were over three signaling the positively skewed nature of these data. For waist-to-height ratio, the identity link and Gaussian family distribution was used. Identity links and gaussian distributions are typically characterized as continuous, normally-distributed data (Zorn, 2001) and inspecting the distribution of waist-to-height ratio revealed an approximate normal distribution making it the most appropriate choice. Analyses included maternal age and income as covariates.

Table 3.1. Descriptive statistics for all maternal variables of interest (n = 57)

Variables	*M*	*SD*	Min	Max
Physician-diagnosed health ailments[a]	2.35	3.04	0	21
Waist-to-height ratio (WHtR)[a]	.55	.10	.41	.85
Self-rated health (SRH)[a]	2.63	.96	1	5
General adversity[a]				
Childhood	5.89	5.72	0	25
Preconception	3.98	5.23	0	24
Post-conception	4.07	4.53	0	23
Lifetime	13.95	13.18	0	71
Law enforcement adversity[a]				
Childhood	1.02	2.15	0	11
Preconception	1.25	2.12	0	11
Post-conception	1.25	2.00	0	11
Lifetime	3.53	5.73	0	33
Racial discrimination[a]				
Childhood	10.93	11.08	0	44
Preconception	9.86	12.38	0	53
Lifetime	20.82	21.21	0	93
Covariates and sample characteristics				
Maternal age	51.11	9.79	37	82
	%			
Education during year child was born[a]				
Less than high school diploma	9			
High school diploma	10			
Some college	39			
Bachelor's degree or higher	42			
Annual income during year child was born[a]				
Less than $24,9999	43			
$25,000 to $49,999	20			
$50,000 to $74,999	20			
$75,000 or more	17			
Race				
African-American/Black	77			
African	5			
African-Caribbean/Afro-Caribbean	2			
Multi-racial	7			
Other	9			

[a]Sample size varies due to missing data

Children. Descriptive statistics for offspring characteristics were summarized using means, standard deviations, and ranges for continuous data and proportions for categorical data and are reported in Table 3.2. To explore associations between offspring adversity exposure and their own health outcomes, several analyses were conducted with the use of generalized linear models (GLMs). Adversity exposures were categorized by timing (e.g., childhood before age 18, adulthood, lifetime) and type (e.g., general, law enforcement, racial discrimination). As was done with the analyses for the parent sample, the most appropriate options for family and link selections were identified and used for each outcome variable. For analyses involving the number of physician-diagnosed ailments as the outcome, GLMs with a log link identity and a Poisson family were used. The first set of analyses explored links between offspring lifetime experiences of each adversity type and the number of physician-diagnosed health ailments in three separate models. Next, a hierarchical approach was used to analyze associations between the timing of each of the offspring general and law enforcement adversity types and health outcomes with each added model including another time period to detect potential differences in their impact. For general and law enforcement adversity types, Model 1 started with childhood experiences while Model 2 added adulthood experiences. A hierarchical approach was not used for offspring racial discrimination adversity because it was only captured at the lifetime level. Due to relatively small sample size, offspring age and gender were included as covariates in each model.

Identical approaches were taken for exploring associations between offspring adversity and self-rated health and waist-to-height ratio except for link and family designations. When exploring self-rated health as the outcome, the identity link and gamma family distribution was used for the same reasons described above. For waist-to-height ratio, the identity link and

Gaussian family distribution was used for the same reasons mentioned above. Analyses included only offspring age and gender as covariates due to small sample size.

Table 3.2. Descriptive statistics for all offspring variables of interest (n = 57)

Variables	***M***	***SD***	**Min**	**Max**
Physician-diagnosed health ailments	1.72	1.88	0	8
Waist-to-height ratio (WHtR)[a]	.48	.10	.29	.94
Self-rated health (SRH)	2.44	.93	1	5
General adversity				
Childhood	5.61	4.19	0	18
Adulthood	3.19	3.39	0	15
Lifetime	8.80	6.58	0	30
Law enforcement adversity				
Childhood	1.00	1.16	0	5
Adulthood	.84	1.31	0	5
Lifetime	1.84	2.09	0	9
Lifetime racial discrimination	14.42	9.60	0	36
Covariates and sample characteristics				
Child age	23.81	8.42	18	56
	%			
Gender				
Female	82			
Male	18			
Race				
African-American/Black	84			
African	4			
African-Caribbean/Afro-Caribbean	2			
Multi-racial	2			
Other	8			
School				
Kentucky State University	32			
Jackson State University	23			
University of California, Irvine	23			
North Carolina Central University	11			
Morehouse College	7			
Florida A & M University	3			
University of California, Davis	1			

[a]Sample size varies due to missing data

Intergenerational Analyses.

For analyses exploring intergenerational associations, a pairwise data set was created that included two rows of observations for each dyad with one containing the parent's data and the other containing the child's data. Dyads were identified by a common ID value and distinctions between parents and children within each dyad were made using a person variable. Generalized estimating equations (GEEs) were used to investigate links between parental adversity and offspring health outcomes. The GEE method is commonly used for modeling longitudinal and other correlated response data that deviate from a normal distribution (Hanley et al., 2003). GEEs provide ample flexibility for handling different covariance and correlation structures and this was an ideal approach for conducting dyadic analyses with several outcome variables that have disparate correlation structures and unique, non-normal distributions.

Parent-child dyads were first designated by the ID grouping variable and then distinguished by the person variable that signaled either parent or child. A process of exploring the most appropriate selection of family and link options for each outcome variable was conducted and the configurations producing the best overall model fit were used. Overall model fit was evaluated with Wald Chi-Squared Test statistics, the range between confidence intervals of predictor variables, and size of standard errors. When exploring the number of physician-diagnosed health ailments in offspring as an outcome, the log link function was used along with a Poisson family distribution and an unstructured correlation structure as Poisson distributions are specifically designed to examine count variable data that deviate from a normal distribution (Coxe, West, & Aiken, 2009). Often used as an approach to repeated measures or other clustered data, an unstructured correlation structure was selected because it does not impose any constraints on the variance or covariance values between predictors and outcomes; instead, each

of these values is estimated uniquely from the data providing the most unbiased parameter estimates (Zorn, 2001). This represents a more ideal approach than identifying specific constraint values as there may be great variability in the associations between maternal adversity and offspring health across dyads. When exploring offspring self-rated health as the outcome, the identity link function was used with a gamma distribution and an unstructured correlation structure. Identity links and gamma distributions are commonly used for variables that are continuous, non-negative, and positively skewed (Coxe, et al., 2013) and the self-rated health outcome variable satisfied these conditions. Lastly, analyses with offspring waist-to-height ratio as the outcome used the identity link function, a Gaussian family distribution, and an unstructured correlation structure. Identity links and gaussian distributions are used for continuous, normally-distributed data (Zorn, 2001) and the distribution of waist-to-height ratio closely resembled a normal distribution.

The first set of analyses explored links between the three parental lifetime adversity types (general, law enforcement, racial discrimination) and each of the offspring health outcomes in separate models. Offspring age, cumulative adversity, and gender were included as covariates. Next, a hierarchical approach was used to analyze associations between the timing of each of the parental adversity types and offspring outcomes with each subsequent model including an additional time period to detect potential differences in their impact on offspring health. For general and law enforcement adversity types, Model 1 started with childhood experiences; Model 2 added adulthood preconception experiences and Model 3 included post-conception experiences. For racial discrimination, only two models were run with the first including childhood experiences and the second including adulthood preconception. All models included offspring age, cumulative adversity, and gender as covariates. Models were also conducted

without offspring age and gender, but only models including offspring covariates are shown due to superior model fit. There was a single outlier case in which a maternal respondent indicated that all of the general, law enforcement, and racial discrimination experiences happened to them in all time periods that was removed from the analysis, but results did not change with the exclusion of the case.

Results

Results for single generation analyses in mothers indicated that there were no significant associations between any of the lifetime maternal adversity types and maternal health outcomes after controlling for maternal age and income (see Tables 3.3, 3.4, and 3.5). However, breaking out various adversity types by timing uncovered several significant associations. First, maternal general adversity in the post-conception period was associated with reporting a greater number of physician-diagnosed health ailments after controlling for childhood and preconception general adversity and covariates (incident rate ratio, 1.04; 95% confidence interval: 1.00-1.10; see Table 3.6). In addition, maternal law enforcement adversity experienced post-conception was negatively associated with the number of physician-diagnosed health ailments (IRR, 0.81; 95% CI: 0.68-0.97), while controlling for childhood and preconception law enforcement adversity and covariates (see Table 3.7). In addition, waist-to-height ratio showed significant associations with general adversity such that childhood experiences were linked to a lower ratio (i.e., better health; unstandardized b = -.00, SE = .00, z = -1.99, p = .046), but preconception associations were linked to a higher ratio (i.e., poorer health; unstandardized b = .01, SE = .00, z = 3.09, p = .002); post-conception was not associated with the outcome (see Table 3.8). No other significant links between adversity types and their timing were observed (see Appendices L-Q).

For single generation analyses in offspring, results indicated that general lifetime adversity was significantly associated with both a higher number of physician-diagnosed health ailments (IRR, 1.04; 95% CI: 1.01-1.08); see Table 3.9) and poorer self-rated health (unstandardized b = .05, SE = .02, z = 2.54, p = .011; see Table 3.10), after controlling for several covariates while lifetime law enforcement adversity and racial discrimination were not. None of the lifetime offspring adversity types were significantly associated with waist-to-height ratio (see Table 3.11). When further broken down by timing, general adversity in childhood was a significant predictor of a greater number of physician-diagnosed health ailments (IRR, 1.07; 95% CI: 1.01-1.14; see Table 3.12) and poorer self-rated health (unstandardized b = .10, SE = .03, z = 2.96, p = .003; see Table 3.13), while adulthood general adversity was not. None of the other adversity types showed significant associations to offspring waist-to-height ratio based on their timing (see Appendix R).

Table 3.3. Generalized linear models examining associations between maternal lifetime adversity and number of physician-diagnosed ailments (n = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Age[a]	**0.97(0.95, 0.99)**	**.016**	**0.97(0.95, 0.99)**	**.027**	**0.97(0.95, 0.99)**	**.039**
Income during year child was born[b]						
$25k-$49,999	**0.38(0.20, 0.70)**	**.002**	**0.36(0.19, 0.67)**	**.001**	**0.33(0.16, 0.64)**	**.001**
$50k-$74,999	0.78(0.48, 1.28)	.337	0.74(0.45, 1.23)	.260	0.72(0.43, 1.19)	.207
$75k or more	0.71(0.42, 1.17)	.186	0.65(0.35, 1.20)	.173	0.69(0.38, 1.26)	.236
Adversity experiences						
General	0.99(0.98, 1.01)	.751	—	—	—	—
Law enforcement	—	—	0.95(0.90, 1.00)	.075	—	—
Racial discrimination	—	—	—	—	1.00(0.99, 1.01)	.322
Constant	10.62(3.76, 29.93)	<.001	12.06(4.24, 34.30)	<.001	9.34(3.04, 28.61)	<.001
Model Statistics (AIC, BIC)	(4.64, -70.22)		(4.75, -48.13)		(4.83, -42.40)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.4. Generalized linear models examining associations between maternal lifetime adversity and self-rated health (*n* = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	0.00(-0.01, 0.02)	.759	0.00(-0.02, 0.02)	.754	0.00(-0.02, 0.03)	.718
Income during year child was born[b]						
$25k-$49,999	-0.51(-1.14, 0.10)	.106	-0.61(-1.27, 0.05)	.070	-0.61(-1.25, 0.02)	.061
$50k-$74,999	-0.54(-1.15, 0.06)	.078	-0.61(-1.27, 0.05)	.071	**-0.72(-1.38, -0.05)**	**.034**
$75k or more	**-0.66(-1.27, -0.05)**	**.034**	**-0.98(-1.67, -0.30)**	**.005**	**-0.73(-1.44, -0.03)**	**.041**
Adversity experiences						
General	0.00(-0.01, 0.02)	.508	—	—	—	—
Law enforcement	—	—	-0.00(-0.04, 0.03)	.799	—	—
Racial discrimination	—	—	—	—	0.00(-0.00, 0.02)	.348
Constant	2.61(1.33, 3.89)	<.001	2.78(1.46, 4.10)	<.001	2.60(1.24, 3.95)	<.001
Model Statistics (AIC, BIC)	(4.09, -200.07)		(4.10, -171.19)		(4.13, -166.87)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.5. Generalized linear models examining associations between maternal lifetime adversity and waist to height ratio (*n* = 55)

	General Adversity		Law Enforcement		Racial Discrimination	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	0.00(-0.00, 0.00)	.944	0.00(-0.00, 0.00)	.966	0.00(-0.00, 0.00)	.981
Income during year child was born[b]						
$25k-$49,999	-0.01(-0.08, 0.06)	.721	-0.01(-0.09, 0.05)	.618	-0.02(-0.10, 0.05)	.542
$50k-$74,999	0.00(-0.06, 0.07)	.884	-0.00(-0.08, 0.07)	.921	-0.00(-0.07, 0.07)	.996
$75k or more	-0.02(-0.10, 0.04)	.461	-0.04(-0.13, 0.04)	.323	-0.04(-0.13, 0.04)	.365
Adversity experiences						
General	0.00(-0.00, 0.00)	.842	__	__	__	__
Law enforcement	__	__	-0.00(-0.00, 0.00)	.518	__	__
Racial discrimination	__	__	__	__	-0.00(-0.00, 0.00)	.570
Constant	0.54(0.38, 0.69)	<.001	0.56(0.39, 0.72)	<.001	0.56(0.39, 0.73)	<.001
Model Statistics (AIC, BIC)	(-1.64, -195.86)		(-1.58, -166.88)		(-1.58, 166.88)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.6. Hierarchical generalized linear model examining associations between maternal general adversity by timing and number of physician-diagnosed ailments (*n* = 56)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Age[a]	**0.97(0.95, 0.99)**	**.010**	**0.97(0.95, 0.99)**	**.010**	**0.97(0.95, 0.99)**	**.006**
Income during year child was born[b]						
$25k-$49,999	**0.38(0.20, 0.71)**	**.002**	**0.38(0.20, 0.71)**	**.002**	**0.40(0.21, 0.75)**	**.004**
$50k-$74,999	0.78(0.47, 1.27)	.319	0.77(0.47, 1.27)	.318	0.82(0.50, 1.34)	.431
$75k or more	0.69(0.42, 1.15)	.161	0.69(0.42, 1.15)	.159	0.76(0.45, 1.27)	.301
Adversity experiences						
Childhood	0.98(0.95, 1.01)	.304	0.98(0.93, 1.02)	.400	0.97(0.92, 1.01)	.236
Preconception	—	—	1.00(0.95, 1.05)	.898	0.98(0.93, 1.04)	.694
Post-conception	—	—	—	—	**1.04(1.00, 1.10)**	**.048**
Constant	12.39(4.38, 35.01)	<.001	12.47(4.39, 35.43)	<.001	11.20(4.10, 30.54)	<.001
Model Statistics (AIC, BIC)	(4.62, -71.22)		(4.66, -67.21)		(4.63, -66.92)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.7. Hierarchical generalized linear model examining associations between maternal law enforcement adversity by timing and number of physician-diagnosed ailments (n = 51)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Age[a]	**0.97(0.95, 0.99)**	**.030**	**0.97(0.95, 0.99)**	**.030**	**0.97(0.95, 0.99)**	**.021**
Income during year child was born[b]						
$25k-$49,999	**0.37(0.20, 0.70)**	**.002**	**0.38(0.20, 0.71)**	**.003**	**0.35(0.18, 0.67)**	**.001**
$50k-$74,999	0.70(0.42, 1.16)	.169	0.69(0.41, 1.17)	.174	0.80(0.46, 1.39)	.434
$75k or more	0.63(0.34, 1.16)	.143	0.63(0.34, 1.16)	.142	0.76(0.40, 1.45)	.418
Adversity experiences						
Childhood	0.91(0.81, 1.03)	.170	0.90(0.76, 1.08)	.286	1.03(0.84, 1.26)	.743
Preconception	—	—	1.01(0.86, 1.18)	.879	1.00(0.85, 1.17)	.967
After conception	—	—	—	—	**0.81(0.68, 0.97)**	**.023**
Constant	11.02(3.92, 30.97)	<.001	10.99(3.91, 30.88)	<.001	13.49(4.58, 39.70)	<.001
Model Statistics (AIC, BIC)	(4.79, -46.38)		(4.83, -42.49)		(4.75, -44.34)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.8. Hierarchical generalized linear model examining associations between maternal general adversity by timing and waist to height ratio (n = 55)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	-0.00(-0.00, 0.00)	.967	-0.00(-0.00, 0.00)	.779	-0.00(-0.00, 0.00)	.899
Income during year child was born[b]						
$25k-$49,999	-0.01(-0.08, 0.06)	.742	-0.00(-0.07, 0.96)	.791	-0.01(-0.08, 0.05)	.698
$50k-$74,999	0.00(-0.06, 0.07)	.921	0.00(-0.06, 0.07)	.946	-0.00(-0.07, 0.06)	.959
$75k or more	-0.03(-0.10, 0.04)	.422	-0.03(-0.10, 0.03)	.323	-0.04(-0.11, 0.02)	.218
Adversity experiences						
Childhood	-0.00(-0.00, 0.00)	.667	**-0.00(-0.01, -0.00)**	**.027**	**-0.00(-0.01, -0.00)**	**.046**
Preconception	—	—	**0.00(0.00, 0.01)**	**.007**	**0.01(0.00, 0.01)**	**.002**
After conception	—	—	—	—	-0.00(-0.01, 0.00)	.128
Constant	0.56(0.40, 0.71)	<.001	0.57(0.42, 0.72)	<.001	0.57(0.43, 0.72)	<.001
Model Statistics (AIC, BIC)	(-1.65, -195.86)		(-1.75, -191.92)		(-1.76, -187.93)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Table 3.9. Generalized linear models examining associations between offspring lifetime adversity and number of physician-diagnosed ailments (n = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Female gender[a]	1.72(0.91, 3.24)	.090	1.73(0.86, 3.46)	.119	1.68(0.89, 3.15)	.104
Age[b]	1.01(0.99, 1.03)	.105	**1.03(1.01, 1.05)**	**.001**	**1.03(1.02, 1.05)**	**< .001**
Adversity experiences						
General	**1.04(1.01, 1.08)**	**.002**	—	—	—	—
Law enforcement	—	—	1.02(0.91, 1.14)	.717	—	—
Racial discrimination	—	—	—	—	1.01(0.99, 1.03)	.194
Constant	0.42(0.20, 0.87)	.021	0.42(0.19, 0.93)	.033	0.33(0.14, 0.80)	.014
Model Statistics (AIC, BIC)	(3.51, -125.53)		(3.67, -116.43)		(3.64, -117.97)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.10. Generalized linear models examining associations between offspring lifetime adversity and self-rated health (*n* = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**ß(95% CI)**	***p***	**ß(95% CI)**	***p***	**ß(95% CI)**	***p***
Demographics						
Female gender[a]	**0.83(0.27, 1.40)**	**.004**	**0.79(0.14, 1.43)**	**.016**	**0.71(0.13, 1.29)**	**.016**
Age[b]	-0.01(-0.03, 0.01)	.454	0.00(-0.02, 0.02)	.947	0.00(-0.02, 0.02)	.835
Adversity experiences						
General	**0.04(0.00, 0.07)**	**.020**	—	—	—	—
Law enforcement	—	—	0.01(-0.10, 0.14)	.781	—	—
Racial discrimination	—	—	—	—	-0.01(-0.03, 0.00)	.181
Constant	1.61(0.81, 2.40)	<.001	1.71(0.86, 2.56)	<.001	1.99(1.10, 2.89)	<.001
Model Statistics (AIC, BIC)	(2.50, -178.71)		(2.60, -175.14)		(2.57, -176.37)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.11. Generalized linear models examining associations between offspring lifetime adversity and waist to height ratio (n = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	0.04(-0.01, 0.11)	.169	0.03(-0.03, 0.10)	.332	0.04(-0.01, 0.11)	.140
Age[b]	**0.00(0.00, 0.00)**	**.049**	**0.00(0.00, 0.00)**	**.009**	**0.00(0.00, 0.00)**	**.007**
Adversity experiences						
General	0.00(-0.00, 0.00)	.257	—	—	—	—
Law enforcement	—	—	-0.00(-0.01, 0.01)	.703	—	—
Racial discrimination	—	—	—	—	0.00(-0.00, 0.00)	.053
Constant	0.34(0.25, 0.43)	<.001	0.35(0.25, 0.45)	<.001	0.31(0.21, 0.41)	<.001
Model Statistics (AIC, BIC)	(-1.79, -208.84)		(-1.77, -208.83)		(-1.84, -208.86)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.12. Hierarchical generalized linear models examining associations between offspring adversity by timing and number of physician-diagnosed ailments (n = 57)

	General Adversity				Law Enforcement			
	Model 1		Model 2		Model 1		Model 2	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics								
Age[a]	**1.02(1.01, 1.04)**	**.001**	1.03(0.99, 1.06)	.070	**1.03(1.01, 1.05)**	**<.001**	**1.03(1.00, 1.05)**	**.006**
Female gender[b]	1.82(0.96, 3.43)	.065	1.83(0.96, 3.48)	.065	1.66(0.80, 3.44)	.166	1.65(0.80, 3.41)	.171
Adversity experiences								
General childhood	**1.07(1.02, 1.12)**	**.001**	**1.07(1.01, 1.14)**	**.013**	—	—	—	—
General adulthood	—	—	0.99(0.88, 1.10)	.882	—	—	—	—
Law enforcement childhood	—	—	—	—	1.00(0.81, 1.25)	.939	0.97(0.75, 1.25)	.849
Law enforcement adulthood	—	—	—	—	—	—	1.05(0.86, 1.29)	.589
Constant	0.31(0.14, 0.66)	.003	0.29(0.11, 0.80)	.017	0.43(0.17, 1.06)	.069	0.47(0.18, 1.22)	.124
Model Statistics (AIC, BIC)	(3.49, -126.60)		(3.52, -122.58)		(3.67, -116.31)		(3.70, -112.55)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.13. Hierarchical generalized linear models examining associations between offspring adversity by timing and self-rated health (n = 57)

	General Adversity				Law Enforcement			
	Model 1		Model 2		Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics								
Age[a]	0.00(-0.02, 0.02)	.962	0.02(-0.01, 0.06)	.321	0.00(-0.02, 0.02)	.871	0.00(-0.03, 0.03)	.931
Female gender[b]	**0.90(0.35, 1.46)**	**.001**	**0.97(0.40, 1.53)**	**.001**	**0.80(0.11, 1.48)**	**.022**	**0.79(0.10, 1.49)**	**.024**
Adversity experiences								
General childhood	**0.07(0.02, 0.12)**	**.004**	**0.10(0.03, 0.17)**	**.003**	—	—	—	—
General adulthood	—	—	-0.07(-0.18, 0.04)	.222	—	—	—	—
Law enforcement childhood	—	—	—	—	0.03(-0.19, 0.25)	.789	0.02(-0.22, 0.28)	.845
Law enforcement adulthood	—	—	—	—	—	—	0.01(-0.21, 0.23)	.922
Constant	1.24(0.39, 2.08)	.004	0.78(-0.32, 1.89)	.167	1.67(0.71, 2.63)	.001		
Model Statistics (AIC, BIC)	(2.45, -180.37)		(2.46, -177.27)		(2.60, -175.14)		(2.63, -171.10)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

For intergenerational analyses, results demonstrated that general maternal lifetime adversity was significantly associated with a higher number of physician-diagnosed health ailments in offspring, after controlling for offspring adversity (IRR, 1.01; 95% CI: 1.00-1.03); see Table 3.14). Maternal lifetime general adversity was not associated with any of the other offspring health outcomes and neither lifetime law enforcement nor racial discrimination was linked to any offspring health outcomes (see Tables 3.15 and 3.16). When further broken by timing, maternal preconception general adversity was significantly associated with a greater number of physician-diagnosed offspring health ailments (IRR, 1.05; 95% CI: 1.00-1.11), while childhood and post-conception experiences were not (see Table 3.17). Neither maternal law enforcement adversity nor racial discrimination broken down by timing were related to offspring health ailments (see Appendices S and T).

For offspring self-rated health, greater maternal post-conception law enforcement adversity was significantly associated with better offspring self-rated health (unstandardized b = -.23, SE = .07, z = -3.10, p = .002; see Table 3.18); no other associations were identified for general adversity or racial discrimination by timing (see Appendices U and V). For waist-to-height ratio, maternal childhood general adversity was associated with a smaller waist-to-height ratio (i.e., better health; unstandardized b = -.00, SE = .00, z = -3.00, p = .003), while preconception general adversity was linked to a larger waist-to-height ratio (i.e., poorer health; unstandardized b = .00, SE = .00, z = 2.73, p = .006) and post-conception general adversity was not associated (see Table 3.19). However, it is important to note both of these significant effects were considerably small. Neither maternal law enforcement adversity nor racial discrimination broken down by timing were related to offspring health outcomes (see Appendices W and X).

Table 3.14. Generalized estimating equations examining associations between maternal adversity and number of offspring physician-diagnosed ailments (n = 57)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Female gender[a]	1.83(0.96, 3.48)	.064	1.75(0.92, 3.33)	.087	1.73(0.91, 3.30)	.093
Age[b]	**1.03(1.01, 1.05)**	**<.001**	1.02(0.99, 1.04)	.060	**1.02(1.00, 1.05)**	**.025**
Offspring adversity	1.01(0.99, 1.02)	.054	**1.02(1.00, 1.03)**	**.013**	**1.01(1.00, 1.03)**	**.048**
Adversity experiences						
General	**1.01(1.00, 1.03)**	**.023**	—	—	—	—
Law enforcement	—	—	0.99(0.95, 1.04)	.848	—	—
Racial discrimination	—	—	—	—	1.00(0.99, 1.01)	.433
Constant	0.22(0.09, 0.54)	.001	0.34(0.13, 0.84)	.020	0.28(0.11, 0.71)	.008
Model Statistics	Wald's χ^2 (4, 57) = 28.89, p < .001		Wald's χ^2 (4, 51) = 15.30, p = .004		Wald's χ^2 (4, 50) = 16.96, p = .002	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.15. Generalized estimating equations examining associations between maternal adversity and offspring self-rated health (*n* = 51)

	General Adversity		**Law Enforcement**		**Racial Discrimination**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	**0.86(0.41, 1.30)**	**<.001**	**0.87(0.39, 1.35)**	**<.001**	**0.80(0.32, 1.28)**	**.001**
Age[b]	0.00(-0.02, 0.02)	.869	0.00(-0.02, 0.03)	.679	0.00(-0.02, 0.04)	.653
Offspring adversity	0.00(-0.00, 0.02)	.410	0.00(-0.00, 0.02)	.343	0.00(-0.00, 0.02)	.369
Adversity experiences						
General	0.00(-0.01, 0.01)	.901	—	—	—	—
Law enforcement	—	—	-0.00(-0.04, 0.03)	.708	—	—
Racial discrimination	—	—	—	—	-0.00(-0.01, 0.00)	.189
Constant	1.48(0.69, 2.26)	<.001	1.39(0.53, 2.25)	.002	1.54(0.56, 2.51)	.002
Model Statistics	Wald's χ^2 (4, 57) = 14.60, p = .005		Wald's χ^2 (4, 51) = 13.04, p = .011		Wald's χ^2 (4, 50) = 13.97, p = .007	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.16. Generalized estimating equations examining associations between maternal adversity and offspring waist-to-height ratio (n = 56)

	General Adversity		Law Enforcement		Racial Discrimination	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	0.05(-0.00, 0.11)	.099	0.03(-0.01, 0.09)	.170	0.03(-0.01, 0.08)	.203
Age[b]	**0.00(0.00, 0.00)**	**.035**	0.00(-0.00, 0.00)	.090	0.00(-0.00, 0.00)	.138
Offspring adversity	0.00(-0.00, 0.00)	.055	0.00(-0.00, 0.00)	.202	0.00(-0.00, 0.00)	.212
Adversity experiences						
General	-0.00(-0.00, 0.00)	.520	—	—	—	—
Law enforcement	—	—	-0.00(-0.00, 0.00)	.401	—	—
Racial discrimination	—	—	—	—	-0.00(-0.00, 0.00)	.538
Constant	0.32(0.23, 0.42)	<.001	0.36(0.28, 0.45)	<.001	0.37(0.27, 0.46)	<.001
Model Statistics	Wald's χ^2 (4, 56) = 13.58, p = .008		Wald's χ^2 (3, 51) = 7.89, p = .095		Wald's χ^2 (4, 50) = 7.08, p = .131	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.17. Hierarchical generalized estimating equations examining associations between maternal general adversity by timing and number of offspring physician-diagnosed ailments (*n* = 57)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Female gender[a]	1.79(0.94, 3.39)	.073	**1.92(1.01, 3.66)**	**.045**	**1.92(1.01, 3.67)**	**.046**
Age[b]	**1.03(1.01, 1.05)**	**<.001**	**1.03(1.01, 1.05)**	**.001**	**1.03(1.00, 1.05)**	**.006**
Offspring adversity	1.01(1.00, 1.02)	.050	1.01(0.99, 1.02)	.069	1.01(0.99, 1.02)	.064
Adversity experiences						
Childhood	1.02(0.98, 1.06)	.197	0.98(0.93, 1.03)	.611	0.98(0.93, 1.03)	.562
Preconception	—	—	**1.05(1.00, 1.11)**	**.023**	**1.05(1.00, 1.11)**	**.046**
Post-conception	—	—	—	—	1.00(0.96, 1.05)	.727
Constant	0.23(0.09, 0.59)	.002	0.22(0.09, 0.57)	.002	0.23(0.09, 0.60)	.003
Model Statistics	Wald's χ^2 (4, 57) = 24.93, $p < .001$		Wald's χ^2 (5, 57) = 30.84, $p < .001$		Wald's χ^2 (6, 57) = 31.33, $p < .001$	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.18. Hierarchical generalized estimating equations examining associations between maternal law enforcement adversity by timing and offspring self-rated health (*n* = 51)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	**0.87(0.40, 1.33)**	**<.001**	**0.87(0.37, 1.37)**	**.001**	**0.88(0.43, 1.32)**	**<.001**
Age[b]	0.00(-0.02, 0.03)	.601	0.00(-0.02, 0.03)	.606	0.00(-0.02, 0.03)	.555
Offspring adversity	0.00(-0.01, 0.01)	.633	0.00(-0.00, 0.01)	.626	0.00(-0.00, 0.01)	.452
Adversity experiences						
Childhood	0.02(-0.08, 0.13)	.633	0.02(-0.15, 0.21)	.757	0.10(-0.08, 0.29)	.285
Preconception	—	—	-0.00(-0.20, 0.19)	.960	0.03(-0.16, 0.24)	.724
Post-conception	—	—	—	—	**-0.23(-0.37, -0.08)**	**.002**
Constant	1.40(0.55, 2.24)	.001	1.39(0.55, 2.24)	.001	1.50(0.71, 2.29)	<.001
Model Statistics	Wald's χ^2 (4, 51) = 13.88, p = .007		Wald's χ^2 (5, 51) = 13.92, p = .016		Wald's χ^2 (6, 51) = 29.63, p < .001	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Table 3.19. Hierarchical generalized estimating equations examining associations between maternal general adversity by timing and offspring waist-to-height ratio (n = 56)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	0.05(-0.00, 0.11)	.062	**0.07(0.01, 0.12)**	**.018**	0.07(0.01, 0.12)	.017
Age[b]	0.00(-0.00, 0.00)	.127	0.00(-0.00, 0.00)	.131	0.00(-0.00, 0.00)	.102
Offspring adversity	**0.00(0.00, 0.00)**	**.017**	**0.00(0.00, 0.00)**	**.016**	**0.00(0.00, 0.00)**	**.018**
Adversity experiences						
Childhood	-0.00(-0.00, 0.00)	.070	**-0.00(-0.01, -0.00)**	**.001**	**-0.00(-0.01, -0.00)**	**.003**
Preconception	—	—	**0.00(0.00, 0.01)**	**.007**	**0.00(0.00, 0.01)**	**.006**
After conception	—	—	—	—	-0.00(-0.00, 0.00)	.528
Constant	0.34(0.25, 0.44)	<.001	0.34(0.25, 0.43)	<.001	0.33(0.24, 0.43)	<.001
Model Statistics	Wald's χ^2 (4, 56) = 17.12, p = .001		Wald's χ^2 (5, 56) = 26.56, p < .001		Wald's χ^2 (6, 56) = 27.15, p < .001	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Discussion

Using a dyadic sample of African-American mothers and their biological adult children, this study provides evidence that there are associations between adverse experiences and health outcomes both within and across generations. By utilizing a more nuanced approach, findings from this study build on previous empirical work, provide additional insight into how both the timing and type of adversity contribute to health, and deliver justification for further investigation.

When exploring the number of physician-diagnosed health ailments in offspring, results showed that maternal preconception general adversity was significantly associated with more ailments while childhood and post-conception experiences were not after controlling for offspring adversity. In addition, general preconception adversity was linked to a larger offspring waist-to-height ratio, signaling poorer health. One potential explanation for these findings is that mothers experiencing general adversity during the preconception period may have been more likely to initiate behaviors during this life phase such as smoking or substance use that have been linked to poor offspring outcomes (Gavin et al., 2011; Smith et al., 2016), and these impacts may have persisted to influence offspring health in adulthood.

In addition to these findings supporting prior work linking general parental adversity to poorer offspring health (Cammack et al., 2019; Gray et al., 2017), they also introduce new facets to this evidence base. First, these health outcomes in relation to parental adversity were demonstrated in AA adult offspring as opposed to the more frequently studied outcomes captured during the first few years of offspring's life. This may suggest that these intergenerational health associations continue beyond early life and are connected to other, more long-term health outcomes reported by the offspring. Next, these findings suggest that some

forms of parental adversity exposure may have an enduring impact on offspring health, even after accounting for offspring's own cumulative exposure to adversity. By including adult offspring who were able to report on their own adversity exposure and because single generation analyses showed some significant links between their own adversity and health, controlling for these associations allowed for a more accurate assessment of whether parental adversity may be linked to offspring health. Finally, the results from this study shed light on how the timing of parental adversity may be associated with the links to offspring health by demonstrating significant associations in specific phases of life (e.g., childhood, adulthood preconception) while controlling for the impact of other adjacent life phases. This implies that differences may exist in the links between parental adversity and offspring health as a function of the timing in which it occurs and could be a key consideration for future research efforts.

Despite having study findings that would be reinforced by existing empirical literature, there were also several results regarding intergenerational health associations that are not consistent with the hypothesized relationships. Contrary to what past work might suggest, findings showed a link between maternal law enforcement adversity and offspring self-rated health such that post-conception experiences specifically (i.e., during the prenatal period and later) were associated with their offspring reporting better health. Post-conception includes the period during which a mother may have been pregnant with the child who participated in this study and prior empirical research would predict that maternal prenatal adversity would be negatively associated with her child's health (Cao-Lei et al., 2020; Van den Bergh et al., 2020; Walsh et al., 2019). However, it is important to note that the post-conception period spans several years up until when the survey was completed and mothers were not asked specifically to indicate whether events happened while they were pregnant. Consequently, it is possible that the

positive association demonstrated between these maternal events and better self-rated offspring health may be disproportionately driven by more recent events not occurring during the mother's pregnancy. In addition, self-rated health is more subjective in nature and offspring respondents may have answered in a more socially desirable way that did not accurately reflect their health status and accounted for the positive association observed.

Another intriguing finding showed that general childhood adversity was significantly linked to a smaller waist-to-height ratio in offspring, an indication of better health. However, it is important to note that the size of this association was very small as the beta coefficient represents only a slight decrease on a ratio that spans between zero and one, making it difficult to determine the actual health impact in offspring. Waist-to-height ratio is an index of abdominal obesity and 0.5 is generally accepted as a universal cutoff for central obesity, with ratios above 0.5 representing a greater risk for cardiometabolic conditions, cardiovascular disease, and years of life lost (Kazlauskaite et al., 2017; Saava, Lamnisos, & Kafatos, 2013). Consequently, it may have been preferred to analyze offspring waist-to-height ratio as a dichotomous variable to examine the likelihood of being at risk for several other health outcomes, but due to the limited sample size, it was investigated as a continuous outcome.

There were also analyses that reported non-significant findings that would be contradicted by the research literature. Most unexpectedly, maternal racial discrimination was not a significant predictor of any of the offspring health outcomes measured in this study when analyzed by the timing in the mother's life or across her lifetime. One reason for these observed results may lie in the fact that maternal racial discrimination experienced directly was mainly measured with only a few instances of indirect or vicarious discrimination being captured and solely in the context of the law enforcement adversity measure (e.g., if a police officer ever used

physical force against a close friend or family member). Previous research examining intergenerational associations between parental racial discrimination and offspring health in AAs specifically has provided evidence implying that vicarious or indirect forms of parental racial discrimination may be a significant predictor of offspring health above and beyond direct experiences (Daniels et al., 2020; Dominguez et al., 2008). This notion is further supported by additional work explaining that adversity experienced by members of one's social support network is highly salient for AA women and even more so when the adversity involves an element of a shared identity such as racial discrimination (Woods-Giscombé, Lobel, Zimmer, Wiley Cené, & Corbie-Smith, 2015).

In single generation analyses of maternal adversity and their own health, post-conception general adversity was positively associated with the number of physician-diagnosed health ailments while controlling for childhood and preconception law enforcement adversity and covariates. In contrast, maternal post-conception law enforcement adversity was negatively associated with the number of physician-diagnosed health ailments while controlling for childhood and preconception law enforcement adversity as well as covariates. This finding represents a considerable departure from what would be expected with respect to previous work for several reasons. First, greater childhood adversity has been shown to play a significant role in contributing to poorer adulthood health (McKay et al., 2021; Sweeting et al., 2020), but the results of the current study failed to detect an association for childhood law enforcement adversity. Next, past research has also drawn specific links between law enforcement adversity and poorer health (Jackson et al., 2020; Zeiders et al., 2021), but the only significant finding suggested that after conception law enforcement adversity was linked to better health in the form of fewer health ailments. Exploring maternal waist-to-height ratio as an outcome showed that

childhood general adversity was linked to a smaller ratio (i.e., better health), preconception general adversity was linked to a larger ratio (i.e., poorer health), and post-conception was not significantly associated. As mentioned previously, both of these effects were extremely small and the degree to which these results convey meaningful changes in health remains unclear, but undoubtedly warrant future exploration.

Next, examination of links between offspring adversity and their health demonstrated that general lifetime adversity was significantly linked with more physician-diagnosed health ailments and poorer self-rated health after controlling for several covariates while lifetime law enforcement adversity and racial discrimination were not associated with the number of health ailments or self-rated health. After distinguishing general adversity by timing, results illustrated that childhood experiences had the strongest association with more health ailments and poorer self-rated health as adulthood adversity was not significantly related to either of these outcomes. Finally, waist-to-height ratio seemed to be unrelated to offspring adversity regardless of timing or type of adversity. One on hand, these findings are consistent with prior work that emphasizes the importance of early life experiences in poorer subsequent health outcomes (McKay et al., 2021; Sweeting et al., 2020), but deviate from other evidence that links law enforcement and racial discrimination to worse health (Hill et al., 2017; Jackson et al., 2020; Mouzon et al., 2017; Zeiders et al., 2021). Furthermore, none of the lifetime offspring adversity types were significantly associated with waist-to-height ratio. A potential explanation for these findings could be that this sample of adult offspring primarily recruited from HCBUs may differ from the larger AA population in the sense that they may have greater access to social support networks that are better equipped to buffer them from the potential health implications of race-related adversity. In other words, having an affiliation with a HBCU might provide this group with a

community that shares a common experience and can assist in several ways that can help mitigate the repercussions of these experiences including sharing resources, providing emotional support, and finding spiritual strength (Cooper et al., 2013; Seawell, Cutrona, & Russell, 2014)

Limitations

Although this study contributes unique evidence to the current literature investigating intergenerational health in AA families, it is important to address several limitations. First, this dyadic sample was comprised of AA college students and their parents almost exclusively from HBCUs and this may not be representative of this community as a whole with respect to students who do not attend HBCUs or young adults who are not enrolled in college. Even though the sample included some dyads with fathers, there were not enough to properly control for potential gender differences in parents and as a result, fathers were dropped from the final analytic sample. The exclusion of fathers prevented the investigation of how their life adversity experiences may be associated with their offspring's health. There may be inherent differences in the frequency, type, and severity of adversity that AA men encounter compared to women as a function of their gender and this study was unable to address how men's experiences are linked to the health of their offspring. Similarly, most children in the study were women (82%), resulting in a diminished ability to detect potential associations specific to male offspring and the different health outcomes that they may experience.

Another potential limitation revolves around the way in which racial discrimination was captured. The primary focus of these measures was on experiences that happened directly to respondents, but assessed little information regarding indirect or vicarious forms of racial discrimination. This study was unable to detect any significant associations between parental racial discrimination experiences and offspring health despite what previous literature has found

and this may have been exacerbated by the fact that only direct experiences were captured. Additionally, the final analytic sample was comprised only of mothers and past work has pointed to the significance that indirect experiences of adversity can have on health in AA women specifically (Woods-Giscombé et al., 2015). Failing to record information on indirect maternal experiences explicitly may have considerably hampered the ability to detect intergenerational relationships with offspring health (Woods-Giscombé et al., 2015).

Finally, the way in which maternal adversity was captured might have prevented the ability to definitively account for the impact of prenatal adversity. On the various adversity measures, the post-conception period asked mothers to report on events that happened after their child was conceived, which included when they were pregnant, after they had given birth, and up until the moment they were taking the survey. However, the question did not specifically ask mothers to indicate adversity experiences that occurred while they were pregnant and as a result, a clear measure of prenatal adversity was not captured. Furthermore, it may have been challenging for respondents to recall when certain experiences occurred and accurately indicate the corresponding time period on the survey form. Sample size was also a limitation as it prevented the inclusion of all relevant variables within each analysis.

Conclusion

This study highlights the significance of capturing a detailed account of parental adversity with regard to timing and type when exploring intergenerational impacts of adversity in AAs. Additionally, this study illustrates the utility of gathering measures of adversity from adult offspring and simultaneously controlling for the impact that their own experiences may have on their health when analyzing associations with their parents' adversity. This study represents one of the first forays into addressing the intergenerational transmission of adversity on health in a

comprehensive manner and serve as an example of how future research can continue to diminish the gaps in knowledge for these issues in AAs.

References

Aalbaek, F. S., Graff, S., & Vestergaard, M. (2017). Risk of stroke after bereavement—a systematic literature review. *ACTA Neurologica Scandinavica, 136(4)*, 293-297. doi: 10.1111/ane.12736

Alang, S., McAlpine, D., McCreedy, E., & Hardeman, R. (2017). Police brutality and black health: Setting the agenda for public health scholars. *American Journal of Public Health, 107(5)*, 662-665. doi: 10.2105/AJPH.2017.303691

Ambresin, G., Chondros, P., Dowrick, C., Herrman, H., & Gunn, J. M. (2014). Self-rated health and long-term prognosis of depression. *Annals of Family Medicine, 12*, 57-65. doi: 10.1370/afm.1562

American College Health Association (2013). *American College Health Association-National College Health Assessment II: Reliability and Validity Analyses 2011*. Hanover, MD

Anderson, L. M., Riffle, L., Wilson, R., Travlos, G. S., Lubomirski, M. S., & Alvord, W. G. (2006). Preconceptional fasting of fathers alters serum glucose in offspring of mice. *Nutrition, 22*, 327-331. doi: 10.1016/j.nut.2005.09.006

Baldwin, J. R., Reuben, A., Newbury, J. B., & Danese, A. (2019). Agreement between prospective and retrospective measures of childhood maltreatment: A systematic review and meta-analysis. *JAMA Psychiatry, 76(6)*, 584-593. doi: 10.1001/jamapsychiatry.2019.0097

Blum, S. C., Silver, R. C., & Poulin, M. J. (2014). Perceiving risk in a dangerous world: Associations between life experiences and risk perceptions. *Social Cognition, 32*, 297-314. doi: 10.1521/soco.2014.32.3.297

Bor, J., Venkataramani, A. S., Williams, D. R., & Tsai, A. C. (2018). Police killings and their

spillover effects on the mental health of black Americans: A population-based, quasi-experimental study. *The Lancet, 392(10144)*, 302-310. doi: 10.1016/S0140-6736(18)31130-9

Borsini, A., Hepgul, N., Mondelli, V., Chalder, T., & Pariante, C. M. (2014). Childhood stressors in the development of fatigue syndromes: A review of the past 20 years of research. *Psychological Medicine, 44(9)*, 1809-1823. doi: 10.1017/S0033291713002468

Breslau, N., Kessler, R. C., Chilcoat, H. D., Schultz, L. R., Davis, G. C., & Andreski, P. (1998). Trauma and posttraumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma. *Archives of General Psychiatry, 55*, 626-632. doi: 10.1001/archpsyc.55.7.626

Brondolo, E., Byer, K., Gianaros, P. J., Liu, C., Prather, A. A., Thomas, K., Keita, G., & Woods-Giscombé, C. L. (2017). Stress and health disparities: Contexts, mechanisms, and interventions among racial/ethnic minority and low socioeconomic status populations. *American Psychological Association (APA) Working Group Report*. Retrieved from http://www.apa.org/pi/health-disparities/resources/stress-report.aspx

Brondolo, E., Kelly, K. P., Coakley, V., Gordon, T., Thompson, S., Levy, E., Cassells, A., Tobin, J. N., Sweeney, M., & Contrada, R. J. (2005). The perceived ethnic discrimination questionnaire: Development and preliminary validation of a community version 1. *Journal of Applied Social Psychology*, *35*, 335-365. doi: 10.1111/j.1559-1816.2005.tb02124.x

Brunson, R. K., & Miller, J. (2006). Gender, race, and urban policing: The experience of African American youths. *Gender & Society, 20(4)*, 531-552. doi: 10.1177/0891243206287727

Cao-Lei, L., de Rooij, S. R., King, S., Matthews, S. G., Metz, G. A., Roseboom, T. J., & Szyf,

M. (2020). Prenatal stress and epigenetics. *Neuroscience & Biobehavioral Reviews, 117*, 198-210. doi: 10.1016/j.neubiorev.2017.05.016

Cao-Lei, L., Dancause, K. N., Elgbeili, G., Massart, R., Szyf, M., Liu, A., Laplante, D. P., & King, S. (2015). DNA methylation mediates the impact of exposure to prenatal maternal stress on BMI and central adiposity in children at age 13½ years: Project Ice Storm. *Epigenetics, 10(8),* 749-761. doi:10.1080/15592294.2015.1063771

Carone, B. R., Fauquier, L., Habib, N., Shea, J. M., Hart, C. E., Li, R., Bock, C., Li, C., Gu, H., Zamore, P. D., Meissner, A., Weng, Z., Hofmann, H. A., Friedman, N., & Rando, O. J. (2010). Paternally induced transgenerational environmental reprogramming of metabolic gene expression in mammals. *Cell, 143,* 1084-1096. doi: 10.1016/j.cell.2010.12.008

Carr, C. P., Martins, C. M. S., Stingel, A. M., Lemgruber, V. B., & Juruena, M. F. (2013). The role of early life stress in adult psychiatric disorders: A systematic review according to childhood trauma subtypes. *The Journal of Nervous and Mental Disease, 201(12),* 1007-1020. doi: 10.1097/NMD.0000000000000049

Chan, J. C., Nugent, B. M., & Bale, T. L. (2018). Parental advisory: Maternal and paternal stress can impact offspring neurodevelopment. *Biological Psychiatry, 83,* 886-894. doi: 10.1016/j.biopsych.2017.10.005

Chen, E., Shalowitz, M. U., Story, R. E., Ehrlich, K. B., Manczak, E. M., Ham, P. J., Van Le, B. S., & Miller, G. E. (2017). Parents' childhood socioeconomic circumstances are associated with their children's asthma outcomes. *Journal of Allergy and Clinical Immunology, 140(3),* 828-835. doi: 10.1016/j.jaci.2016.11.040

Cheng, E. R., Park, H., Wisk, L. E., Mandell, K. C., Wakeel, F., Litzelman, K., Chatterjee, D., &

Witt, W. P. (2016). Examining the link between women's exposure to stressful life events prior to conception and infant and toddler health: The role of birth weight. *Journal of Epidemiology and Community Health, 70(3),* 245-252. doi: 10.1136/jech-2015-205848

Cook, W. L., & Kenny, D. A. (2005). The actor–partner interdependence model: A model of bidirectional effects in developmental studies. *International Journal of Behavioral Development, 29(2),* 101-109. doi: 10.1080/01650250444000405

Cooper, S. M., Brown, C., Metzger, I., Clinton, Y., & Guthrie, B. (2013). Racial discrimination and African American adolescents' adjustment: Gender variation in family and community social support, promotive and protective factors. *Journal of Child and Family Studies, 22(1),* 15-29. doi: 10.1007/s10826-012-9608-y

Coxe, S., West, S. G., & Aiken, L. S. (2013). Generalized linear models. In T. Little (Ed.) *Oxford handbook of quantitative methods, Vol 2: statistical analysis*. New York: Oxford University Press.

Coxe, S., West, S. G., & Aiken, L. S. (2009). The analysis of count data: A gentle introduction to Poisson regression and its alternatives. *Journal of Personality Assessment, 91*, 121-136. doi: 10.1080/00223890802634175

Daniels, K. P., Valdez, Z., Chae, D. H., & Allen, A. M. (2020). Direct and vicarious racial discrimination at three life stages and preterm labor: Results from the African American Women's Heart & Health Study. *Maternal and Child Health Journal, 24(11),* 1387-1395. doi: 10.1007/s10995-020-03003-4

Davis, E., Whyde, A., & Langton, L. (2018). Contacts between police and the public, 2015. *US*

Department of Justice Office of Justice Programs Bureau of Justice Statistics Special Report, 1-33. Retrieved from https://dailycaller.com/wp-content/uploads/2018/10/BJS-Data.pdf

DeVylder, J., Oh, H., Nam, B., Sharpe, T., Lehmann, M., & Link, B. (2017). Prevalence, demographic variation and psychological correlates of exposure to police victimisation in four US cities. *Epidemiology and Psychiatric Sciences, 26*, 466-477. doi: 10.1017/S2045796016000810

Dominguez, T. P., Dunkel-Schetter, C., Glynn, L. M., Hobel, C., & Sandman, C. A. (2008). Racial differences in birth outcomes: The role of general, pregnancy, and racism stress. *Health Psychology, 27(2)*, 194-203. doi:10.1037/0278-6133.27.2.194

Eberle, C., Fasig, T., Brüseke, F., & Stichling, S. (2021). Impact of maternal prenatal stress by glucocorticoids on metabolic and cardiovascular outcomes in their offspring: A systematic scoping review. *PloS One, 16(1)*, e0245386. doi: 10.1371/journal.pone.0245386

Ennis, J., & Majid, U. (2021). "Death from a broken heart": A systematic review of the relationship between spousal bereavement and physical and physiological health outcomes. *Death Studies, 45(7),* 538-551. doi: 10.1080/07481187.2019.1661884

Everitt, B. S. (Ed.). (2005). Generalized Linear Models (GLM). Encyclopedia of Statistics in Behavioral Science.

Fatal Force: 2018 police shootings database. Washington Post. [cited 2018 Jul 10]. Retrieved from: https://www.washingtonpost.com/graphics/2018/national/police-shootings-2018/

Fayers, P. M., & Sprangers, M. A. (2002). Understanding self-rated health. *The Lancet, 359,* 187-188. doi: 10.1016/S0140-6736(02)07466-4

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14(4),* 245-258. doi: 10.1016/S0749-3797(98)00017-8

Friedman, E. M., Montez, J. K., Sheehan, C. M., Guenewald, T. L., & Seeman, T. E. (2015). Childhood adversities and adult cardiometabolic health: Does the quantity, timing, and type of adversity matter? *Journal of Aging and Health, 27(8),* 1311-1338. doi: 10.1177/0898264315580122

Friedman, W., Lurigio, A. J., Greenleaf, R., & Albertson, S. (2004). Encounters between police officers and youths: The social costs of disrespect. *Journal of Crime and Justice, 27(2),* 1-25. doi: 10.1080/0735648X.2004.9721193

Gapp, K., von Ziegler, L., Tweedie-Cullen, R. Y., & Mansuy, I. M. (2014). Early life epigenetic programming and transmission of stress-induced traits in mammals: How and when can environmental factors influence traits and their transgenerational inheritance? *Bioessays, 36,* 491-502. doi: 10.1002/bies.201300116

Gavin, A. R., Hill, K. G., David Hawkins, J., & Maas, C. (2011). The role of maternal early life and later-life risk factors on offspring low birth weight: Findings from a three-generational study. *Journal of Adolescent Health, 49(2),* 166-171. doi: 10.1016/j.jadohealth.2010.11.246

Gillespie, S. L., Christian, L. M., Alston, A. D., & Salsberry, P. J. (2017). Childhood stress and birth timing among African American women: Cortisol as biological mediator. *Psychoneuroendocrinology, 84,* 32-41. doi:10.1016/j.psyneuen.2017.06.009

Gray, S. A., Jones, C. W., Theall, K. P., Glackin, E., & Drury, S. S. (2017). Thinking across generations: Unique contributions of maternal early life and prenatal stress to infant physiology. *Journal of the American Academy of Child & Adolescent Psychiatry, 56(11)*, 922-929. doi:10.1016/j.jaac.2017.09.001

Hanley, J. A., Negassa, A., Edwardes, M. D. D., & Forrester, J. E. (2003). Statistical analysis of correlated data using generalized estimating equations: An orientation. *American Journal of Epidemiology, 157(4)*, 364-375. doi: 10.1093/aje/kwf215

Harrell, S. P. (2000). A multidimensional conceptualization of racism-related stress: Implications for the well-being of people of color. *American Journal of Orthopsychiatry, 70*, 42-57. doi: 10.1037/h0087722

Harrell, J. P., Hall, S., & Taliaferro, J. (2003). Physiological responses to racism and discrimination: An assessment of the evidence. *American Journal of Public Health, 93(2)*, 243-248. doi: 10.2105/AJPH.93.2.243

Hilmert, C. J., Dominguez, T. P., Dunkel Schetter, C., Srinivas, S. K., Glynn, L. M., Hobel, C. J., & Sandman, C. A. (2014). Lifetime racism and blood pressure changes during pregnancy: Implications for fetal growth. *Health Psychology, 33(1),* 43-51. doi: 10.1037/a0031160

Holman, E. A., Silver, R. C., & Waitzkin, H. (2000). Traumatic life events in primary care patients: A study in an ethnically diverse sample. *Archives of Family Medicine, 9*, 802-810. doi: 10.1001/archfami.9.9.802

Jackson, A. S., Blair, S. N., Mahar, M. T., Wier, L. T., Ross, R. M., & Stuteville, J. E. (1990). Prediction of functional aerobic capacity without exercise testing. *Medicine and Science in Sports and Exercise, 22(6),* 863-870. doi: 10.1249/00005768-199012000-00021

Jackson, D. B., Fahmy, C., Vaughn, M. G., & Testa, A. (2019). Police stops among at-risk youth:

repercussions for mental health. *Journal of Adolescent Health, 65(5),* 627-632. doi: 10.1016/j.jadohealth.2019.05.027

Jackson, D. B., Testa, A., Vaughn, M. G., & Semenza, D. C. (2020). Police stops and sleep behaviors among at-risk youth. *Sleep Health, 6(4),* 435-441. doi: 10.1016/j.sleh.2020.02.006

Kazlauskaite, R., Avery-Mamer, E. F., Li, H., Chataut, C. P., Janssen, I., Powell, L. H., & Kravitz, H. M. (2017). Race/ethnic comparisons of waist-to-height ratio for cardiometabolic screening: The study of women's health across the nation. *American Journal of Human Biology, 29(1),* e22909. doi: 10.1002/ajhb.22909

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry, 52,* 1048-1060. doi: 10.1001/archpsyc.1995.03950240066012

Kuzawa, C. W., & Quinn, E. A. (2009). Developmental origins of adult function and health: Evolutionary hypotheses. *Annual Review of Anthropology, 38,* 131-147. doi: 10.1146/annurev-anthro-091908-164350

Margerison-Zilko, C. E., Strutz, K. L., Li, Y., & Holzman, C. (2017). Stressors across the life-course and preterm delivery: Evidence from a pregnancy cohort. *Maternal and Child Health Journal, 21(3),* 648-658. doi: 10.1007/s10995-016-2151-5

McKay, M. T., Cannon, M., Chambers, D., Conroy, R. M., Coughlan, H., Dodd, P., Healy, C., O'Donnell, L., & Clarke, M. C. (2021). Childhood trauma and adult mental disorder: A systematic review and meta-analysis of longitudinal cohort studies. *Acta Psychiatrica Scandinavica, 143(3),* 189-205. doi: 10.1111/acps.13268

McLeod, M. N., Heller, D., Manze, M. G., & Echeverria, S. E. (2020). Police interactions and

the mental health of Black Americans: A systematic review. *Journal of Racial and Ethnic Health Disparities, 7(1),* 10-27. doi: 10.1007/s40615-019-00629-1

Mersky, J. P., & Lee, C. P. (2019). Adverse childhood experiences and poor birth outcomes in a diverse, low-income sample. *BMC Pregnancy and Childbirth, 19(1)*, 387. doi: 10.1186/s12884-019-2560-8

Mossey, J. M., & Shapiro, E. (1982). Self-rated health: A predictor of mortality among the elderly. *American Journal of Public Health, 72(8)*, 800-808. doi: 10.2105/AJPH.72.8.800

Myers, R. H., & Montgomery, D. C. (1997). A tutorial on generalized linear models. *Journal of Quality Technology, 29(3)*, 274-291. doi: 10.1080/00224065.1997.11979769

Negriff, S. (2020). ACEs are not equal: Examining the relative impact of household dysfunction versus childhood maltreatment on mental health in adolescence. *Social Science & Medicine, 245,* 112696. doi: 10.1016/j.socscimed.2019.112696

Nelson, C. A., Bhutta, Z. A., Harris, N. B., Danese, A., & Samara, M. (2020). Adversity in childhood is linked to mental and physical health throughout life. *BMJ, 371,* m3048. doi: 0.1136/bmj.m3048

Pembrey, M. E., Bygren, L. O., Kaati, G., Edvinsson, S., Northstone, K., Sjöström, M., & Golding, J. (2006). Sex-specific, male-line transgenerational responses in humans. *European Journal of Human Genetics, 14*, 159-166. doi: 10.1038/sj.ejhg.5201538

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health diagnostic interview schedule: Its history, characteristics, and validity. *Archives of General Psychiatry, 38*, 381-389. doi: 10.1001/archpsyc.1981.01780290015001

Savva, S. C., Lamnisos, D., & Kafatos, A. G. (2013). Predicting cardiometabolic risk: waist-to-

height ratio or BMI. A meta-analysis. *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy, 6*, 403-419. doi: 10.2147/DMSO.S34220

Seawell, A. H., Cutrona, C. E., & Russell, D. W. (2014). The effects of general social support and social support for racial discrimination on African American women's well-being. *Journal of Black Psychology, 40(1)*, 3-26. doi: 10.1177/0095798412469227

Seery M. D., Holman, E. A., & Silver, R. C. (2010). Whatever does not kill us: Cumulative lifetime adversity, vulnerability, and resilience. *Journal of Personality and Social Psychology, 99*, 1025-1041. doi:10.1037/a0021344

Sewell, A. A., & Jefferson, K. A. (2016). Collateral damage: The health effects of invasive police encounters in New York City. *Journal of Urban Health, 93(1)*, 42-67. doi: 10.1007/s11524-015-0016-7

Singh-Manoux, A., Guéguen, A., Martikainen, P., Ferrie, J., Marmot, M., & Shipley, M. (2007). Self-rated health and mortality: Short- and long-term associations in the Whitehall II study. *Psychosomatic Medicine, 69*, 138-143. doi: 10.1097/PSY.0b013e318030483a

Skiba, R. J., Horner, R. H., Chung, C. G., Karega Rausch, M., May, S. L., & Tobin, T. (2011). Race is not neutral: A national investigation of African American and Latino disproportionality in school discipline. *School Psychology Review, 40*, 85-107. Doi: 10.1080/02796015.2011.12087730

Smith, M. V., Gotman, N., & Yonkers, K. A. (2016). Early childhood adversity and pregnancy outcomes. *Maternal and Child Health Journal, 20(4)*, 790-798. doi:10.1007/s10995-015-1909-5

Soubry, A., Schildkraut, J. M., Murtha, A., Wang, F., Huang, Z., Bernal, A., Kurtzberg, J., Jirtle,

R. L., Murphy, S. K., & Hoyo, C. (2013). Paternal obesity is associated with IGF2 hypomethylation in newborns: Results from a Newborn Epigenetics Study (NEST) cohort. *BMC Medicine, 11*, 29. doi: 10.1186/1741-7015-11-29

Sweeting, J. A., Akinyemi, A. A., & Holman, E. A. (2022). Parental preconception adversity and offspring health in African Americans: A systematic review of intergenerational studies. *Trauma, Violence, & Abuse, 0,* 1-16. doi: 10.1177/15248380221074320

Sweeting, J. A., Garfin, D. R., Holman, E. A., & Silver, R. C. (2020). Associations between exposure to childhood bullying and abuse and adulthood outcomes in a representative national U.S. sample. *Child Abuse & Neglect, 101,* 104048. doi: 10.1016/j.chiabu.2019.104048

U.S. Census Bureau QuickFacts: United States. (2021). Retrieved April 30, 2022, from https://www.census.gov/quickfacts/fact/table/US/PST045217.

U.S. Department of Health and Human Services, National Center for Health Statistics (2000). *National Health Interview Survey Questionnaire*. Hyattsville, MD.

Van den Bergh, B. R., van den Heuvel, M. I., Lahti, M., Braeken, M., de Rooij, S. R., Entringer, S., Hoyer, D., Roseboom, T., Räikkönen, K., & Schwab, M. (2020). Prenatal developmental origins of behavior and mental health: The influence of maternal stress in pregnancy. *Neuroscience & Biobehavioral Reviews, 117,* 26-64. doi: 10.1016/j.neubiorev.2017.07.003

Walsh, K., McCormack, C. A., Webster, R., Pinto, A., Lee, S., Feng, T., Sloan Krakovsky, H., O'Grady, S. M., Tycko, B., Champagne, F. A., Werner, E. A., Liu, G., & Monk, C. (2019). Maternal prenatal stress phenotypes associate with fetal neurodevelopment and

birth outcomes. *Proceedings of the National Academy of Sciences, 116(48),* 23996-24005. doi: 10.1073/pnas.1905890116

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54(6),* 1063-1070. doi: 10.1037/0022-3514.54.6.1063

Wei, Y., Yang, C. R., Wei, Y. P., Zhao, Z. A., Hou, Y., Schatten, H., & Sun, Q. Y. (2014). Paternally induced transgenerational inheritance of susceptibility to diabetes in mammals. *Proceedings of the National Academy of Sciences, 111,* 1873-1878. doi: 10.1073/pnas.1321195111

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

Williams, D. R., & Williams-Morris, R. (2000). Racism and mental health: The African American experience. *Ethnicity & Health, 5(3-4),* 243-268. doi: 10.1080/713667453

Woods-Giscombé, C. L., Lobel, M., Zimmer, C., Wiley Cené, C., & Corbie-Smith, G. (2015). Whose stress is making me sick? Network-stress and emotional distress in African-American women. *Issues in Mental Health Nursing, 36(9),* 710-717. doi: 10.3109/01612840.2015.1011759

Zeiders, K. H., Umaña-Taylor, A. J., Carbajal, S., & Pech, A. (2021). Police discrimination among Black, Latina/x/o, and White adolescents: Examining frequency and relations to academic functioning. *Journal of Adolescence, 90,* 91-99. doi: 10.1016/j.adolescence.2021.06.001

Zorn, C. J. (2001). Generalized estimating equation models for correlated data: A review with

applications. *American Journal of Political Science, 45(2)*, 470-490.

Chapter 4: Epilogue

Epilogue

For the past several hundred years, AAs in the United States have been exposed to an ongoing epidemic of cascading stress and trauma that is distinct from other racial groups while simultaneously having some of the worst health outcomes. Empirical research has gradually begun to illuminate the interdependent relationships between adversity and health, as well as how these relationships can reverberate throughout multiple familial generations. This dissertation contributes to the expanding body of intergenerational work by exploring how the specific timing of adversity experienced in one generation is associated with the health outcomes of a subsequent generation, the differential impact that various types of life adversity can have on generational health, and how these associations emerge in the AA population specifically.

Chapter 2 utilized a systematic review to characterize the presently available empirical literature exploring associations between parental preconception adversity and offspring health in AA families. Twenty-five studies reported significant associations between parental preconception adversity and offspring health, but only six of these studies reported associations specific to AAs. Findings highlighted links between predominantly universal forms of parental adversity (e.g., ACEs, general life stress), along with some race-specific experiences (e.g., racism exposure) and several birth and early life outcomes in offspring (e.g., asthma symptoms, birth timing, stress responsivity). Furthermore, a number of potential mechanisms responsible for the intergenerational transmission of adversity on subsequent health were identified and measured, including maternal prenatal physiological changes (e.g., blood pressure, cortisol, inflammatory biomarkers) and behavioral factors (e.g., smoking, substance use). Despite the limited nature of the findings with respect to AA families specifically, this review identified several important gaps in the literature.

Building on the findings from the previous chapter, Chapter 3 aimed to address several of the research gaps uncovered by examining links between lifetime parental adversity and offspring health in a sample of biological mothers and their adult children. More specifically, this study aimed to further tease apart the distinctive effects that parental adversity can have on adult offspring health as a function of the timing and type of adversity experienced, while concurrently accounting for the impact of the offspring's own adversity exposure. Results showed that maternal preconception general adversity experiences were associated with a greater number of offspring health ailments, while greater maternal post-conception law enforcement adversity was linked to their offspring reporting better self-rated health. Lastly, maternal childhood general adversity was associated with a smaller offspring waist-to-height ratio (i.e., better health), while preconception general adversity was linked to a larger waist-to-height ratio (i.e., poorer health).

This is likely one of the first studies to explicitly examine multiple types of parental adversity while also taking into account the timing of each adversity type and offspring adversity exposure in a dyadic sample of AA mothers and their adult children. The overwhelming majority of current intergenerational health studies address prenatal adversity, but do not control for other time periods of adversity, do not control for offspring adversity experiences, focus on singular types of adversity, or do not report results that speak directly to the unique experiences of AAs. Taken together, these projects provide a unique, more nuanced approach to studying associations between parental adversity and subsequent offspring health through the specification of the timing of parental adversity, comparing potential differences in the impact that adversities may have based on their type, capturing directly-reported adult offspring health beyond the birth and early life outcomes that have been most frequently studied in intergenerational health research in

AAs explicitly, and accounting for the impact of offspring's own adversity exposure on their health.

Limitations and other considerations

Throughout this dissertation, the focus of parental adversity has been almost exclusively focused on mothers, and this presents a significant sex-based, gender-role bias. In addition to the importance that maternal experiences have in shaping offspring outcomes, mounting evidence highlights the need to account for the impact of paternal adversity (Braun et al., 2017; Day et al., 2016; Gapp, von Ziegler, Tweedie-Cullen, & Mansuy, 2014). The foundation of this evidence originates from the animal literature showing that paternal experiences prior to conception may be linked to changes in paternal sperm microRNAs associated with olfactory system neuroanatomy in offspring, lower HPA-axis response, and increased expression of glucocorticoid-responsive genes in the brains of offspring (Dias & Ressler, 2014; Rodgers, Morgan, Bronson, Revello, & Bale, 2013; Rodgers, Morgan, Leu, & Bale, 2015). In addition, empirical work has demonstrated how irregular paternal preconception diet and metabolic states may impact offspring adiposity, cholesterol ester concentrations in the liver, disease vulnerability, insulin resistance, obesity, serum glucose levels, and related cardiometabolic outcomes (Anderson et al., 2006; Carone et al., 2010; Fullston et al., 2015; Ng et al., 2010; Sharma et al., 2016; Wei et al., 2014). Drawing parallels to the foundational animal research, some of the earliest work investigating paternal exposures and offspring health outcomes in humans was also conducted within the context of nutrition. In multiple studies using birth measures and food supply records from the Swedish Famine in 1836, researchers observed significant links between paternal overeating and an increased risk of diabetes mortality in their

descendants (Kaati, Bygren, & Edvinsson, 2002; Kaati, Bygren, Pembrey, & Sjöström, 2007; Pembrey et al., 2006).

More recently, work has shown that paternal obesity may have an epigenetic effect on DNA methylation in their offspring (Soubry et al., 2013). In addition, scholars have also drawn links between paternal exposure to several environmental toxicants and adverse offspring health outcomes. Studies suggest that paternal exposure to endocrine-disrupting chemicals, hydrocarbons like benzene, diesel and turpentine, pesticides, and wood dust are associated with increased risk of astrocytoma, cryptorchidism, hypospadias, leukemia, and neuroblastoma in offspring (Carlos-Wallace, Zhang, Smith, Rader, & Steinmaus, 2016; De Roos et al., 2001; Morales-Suárez-Varela et al., 2011; Nassar, Abeywardana, Barker, & Bower, 2010; Rocheleau, Romitti, & Dennis, 2009; van Wijngaarden, Stewart, Olshan, Savitz, & Bunin, 2003). It is important to note that even though irregular nutrition and environmental toxicant exposure may not disproportionately affect African Americans as a function of their race directly, other factors that overwhelmingly affect this population (e.g., low SES, residential segregation) may indirectly place them at an increased risk for these exposures. While the contexts of environmental stressors for animals and humans differ considerably, it is clear that negative paternal exposures can contribute to epigenetic modifications in both groups, suggesting that these experiences can be transmitted through the sperm genome to offspring.

Future directions

This body of empirical research would benefit greatly from addressing several essential areas moving forward. First, substantial efforts should be made to engage and involve AA boys, men, and fathers specifically in research efforts aiming to explore the intergenerational health impacts of adversity. In order to improve the frequency of participation by AA men and the

larger AA community in empirical research, it is important to understand the barriers that often prevent them from doing so. Investigators have highlighted numerous potential hurdles for AAs, including distrust due to historical research abuse and racism (e.g., Tuskegee Syphilis study, Moynihan report), inconvenience of participation, lack of information or misinformation regarding research and informed consent processes, lack of meaningful relationship building by research teams, potential stigma about certain topics, privacy and confidentiality concerns, and inadequate recruitment efforts (George, Duran, & Norris, 2014; Huang & Coker, 2010; Wallace & Bartlett, 2013).

Due to the sensitive natures of both life adversity experiences and health status, it is critical for investigators to address and alleviate concerns from potential participants regarding sharing their personal information to obtain meaningful participation. Efforts have been made to survey members from historically underrepresented groups in research and several useful strategies for engaging, reassuring, and ultimately facilitating participation from potential study participants have been compiled (Coker, Huang, & Kashubeck-West, 2009; George et al., 2014; Wallace & Bartlett, 2013). First, it is important for members of a study team to engage in culturally sensitive learning and personal reflection that promote self-awareness regarding how factors like racism and sexism operate to oppress marginalized communities in society. Building a stronger awareness of these issues can allow for a more accurate understanding of the participant's experience and minimize the likelihood of drawing unwarranted conclusions or unknowingly acting in prejudiced ways. Second, building a strong rapport between participants and research staff is critical and this can be accomplished by genuinely explaining the reasons for interest in the study topic, as well as how the data will be used. Incorporating qualified, well-trained research personnel who are members of the community being studied and can

communicate in a relatable, more transparent manner, as well as maintaining an open, honest dialogue with participants to address any concerns or fears that may arise, are also helpful approaches to rapport building. Third, clearly outlining the meaningful benefits for study participation that go beyond monetary compensation, including providing participants with valuable information about effectively coping with adversity experiences, improving health in their community, and how their information is being used to addressing knowledge gaps in the literature (e.g., sharing condensed research reports or publications) is another effective strategy. Other recommendations include diverse, personalized recruitment and retention practices (e.g., face-to-face presentations, printed materials, traditional and social media broadcasts), providing support for transportation costs if travel is involved, building relationships with leaders and influential individuals associated with frequently used venues (e.g., religious organizations, barbershops, salons, local chapters of fraternities and sororities), and increasing involvement and visibility within a community to build familiarity (Graves & Sheldon, 2018; Ibrahim & Sidani, 2014).

Next, by eliminating barriers to participation in research and building genuine, mutually beneficial relationships with study populations, it may facilitate the use of more robust study designs with larger, more generalizable study samples. For example, being able to identify future parents early in their lives before the conception of their future children and following them longitudinally can provide richer data than methods relying primarily on cross-sectional, retrospective data collection. This long-term research approach can also be applied to offspring in a way that can help capture their health outcomes at birth, during childhood, and adulthood, and their self-reported adversity experiences. Furthermore, utilizing an array of measures (e.g., biological data, in-depth interviews, surveys) simultaneously can help characterize adversity and

its intergenerational health impact on multiple levels (e.g., biological, psychological, social), as well as how the various levels interact with each other. Another important area of focus might be to better understand the mechanisms that are responsible for the transmission of parental adversity and how they shape child health outcomes. Although some research has measured and identified potential mechanisms (Hilmert et al. 2014; Jones et al., 2019; Smith et al., 2016), these studies have not focused specifically on AA families, addressed the unique adversity experiences they encounter, or attempted to explain how a certain adversity type may activate mechanisms in relation to other types. Finally, including more than two generations of participants from the same family may also contribute to a clearer picture of how these associations operate.

References

Anderson, L. M., Riffle, L., Wilson, R., Travlos, G. S., Lubomirski, M. S., & Alvord, W. G. (2006). Preconceptional fasting of fathers alters serum glucose in offspring of mice. *Nutrition, 22*, 327-331. doi: 10.1016/j.nut.2005.09.006

Braun, J. M., Messerlian, C., & Hauser, R. (2017). Fathers matter: Why it's time to consider the impact of paternal environmental exposures on children's health. *Current Epidemiology Reports, 4(1)*, 46-55. doi:10.1007/s40471-017-0098-8

Cammack, A. L., Hogue, C. J., Drews-Botsch, C. D., Kramer, M. R., & Pearce, B. D. (2019). Associations between maternal exposure to child abuse, preterm birth, and very preterm birth in young, nulliparous women. *Maternal and Child Health Journal, 23(6)*, 847-857. doi:10.1007/s10995-018-02709-w

Carlos-Wallace, F. M., Zhang, L., Smith, M. T., Rader, G., & Steinmaus, C. (2015). Parental, in utero, and early-life exposure to benzene and the risk of childhood leukemia: A meta-analysis. *American Journal of Epidemiology*, *183*, 1-14. doi: 10.1093/aje/kwv120

Carone, B. R., Fauquier, L., Habib, N., Shea, J. M., Hart, C. E., Li, R., Bock, C., Li, C., Gu, H., Zamore, P. D., Meissner, A., Weng, Z., Hofmann, H. A., Friedman, N., & Rando, O. J. (2010). Paternally induced transgenerational environmental reprogramming of metabolic gene expression in mammals. *Cell*, *143*, 1084-1096. doi: 10.1016/j.cell.2010.12.008

Coker, A. D., Huang, H. H., & Kashubeck-West, S. (2009). Research with African Americans: Lessons learned about recruiting African American women. *Journal of Multicultural Counseling and Development, 37(3)*, 153-165. doi: 10.1002/j.2161-1912.2009.tb00099.x

Day, J., Savani, S., Krempley, B. D., Nguyen, M., & Kitlinska, J. B. (2016). Influence of

paternal preconception exposures on their offspring: Through epigenetics to phenotype. *American Journal of Stem Cells, 5,* 11-18.

De Roos, A. J., Olshan, A. F., Teschke, K., Poole, C., Savitz, D. A., Blatt, J., Bondy, M. L., & Pollock, B. H. (2001). Parental occupational exposures to chemicals and incidence of neuroblastoma in offspring. *American Journal of Epidemiology, 154,* 106-114. doi: 10.1093/aje/154.2.106

Dias, B. G., & Ressler, K. J. (2014). Parental olfactory experience influences behavior and neural structure in subsequent generations. *Nature Neuroscience, 17,* 89-96. doi: 10.1038/nn.3594

Fullston, T., McPherson, N. O., Owens, J. A., Kang, W. X., Sandeman, L. Y., & Lane, M. (2015). Paternal obesity induces metabolic and sperm disturbances in male offspring that are exacerbated by their exposure to an "obesogenic" diet. *Physiological Reports, 3,* e12336. doi: 10.14814/phy2.12336

Gapp, K., von Ziegler, L., Tweedie-Cullen, R. Y., & Mansuy, I. M. (2014). Early life epigenetic programming and transmission of stress-induced traits in mammals: How and when can environmental factors influence traits and their transgenerational inheritance? *Bioessays, 36,* 491-502. doi: 10.1002/bies.201300116

George, S., Duran, N., & Norris, K. (2014). A systematic review of barriers and facilitators to minority research participation among African Americans, Latinos, Asian Americans, and Pacific Islanders. *American Journal of Public Health, 104(2),* e16-e31. doi: 10.2105/AJPH.2013. 301706

Graves, D., & Sheldon, J. P. (2018). Recruiting African American children for research: An

ecological systems theory approach. *Western Journal of Nursing Research, 40(10),* 1489-1521. doi: 10.1177/0193945917704856

Hilmert, C. J., Dominguez, T. P., Dunkel Schetter, C., Srinivas, S. K., Glynn, L. M., Hobel, C. J., & Sandman, C. A. (2014). Lifetime racism and blood pressure changes during pregnancy: Implications for fetal growth. *Health Psychology, 33(1),* 43-51. doi:10.1037/a0031160

Huang, H. H., & Coker, A. D. (2010). Examining issues affecting African American participation in research studies. *Journal of Black Studies, 40(4),* 619-636. doi: 10.1177/0021934708317749

Ibrahim, S., & Sidani, S. (2014). Strategies to recruit minority persons: A systematic review. *Journal of Immigrant and Minority Health, 16(5),* 882-888. doi: 10.1007/s10903-013-9783-y

Jones, C. W., Esteves, K. C., Gray, S. A., Clarke, T. N., Callerame, K., Theall, K. P., & Drury, S. S. (2019). The transgenerational transmission of maternal adverse childhood experiences (ACEs): Insights from placental aging and infant autonomic nervous system reactivity. *Psychoneuroendocrinology, 106,* 20-27. doi:10.1016/j.psyneuen.2019.03.022

Kaati, G., Bygren, L. O., Pembrey, M., & Sjöström, M. (2007). Transgenerational response to nutrition, early life circumstances and longevity. *European Journal of Human Genetics, 15,* 784-790. doi: 10.1038/sj.ejhg.5201832

Kaati, G., Bygren, L. O., & Edvinsson, S. (2002). Cardiovascular and diabetes mortality determined by nutrition during parents' and grandparents' slow growth period. *European Journal of Human Genetics, 10,* 682-688. doi: 10.1038/sj.ejhg.5200859

Morales-Suárez-Varela, M. M., Toft, G. V., Jensen, M. S., Ramlau-Hansen, C., Kaerlev, L.,

Thulstrup, A. M., Llopis-González, A., Olsen, J., & Bonde, J. P. (2011). Parental occupational exposure to endocrine disrupting chemicals and male genital malformations: A study in the Danish National Birth Cohort study. *Environmental Health, 10*, 3. doi: 10.1186/1476-069X-10-3

Nassar, N., Abeywardana, P., Barker, A., & Bower, C. (2010). Parental occupational exposure to potential endocrine disrupting chemicals and risk of hypospadias in infants. *Occupational and Environmental Medicine, 67*, 585-589. doi: 10.1136/oem.2009.048272

Ng, S. F., Lin, R. C., Laybutt, D. R., Barres, R., Owens, J. A., & Morris, M. J. (2010). Chronic high-fat diet in fathers programs β-cell dysfunction in female rat offspring. *Nature, 467*, 963-966. doi: 10.1038/nature09491

Pembrey, M. E., Bygren, L. O., Kaati, G., Edvinsson, S., Northstone, K., Sjöström, M., & Golding, J. (2006). Sex-specific, male-line transgenerational responses in humans. *European Journal of Human Genetics, 14*, 159-166. doi: 10.1038/sj.ejhg.5201538

Rocheleau, C. M., Romitti, P. A., & Dennis, L. K. (2009). Pesticides and hypospadias: A meta-analysis. *Journal of Pediatric Urology, 5*, 17-24. doi: 10.1016/j.jpurol.2008.08.006

Rodgers, A. B., Morgan, C. P., Leu, N. A., & Bale, T. L. (2015). Transgenerational epigenetic programming via sperm microRNA recapitulates effects of paternal stress. *Proceedings of the National Academy of Sciences, 112*, 13699-13704. doi: 10.1073/pnas.1508347112

Rodgers, A. B., Morgan, C. P., Bronson, S. L., Revello, S., & Bale, T. L. (2013). Paternal stress exposure alters sperm microRNA content and reprograms offspring HPA stress axis regulation. *Journal of Neuroscience, 33*, 9003-9012. doi: 10.1523/JNEUROSCI.0914-13.2013

Sharma, U., Conine, C. C., Shea, J. M., Boskovic, A., Derr, A. G., Bing, X. Y., Belleannee, C.,

Kucukural, A., Serra, R. W., Sun, F., Song, L., Carone, B. R., Ricci, E. P., Li, X. Z., Fauquier, L., Moore, M. J., Sullivan, R., Mello, C. C., Garber, M., & Rando, O. J. (2016). Biogenesis and function of tRNA fragments during sperm maturation and fertilization in mammals. *Science, 351*, 391-396. doi: 10.1126/science.aad6780

Smith, M. V., Gotman, N., & Yonkers, K. A. (2016). Early childhood adversity and pregnancy outcomes. *Maternal and Child Health Journal, 20(4)*, 790-798. doi:10.1007/s10995-015-1909-5

Soubry, A., Schildkraut, J. M., Murtha, A., Wang, F., Huang, Z., Bernal, A., Kurtzberg, J., Jirtle, R. L., Murphy, S. K., & Hoyo, C. (2013). Paternal obesity is associated with IGF2 hypomethylation in newborns: Results from a Newborn Epigenetics Study (NEST) cohort. *BMC Medicine, 11*, 29. doi: 10.1186/1741-7015-11-29

Van Wijngaarden, E., Stewart, P. A., Olshan, A. F., Savitz, D. A., & Bunin, G. R. (2003). Parental occupational exposure to pesticides and childhood brain cancer. *American Journal of Epidemiology, 157*, 989-997. doi: 10.1093/aje/kwg082

Wallace, D. C., & Bartlett, R. (2013). Recruitment and retention of African American and Hispanic girls and women in research. *Public Health Nursing, 30(2)*, 159-166. doi: 10.1111/phn.12014

Wei, Y., Yang, C. R., Wei, Y. P., Zhao, Z. A., Hou, Y., Schatten, H., & Sun, Q. Y. (2014). Paternally induced transgenerational inheritance of susceptibility to diabetes in mammals. *Proceedings of the National Academy of Sciences, 111*, 1873-1878. doi: 10.1073/pnas.1321195111

Appendix A: Keyword Search Terms

Race-terms- "african american" OR "african americans" OR "African Americans"[Mesh] OR blacks[tiab] OR afro-american OR afro american OR afro OR "black people" OR "People of Color" OR negro* OR "African Continental Ancestry Group"[Mesh] OR colored* OR "Race Factors"[Mesh] OR race[ti] OR races[ti] OR racial[ti] OR racially[ti] OR "Ethnic Groups"[Majr] OR ethnic[ti] OR ethnical*[ti]

Intergenerational-related terms- generation OR generations OR generational OR intergenerational OR inter-generational OR transgenerational OR trans-generational OR multigenerational OR multi-generational OR intergenerationality OR transmit OR transmitting OR transmission OR "Parents"[Mesh:NoExp] OR "Fathers"[Mesh] OR "Mothers"[Mesh] OR "Single Parent"[Mesh] OR maternal OR paternal OR mother OR father OR parental[tw] OR "Grandparents"[Mesh] OR grandparent* OR grand-parent* OR grandmother* OR grandfather* OR grandchild* OR granddaughter* OR grandson* OR grand-mother* OR grand-father* OR grand-child* OR grand-daughter* OR grand-son* OR child OR children OR childhood OR infant OR infants OR "Child"[Mesh] OR "Infant"[Mesh] OR "Adolescent"[Mesh] OR offspring* OR neighborhood* OR neighbourhood* OR "family history" OR "family medical history" OR "family histories" OR "family medical histories" OR fetal OR fetus OR preconception OR pediatric OR paediatric OR newborn* OR "Infant, Newborn"[Mesh] OR unborn

Stress and trauma-related terms- "Gene-Environment Interaction"[Majr] OR "social discrimination"[Majr] OR "Social Segregation"[Majr] OR "Socioeconomic Factors"[Majr] OR "Stress Disorders, Traumatic"[Majr] OR "Stress, Physiological"[Majr:NoExp] OR "Stress, Psychological"[Majr] OR "Violence"[Majr] OR "Warfare"[Majr] OR "Working Poor"[Majr] OR "Wounds and Injuries"[Majr] OR abuse[ti] OR abused[ti] OR accident[ti] OR accidental[ti] OR

accidents[ti] OR adverse[ti] OR adversity[ti] OR aggress[ti] OR aggression[ti] OR aggressive[ti] OR altercation[ti] OR assault[ti] OR attack[ti] OR attacks[ti] OR attacked[ti] OR attacking[ti] OR bereavement[ti] OR bully[ti] OR burden*[ti] OR childhood maltreatment[ti] OR coerce*[ti] OR coercive[ti] OR danger*[ti] OR death[ti] OR deprivation[ti] OR deprived[ti] OR destruct[ti] OR destructing[ti] OR destructive[ti] OR disadvantage*[ti] OR disaster[ti] OR discriminate[ti] OR discrimination[ti] OR discriminatory[ti] OR dislocation[ti] OR economic environment[ti] OR economic[ti] OR economically[ti] OR economics[ti] OR exploit[ti] OR exploitation[ti] OR exploited[ti] OR exposure[ti] OR exposures[ti] OR financial[ti] OR frighten[ti] OR gene-environment*[ti] OR genocidal[ti] OR genocide[ti] OR grief[ti] OR grieving[ti] OR hardship*[ti] OR harm[ti] OR harmed[ti] OR harmful[ti] OR harmfulness[ti] OR harming[ti] OR harms[ti] OR homeless[ti] OR homicide[ti] OR humiliate[ti] OR humiliated[ti] OR humiliation[ti] OR incarcerate[ti] OR incarceration[ti] OR intimidate[ti] OR intimidating[ti] OR lose[ti] OR loss[ti] OR losses[ti] OR lost[ti] OR maltreatment[ti] OR neglect[ti] OR neglected[ti] OR neglectful[ti] OR neglecting[ti] OR pain[ti] OR poor[ti] OR poverty[ti] OR prison[ti] OR punishment[ti] OR punishments[ti] OR racism[ti] OR rape[ti] OR relocation[ti] OR segregat*[ti] OR shooting[ti] OR shot[ti] OR slavery[ti] OR socio-economic status[ti] OR socioeconomic status[ti] OR stress[ti] OR stressed[ti] OR stressor[ti] OR terror[ti] OR terrorized[ti] OR terrors[ti] OR threat[ti] OR threatening[ti] OR threats[ti] OR trauma[ti] OR traumas[ti] OR traumatic[ti] OR traumatisation[ti] OR traumatised[ti] OR traumatization[ti] OR traumatized[ti] OR violence[ti] OR violent[ti] OR war[ti] OR worried[ti] OR worries[ti] OR worry[ti] OR wound[ti] OR wounded[ti] OR wounds[ti]

Physical health terms- "acute disease"[ti] OR "Acute Disease"[Majr] OR "acute diseases"[ti] OR "Allostasis"[Mesh] OR "Arthritis"[Majr] OR "birth outcome"[ti] OR "birth outcomes"[ti]

OR "birth weight"[ti] OR "blood pressure"[ti] OR "Blood Pressure"[Mesh] OR "Bronchial Diseases"[Majr] OR "Cardiovascular Diseases"[Majr] OR "chronic disease"[ti] OR "Chronic Disease"[Majr] OR "chronic diseases"[ti] OR "Diabetes Mellitus"[Majr] OR "Fatigue"[Majr:NoExp] OR "Female Urogenital Diseases"[Majr] OR "Fetal Death"[Majr] OR "Fetal Mortality"[Majr] OR "Gastrointestinal Diseases"[Majr] OR "Headache"[Majr:NoExp] OR "health care" disparities[ti] OR "health care" disparity[ti] OR "health care" inequalities[ti] OR "health care" inequality[ti] OR "health disparities"[ti] OR "health disparity"[ti] OR "health outcome"[ti] OR "health outcomes"[ti] OR "health status"[ti] OR "heart failure"[ti] OR "heart murmur"[ti] OR "heart murmurs"[ti] OR "Hemophilia A"[Majr] OR "Hemophilia B"[Majr] OR "Hypersensitivity"[Majr] OR "Hypertension"[Majr] OR "Infant Mortality"[Majr] OR "Infection"[Majr] OR "Liver Cirrhosis"[Majr] OR "Male Urogenital Diseases"[Majr] OR "Migraine Disorders"[Majr] OR "minorities health"[ti] OR "minority health"[ti] OR "Minority Health"[Majr] OR "Morbidity"[Majr] OR "Mortality"[Majr] OR "multiple sclerosis"[ti] OR "Musculoskeletal Diseases"[Majr] OR "Neoplasms"[Majr] OR "Nervous System Diseases"[Majr] OR "Neuroanatomy"[Majr] OR "Neurochemistry"[Majr] OR "Neuropathology"[Majr] OR "Obesity"[Majr] OR "Outcome Assessment (Health Care)"[Majr:NoExp] OR "outcome assessment"[ti] OR "Pain"[Majr] OR "peptic ulcer"[ti] OR "peptic ulcers"[ti] OR "Perinatal Death"[Majr] OR "Peripartum Period"[Majr] OR "physical ailment"[ti] OR "physical ailments"[ti] OR "physical health"[ti] OR "physical outcome"[ti] OR "physical outcomes"[ti] OR "Postpartum Period"[Majr] OR "Pregnancy"[Majr] OR "Premature Birth"[Majr] OR "Pulmonary Emphysema"[Majr] OR "Respiration Disorders"[Majr] OR "Risk"[Majr] OR "Seizures"[Majr] OR "Signs and Symptoms, Respiratory"[Majr] OR "Social Determinants of health"[Majr] OR "Stroke"[Majr] OR allergic[ti] OR allergies[ti] OR allergy[ti]

OR allostatic[ti] OR ante-natal*[ti] OR ante-partum[ti] OR antenatal*[ti] OR antepartum[ti] OR arrhythmia[ti] OR arthritis[ti] OR asthma*[ti] OR birth-weight*[ti] OR birthweight*[ti] OR blood[ti] OR bowel[ti] OR bronchitis[ti] OR cancer[ti] OR cardiac[ti] OR cardio[ti] OR cardiovascular[ti] OR circulatory[ti] OR cirrhosis[ti] OR colitis[ti] OR diabetes[ti] OR emphysema[ti] OR endocrine[ti] OR epigenetic*[ti] OR epigenomic[ti] OR epigenomics[ti] OR fatigue*[ti] OR gastrointestinal[ti] OR genitourin*[ti] OR headache*[ti] OR healthcare disparities[ti] OR healthcare disparity[ti] OR healthcare inequalities[ti] OR healthcare inequality[ti] OR hemophilia[ti] OR hypertension[ti] OR immune[ti] OR incongruit* OR infection[ti] OR infections[ti] OR infectious[ti] OR inflammatory[ti] OR intrauterine[ti] OR migraine*[ti] OR morbidities[ti] OR morbidity[ti] OR mortalities[ti] OR mortality[ti] OR musculoskeletal[ti] OR myocardial[ti] OR neonatal[ti] OR neuroanatomy[ti] OR neurologic*[ti] OR neurological[ti] OR neuropathology[ti] OR obesity[ti] OR pain[ti] OR palpitation*[ti] OR perinatal*[ti] OR peri-natal*[ti] OR pregnan*[ti] OR premature[ti] OR pre-mature[ti] OR prenatal[ti] OR pre-natal[ti] OR preterm[ti] OR pre-term OR reproduc*[ti] OR reproduction[ti] OR reproductive[ti] OR respiration[ti] OR respiratory[ti] OR risks[ti] OR risky[ti] OR seizure*[ti] OR still-birth*[ti] OR still-born*[ti] OR stillbirth*[ti] OR stillborn*[ti] OR stroke[ti] OR strokes[ti] OR tumor*[ti] OR tumour*[ti] OR ulcer[ti] OR ulcers[ti] OR weathering OR well-being[ti] OR well-ness[ti] OR wellbeing[ti] OR wellness[ti] OR wheez*[ti] OR "metabolic syndrome"[ti] OR "Metabolic Syndrome"[Majr] OR "high cholesterol"[ti] OR "Hypercholesterolemia"[Majr] OR hyperlipidemia[ti] OR "Hyperlipidemias"[Majr] OR "irritable bowel syndrome"[ti] OR "Irritable Bowel Syndrome"[Majr] OR "inflammatory bowel disease"[ti] OR "Inflammatory Bowel Diseases"[Mesh] OR "crohn's disease"[ti] OR autoimmune[ti] OR "Autoimmune Diseases"[Majr]

Appendix B: Articles with Parental Adversity Measured Clearly Before Pregnancy (Full AA Sample)

Study & Sample	Key Measures	Key Results	Risk of Bias (ROB)
Study: Gillespie et al., 2017 Sample: 96 pregnant African-American women and their infants Design: Prospective cohort	Predictor: Cumulative maternal childhood stress measured using STRAIN Outcomes: Infant birth timing; Infant birth following spontaneous labor Mediator: Prenatal maternal plasma cortisol	- ↑ Maternal cumulative childhood stress → earlier birth timing (controls: adult stress, cortisol) - Maternal cortisol mediated link between childhood stress and earlier birth timing in women who had spontaneous labor Mechanism of Transmission: Childhood stress alters birth outcomes through prenatal maternal cortisol	ROB: Moderate; primary source of bias was non-representative sample; maternal preconception adversity assessed retrospectively
Study: Hilmert et al., 2014 Sample: 39 pregnant African-American women and their infants Design: Retrospective longitudinal cohort	Predictor: Maternal lifetime racism Outcomes: Infant BW and GA via medical charts Moderators: Maternal prenatal SBP and DBP	- 2+ domains of maternal exposure to indirect racism in childhood → ↓BW as mom's prenatal DBP↑ (controls: BMI, SES, and SLEI) Mechanisms of Transmission: Maternal racism exposure affects birth outcomes through prenatal BP	ROB: Moderate; primary source of bias was non-representative sample used; maternal preconception adversity assessed retrospectively
Study: Jovanovic et al., 2011 Sample: 36 African-American children 6-13 years old and their mothers	Predictors: Maternal exposure to perceived childhood emotional, physical, and sexual abuse using CTQ	- Maternal physical abuse → ↑child dark-enhanced startle (controls: child age, sex) - ↑ vs. ↓Maternal emotional abuse → ↑child LF/HF ratios	ROB: High; primary sources of bias were small, non-representative sample and participants' response rate not reported; maternal preconception adversity assessed retrospectively

Design: Retrospective cross-sectional	Outcomes: Child startle response; Child HRV acquired via electrocardiogram		
Study: Rowell, 2020 Sample: 31 expectant African-American mothers Design: Retrospective cross-sectional	Predictor: Maternal ACEs using ACEs Questionnaire Outcomes: Infant BW and GA as reported by doulas at birth	- Maternal ACEs not significantly associated with GA or BW (controls: maternal age)	ROB: High; primary sources of bias were small, non-representative sample and participants' response rate not reported; maternal preconception adversity assessed retrospectively
Study: Sealy-Jefferson et al., 2019 Sample: 1365 African-American women and their infants Design: Retrospective cohort	Predictor: Current PS during past month using PSS Outcome: Infant PTB Moderators: Early-life neighborhood social disorder; Early-life neighborhood social control	- ↑Early-life neighborhood social disorder and ↑current stress → ↑odds of PTB relative to ↓early-life social disorder (controls: maternal age, marital status, education, income) - ↓early-life neighborhood social disorder → No association between PS and PTB	ROB: Moderate; primary source of bias was non-representative sample used; maternal preconception adversity assessed retrospectively

Appendix C: Articles with Parental Adversity Measured Clearly Before Pregnancy (Partial AA Sample Testing for Racial Differences)

Study & Sample	Key Measures	Key Results	Risk of Bias (ROB)
Study: Dominguez et al., 2008 Sample: 124 pregnant women (41.1% African American) and their infants Design: Prospective longitudinal cohort	Predictor: Race using self-identification as "African American or Black" or "Non-Hispanic White" Outcomes: Infant BW and GA using medical charts Mediators: Maternal childhood and adulthood direct/vicarious exposure to racism	- Mom's lifetime and vicarious childhood exposure to racism →↓BW for Black moms only (controls: parents' childhood education) - No group differences in GA	ROB: Moderate; primary source of bias was the non-representative sample; maternal preconception adversity assessed retrospectively
Study: Gray et al., 2017 Sample: 167 infants (49% female) and their mothers (61% African American) Design: Retrospective cohort	Predictor: Maternal exposure to ACEs using ACEs survey Outcomes: 4-mo old infant RSA measured during dyadic play and dyadic completion of the SFP Moderators: Infant sex and race using maternal report	- ↑Maternal ACEs → ↓infant RSA during dyadic play (controls: infant sex, race) - ↑Maternal ACEs → ↓infant RSA during SFP - No significant sex or race differences found in infant RSA	ROB: High; primary sources of bias were lack of information about participants' follow-up rate and the non-representative sample; maternal preconception adversity assessed retrospectively
Study: Margerison-Zilko et al., 2017 Sample: 2,559 women (21% African American) and their infants Design: Retrospective cohort	Predictors: Maternal childhood, adulthood SLE using Turner, Wheaton, and Lloyd Checklist; events scored as never, in childhood, in adulthood, or both Outcomes: Infant early and late PTB Moderator: Maternal race/ethnicity	- Maternal abuse/violence in childhood only vs. never → ↑late PTB (controls: race/ethnicity, education, parity, marital status) - Race/ethnicity did not moderate the association between SLE and PTB or PTB by timing	ROB: Moderate; primary source of bias was the non-representative sample; maternal preconception adversity assessed retrospectively

Study: Masho et al., 2015 Sample: 231 pregnant women (72% African American) and their infants Design: Retrospective cohort	Predictors: Maternal lifetime and past year SLE using SLEI; Maternal PS in her life, past year, and last month using PSS; Maternal prenatal cortisol via saliva samples Outcome: Infant PTB	- Lifetime exposure to SLE was not associated with PTB in either the AA subsample or the full sample (controls: maternal age, education, adequacy of prenatal care)	ROB: High; primary sources of bias were participants' follow-up rate not reported and non-representative sample; maternal preconception adversity assessed retrospectively
Study: Seng et al., 2011 Sample: 839 women (41.4% African American) and their infants Design: Prospective longitudinal cohort	Predictor: Current and lifetime maternal PTSD diagnoses using National Women's Study PTSD Module Outcomes: Infant BW and GA Moderator: Maternal childhood abuse using LSC	- Maternal childhood abuse not related to infant BW or GA (controls: comorbidity, risk behaviors, medical and obstetric risk factors, modifiable health care related factors, chronic stress) - Among women who experienced child abuse, African-American race was the strongest predictor of LBW	ROB: High; primary sources of bias were the non-representative sample and inadequate % of participants retained at follow up; maternal preconception adversity assessed retrospectively

Appendix D: Articles with Parental Adversity Measured Clearly Before Pregnancy (Partial AA Sample Not Testing Racial Differences)

Study & Sample	Key Measures	Key Results	Risk of Bias (ROB)
Study: Blackmore et al., 2016 Sample: 358 pregnant women (49.7% African American) and their infants Design: Prospective cohort	Predictors: Symptoms of maternal depression and anxiety using EPDS and PSWQ, respectively Outcomes: Infant BW and GA Moderator: Traumatic events exposure using PTSD section of the SCID	- ↑Anxiety among women who experienced childhood trauma → ↓BW (controls: maternal ethnicity, BMI, prenatal alcohol use and smoking, pregnancy history, SES) - Maternal trauma, depression, and anxiety were not linked to GA	ROB: Moderate; primary source of bias was the non-representative sample; maternal preconception adversity assessed retrospectively
Study: Chen et al., 2017 Sample: 150 children aged 9 to 17 years (25% African American) with physician diagnosed asthma and a parent Design: Retrospective cross-sectional	Predictor: Parents' childhood SES using early childhood home ownership (renting=↓SES; owning=↑SES) Outcomes: ACT completed by children and parents; child T_H2 and T_H1 cytokine production	- ↓ parental childhood SES → ↓child asthma control vs. ↑ parental childhood SES (controls: child age, sex, ethnicity, usage of beta agonists and inhaled corticosteroids) - ↓Parental childhood SES → ↑child T_H2 and T_H1 cytokine production vs. offspring of ↑parental SES	ROB: Moderate; primary source of bias was the non-representative sample; maternal preconception adversity assessed retrospectively
Study: Cheng et al., 2016 Sample: 6,900 children and their mothers (14.3% African American) Design: Retrospective cohort	Predictor: Maternal PSLEs Outcomes: VLBW infant; Maternal reported infant/toddler health at 9 and 24 months including overall health status, clinically diagnosed SHCN, and any severe health condition	- ↑Maternal PSLEs → ↑odds VLBW infant (controls: maternal chronic conditions, # of children, parity, age, race/ethnicity, marital status, insurance status, SES, region, pregnancy complications, pre-pregnancy BMI, initiation of prenatal care)	ROB: Low; primary source of bias was maternal preconception adversity assessed retrospectively

		- ↑Maternal PSLEs → poorer child health status, ↑odds SHCN, and ↑severe health conditions at 9 mos.	
Study: Cowell et al., 2021 Sample: 829 mother-newborn pairs (45% African American) Design: Prospective cohort	Predictors: Maternal childhood IPT using CTQ; Maternal lifetime trauma and non-traumatic stress in prior year using LSC-R; Maternal trait anger expression using STAXI-2 subscales Outcome: Infant GA	- Maternal childhood IPT not associated with PTB risk (controls: maternal ethnicity, age, parity, relationship status, education level, prenatal smoking, pre-pregnancy BMI)	ROB: Moderate; primary source of bias was the non-representative sample; maternal preconception adversity assessed retrospectively
Study: Freedman et al., 2017 Sample: 133 women experiencing stillbirth (17% African American) and 500 women delivering a healthy term live birth (12% African American) Design: Case-control	Predictors: Maternal childhood maltreatment using CTQ with physical abuse, CSA, emotional abuse, physical neglect, and emotional neglect subscales Outcome: Infant stillbirth status gathered from medical records	- Maternal childhood emotional neglect → ↑infant stillbirth risk (controls: maternal age, education) - No other forms of maternal maltreatment significantly associated with stillbirth risk	ROB: Moderate; primary source of bias was the different participant response rates in the case and control groups; maternal preconception adversity assessed retrospectively

Study: Jones et al., 2019 Sample: 67 pregnant mothers (56.7% African American) and their four-month-old infants (43.3% female) Design: Prospective cohort	Predictor: Maternal ACEs using ACEs Questionnaire Outcome: Infant RSA stress responsivity Moderator: Placental TL	- ↑Maternal ACEs → ↑infant RSA stress responsivity (controls: sex, race, maternal prenatal smoking) - ↑Maternal ACEs → shorter placental TL - Placental TL and maternal ACEs interacted to predict both infant RSA reactivity and recovery Mechanism of Transmission: Maternal ACEs → changes in placental TL → ↑infant stress responsivity	ROB: High; primary sources of bias were the lack of information about participants' follow-up rate and non-representative sample; maternal preconception adversity assessed retrospectively
Study: Mersky et al., 2019 Sample: 1848 women (24% African American) with children Design: Retrospective cohort	Predictor: Maternal ACEs using CES with responses summed & categorized (0, 1-2, 3-4, and 5+ ACEs) Outcomes: Pregnancy loss (e.g., miscarriage or still birth), PTB, and LBW using archival program records	- ↑Maternal ACEs → ↑odds of pregnancy loss, PTB, and LBW (controls: maternal age, race/ ethnicity, educational attainment) - 5+ ACEs → ↑odds of pregnancy loss; no differences in PTB or LBW odds - No differences in birth outcomes between mothers with 0 ACEs and those with 1-2 or 3-4 ACEs	ROB: High; primary sources of bias were the lack of information about participants' response rate and non-representative sample; maternal preconception adversity assessed retrospectively
Study: Miller et al., 2017	Predictor: Maternal childhood economic hardship	- ↑Maternal childhood disadvantage → ↑odds of adverse birth outcomes (controls: age,	ROB: Moderate; primary source of bias was non-representative sample;

Sample: 744 pregnant women (16.3% African American) and their infants Design: Prospective cohort	Outcomes: Infant birth outcomes using medical charts including length of gestation (e.g., PTB), fetal growth (e.g., BW; SGA), length of hospital stay, and SCN Mediators: Maternal inflammatory biomarkers (e.g., IL-6, IL-8); Psychosocial pathways (e.g., maternal education); Lifestyle pathways (e.g., pre-pregnancy BMI); Obstetric pathways (e.g., history of PTB)	race/ethnicity, nulliparity, gestational hypertension, pre-eclampsia, PTB history) - Maternal childhood disadvantage → ↑pre-pregnancy BMI → ↑Maternal IL-6 levels → adverse birth outcomes - Maternal childhood disadvantage → ↓Maternal education & ↑pre-pregnancy BMI → adverse birth outcomes Mechanisms of Transmission: Maternal disadvantage → maternal inflammatory, psychosocial, lifestyle, and obstetric factors → adverse birth outcomes	maternal preconception adversity assessed retrospectively

Study: Noll et al., 2007 Sample: 67 offspring (~49% African American) born to mothers who experienced CSA and 56 offspring (46% African American) born to nonabused comparison mothers Design: Prospective cohort	Predictor: Maternal CSA determined by records of substantiated contact CSA from CPS agencies Outcome: Infant PTB using hospital records Mediators: Maternal salivary cortisol; Maternal prenatal alcohol use reported in labor and delivery records	- Maternal CSA → ↑odds of PTB status (controls: minority status, sibling number) - Maternal prenatal alcohol use partially mediated link between maternal CSA and PTB Mechanisms of Transmission: Maternal prenatal alcohol use partially mediated link between maternal CSA and PTB status	ROB: Moderate; primary source of bias was non-representative sample
Study: Smith et al., 2016 Sample: 2303 pregnant women (7% African American) and their infants Design: Prospective cohort	Predictor: Maternal ACEs using ETI-SF Outcomes: PTB and LBW Mediators: Maternal prenatal smoking and substance use via interview	- ↑Maternal ACEs →↑LBW and ↓GA (controls: maternal marital status, prenatal illicit substance use and alcohol use, SRI use, psychiatric disorder, education, smoking, social support) Mechanisms of Transmission: Maternal prenatal smoking and substance use mediated impact of ACEs on BW; Prenatal smoking was the strongest mediator of the impact of ACEs on GA	ROB: Moderate; primary source of bias was non-representative sample; maternal preconception adversity assessed retrospectively

Study: Sternthal et al., 2011 Sample: 510 pregnant women (28% African American) and their infants Design: Prospective cohort	Predictor: Maternal childhood SES using parental home ownership from birth to age 10 Outcomes: Child cord blood IgE levels (IU/mL) using CAP fluorescent enzyme immunoassay; Maternal report of infant wheezing at 2 years old Mediators: Social pathways (e.g., maternal IPT exposure); Physical pathways (e.g., prenatal household allergens)	- ↓Maternal childhood SES → ↑cord blood IgE levels (controls: child sex, maternal race/ethnicity, atopy, nativity status) - No mediators linking maternal childhood SES and cord blood IgE - Maternal lifetime IPT → ↑cord blood IgE; maternal childhood SES not related to maternal IPT - Significant indirect effects linking low maternal childhood SES and child wheeze via adult SES and prenatal environmental exposures	ROB: Moderate; primary source of bias was non-representative sample; maternal preconception adversity assessed retrospectively
Study: Witt et al., 2014a Sample: 9,350 children and their mothers (14.1% African American) Design: Retrospective cohort	Predictor: Maternal PSLEs Outcomes: Infant LBW and VLBW	- Any maternal PSLEs vs. no PSLEs → ↑odds VLBW infant (controls: see Cheng et al. 2016) - Maternal PSLEs not linked w/LBW - Timing of PSLEs exposure affected associations such that PSLEs ≥ 1 year pre-conception → ↑odds of VLBW baby	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity

Study: Witt et al., 2014b Sample: 9,350 children and their mothers (14.1% African American) Design: Retrospective cohort	Predictor: Maternal PSLEs Outcome: Infant PTB Moderator: Maternal age	- Maternal PSLEs and age interacted to predict PTB: younger women with PSLE → ↑ PTB risk vs. older women (controls: see Cheng et al. 2016) - Women aged 20-24 or 30 years or older exposed to PSLEs 1 year or more prior to conception had ↑ PTB risk than women aged 25-29 years without such an event	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity
Study: Witt et al., 2015 Sample: 9,300 children and their mothers (14% African American) Design: Retrospective cohort	Predictor: Maternal PSLEs Outcome: Infant LBW and VLBW Moderator: Maternal neighborhood disadvantage	-↑Maternal PSLEs → ↑risk of VLBW (controls: see Cheng et al. 2016)	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity
Study: Witt et al., 2016 Sample: 9,350 children and their mothers (14.1% African American) Design: Retrospective cohort	Predictor: Maternal PSLEs Outcome: Infant LBW and VLBW	- ↑Maternal PSLEs →↑risk VLBW vs. no PSLEs (controls: see Cheng et al. 2016) - PSLE exposure → ↑ risk LBW	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity

Appendix E: Articles with Parent-Reported Offspring Health Outcomes

Study & Sample	Key Measures	Key Results	Risk of Bias (ROB)
Study: Astone et al., 2007 Sample: 987 infant (G3), mother (G2), and grandmother (G1) groups (82.5% African American) Design: Prospective cohort	Predictors: Grandmother's (G1) education; Maternal (G2) childhood household income; G2 family structure; G2's household receipt of public assistance at birth or age 7 Outcome: Maternal reported infant BW (G3)	- If mother was poor → ↑risk LBW (controls: G3 sex, G2 adult height, multipara, prenatal smoking, difference between G2 BW & G1 BW, G1 pre-pregnancy BMI, infant BWs, STDs, prenatal smoking) - ↑ income/needs ratio → ↑ BW	ROB: Moderate; primary source of bias was non-representative sample; maternal report of offspring health
Study: Brunst et al., 2017 Sample: 857 pregnant women (30% African American) and their infants Design: Prospective cohort	Predictors: Maternal lifetime IPT using R- CTS: unexposed, child/adolescent IPT, adult/index pregnancy IPT, or chronic IPT Outcome: Maternal report of MD-diagnosed asthma from birth up to age six years Mediator: Maternal prenatal asthma	- Chronic maternal IPT vs. no IPT → ↑male child asthma risk, (controls: maternal age, education, child sex & birthweight, race/ethnicity) - Early life IPT not linked with child asthma - Maternal prenatal asthma mediated link between chronic IPT and child asthma	ROB: Moderate; primary source of bias was non-representative sample; retrospective measure of maternal preconception adversity and maternal report of offspring health

Study: Cammack et al., 2019 Sample: 4,181 female adolescents (18.2% African American) and their infants Design: Retrospective cohort	Predictors: Maternal childhood abuse and age each abuse first occurred Outcomes: Maternal reported infant PTB and VPTB	- Maternal CSA exposure between ages 9-18 by non-parental/adult caregivers using physical force → ↑VPTB risk (controls: race, childhood SES)	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity; maternal report of offspring health
Study: Daniels et al., 2020 Sample: 208 African-American women and their infants Design: Retrospective, cross-sectional	Predictors: Direct and vicarious racial discrimination in childhood, adolescence, & adulthood Outcome: Maternal reported infant PTB	- ↑Adolescent direct racial discrimination → ↑PTB risk (controls: # of pregnancies, education, employment status, marital status) - ↑Childhood vicarious racial discrimination → ↑PTB risk	ROB: High; primary sources of bias were non-representative sample and inadequate participants' response rate information; retrospective measure of maternal preconception adversity and maternal report of offspring health
Study: Flagg et al., 2014 Sample: 535 adolescent mothers (28% African American), their parents, and their infants Design: Retrospective cohort	Predictor: Grandparental perceived neighborhood disorder Outcome: Maternal reported infant BW	- Grandparental exposure to neighborhood disorder not linked to grandchild's BW (controls: grandparent education, maternal race, age, BW, prenatal care, drug use, PTB)	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity; maternal report of offspring health

Study: Freeman, 2014 Sample: 2,332 mothers (43.7% African American) and their infants Design: Retrospective cohort	Predictor: Grandmother report of maternal early life poverty Outcome: Maternal reported infant birthweight, with LBW	- Maternal early life poverty not associated with LBW (controls: maternal race, infant sex, maternal health	ROB: Moderate; primary source of bias was inadequate participants' response rate information; maternal report of offspring health
Study: Gavin et al., 2011 Sample: 136 mother-child dyads (26% African American) Design: Retrospective cohort	Predictors: Maternal childhood maltreatment using CTQ; Maternal childhood low SES Outcome: Maternal reported BW Mediators: Maternal adolescent substance use; Maternal prenatal tobacco and alcohol use	- Maternal low childhood SES → ↓BW (controls: maternal substance use) Mechanisms of Transmission: - Maternal early childhood maltreatment → ↑adolescent substance use and ↑prenatal tobacco and alcohol use → ↑risk LBW	ROB: Moderate; primary source of bias was non-representative sample; retrospective measure of maternal preconception adversity and maternal report of offspring health
Study: Hillis et al., 2004 Sample: 9,159 women (4.7% African American) and their infants Design: Retrospective cohort	Predictor: Maternal ACEs Outcome: Maternal reported pregnancy outcome (e.g., live birth, stillbirth/miscarriage)	- ↑Maternal ACEs → ↑risk of fetal death in 1st pregnancy (controls: maternal age, race, education, adolescent pregnancy) - In 2nd pregnancy, ↑ maternal ACEs → ↑ risk of fetal death - If 1st pregnancy as teen → no elevated risk of fetal death	ROB: High; primary sources of bias were non-representative sample used and inadequate participant response rate obtained; retrospective measure of maternal preconception adversity and maternal report of offspring health

Study: Ihongbe, T. O. Sample: 4,419 women (20.7% African American) and their infants Design: Retrospective cohort	Predictor: Maternal exposure to neighborhood violence in study waves prior to the delivery of their infant Outcome: Maternal reported PTB Moderator: Maternal social support	- ↑maternal exposure to neighborhood violence → ↑PTB risk vs. women not exposed to neighborhood violence (controls: maternal age, insurance status, marital status, household income, prenatal alcohol use) - Social support did not moderate association	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity; maternal report of offspring health
Study: Kerkar et al., 2021 Sample: 1,511 women (63.3% African American) and their infants Design: Retrospective cohort	Predictors: Maternal ACEs using ACEs survey Outcomes: Maternal reported pregnancy outcome (e.g., MAP; MFP)	- ↑Maternal ACEs → ↑risk of MFP and MAP (controls: maternal age at pregnancy, race, BMI, education, marital status, smoking)	ROB: High; primary sources of bias were inadequate participants' response rate information and non-representative sample; retrospective measure of maternal preconception adversity and maternal report of offspring health
Study: Lê-Scherban et al., 2018 Sample: 350 parent-child dyads (45.1% African American; 80% adult women) Design: Retrospective cross-sectional	Predictors: Parental exposure to ACEs using ACE study and Behavioral Risk Factor Surveillance Survey ACE module; Parental community-based childhood stress Outcomes: Proxy report (92% parent) of child health outcomes	- ↑Parental ACEs → ↑risk of poor offspring health not related to risk of obesity or asthma (controls: parent age, sex, race/ethnicity, child age, sex) - ↑Parental expanded ACE exposure → ↑odds of poorer offspring health, obesity, and asthma	ROB: Moderate; primary source of bias was inadequate participant response rate obtained; retrospective measure of maternal preconception adversity and maternal report of offspring health

Study: Stein et al., 2000 Sample: 974 homeless women (57.4% African American) and their infants Design: Retrospective, cross-sectional	Predictors: Maternal history of rape or CSA before age 18; Maternal assault before age 18 Outcomes: Maternal reported PTB & LBW	- Women reporting rape or CSA before age 18 → ↑ PTB risk and ↓GA vs. no rape/CSA; (controls: ethnicity, income) - No significant difference in PTB and LBW risk for women reporting an assault before age 18	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity; maternal report of offspring health
Study: Strutz et al., 2014 Sample: 3,512 1st-time (23.7% African American) and 1,901 (25.5% African American) 2nd-time mothers Design: Retrospective cohort	Predictors: Maternal PSLEs in adolescence and emerging adulthood; Maternal PSLEs pertaining to family of origin and early experiences Outcome: Maternal report infant BW	- ↑ Chronic maternal PSLEs → ↑ risk LBW in 1st & 2nd births (controls: age, parity, BMI, smoking, alcohol, marital status) - Acute maternal PSLEs not linked with infant BW	ROB: Low; primary source of bias was retrospective measure of maternal preconception adversity; maternal report of offspring health

Abbreviations for table: ACEs=Adverse Childhood Experiences; ACT=Asthma Control Test; BMI=body mass index; BW=Birth weight; CES=Childhood Experiences Survey; CTQ=Childhood Trauma Questionnaire; DBP= diastolic blood pressure; EPDS=Edinburgh Postnatal Depression Scale; ETI-SF=Early Trauma Inventory Self Report Short Form; GA=gestational age; HRV=heart-rate variability; IPT= interpersonal trauma; LBW=low birth weight (< 2500 grams or 5.5 pounds); LF/HF=low-frequency to high-frequency band; LSC-R=Life Stressor Checklist-Revised; MAP=miscarriage at any pregnancy; MFP=miscarriage at first pregnancy; NDI=neighborhood disadvantage index; PS=perceived stress; PSLEs= stressful life events prior to conception; PSS=Cohen's Perceived Stress Scale; PSWQ=Penn State Worry Questionnaire; PTB=preterm birth (birth < 37 completed weeks gestation); early PTB= birth ≤ 34 weeks gestation; late PTB= birth between 35-36 weeks gestation; PTSD=post-traumatic stress disorder; R- CTS=Revised Conflict Tactics Scale short form; RSA=respiratory sinus arrhythmia; SBP= systolic blood pressure; SCID=Structural Clinical Interview for DSM; SCN=admission to special care nursery; SES=socioeconomic status; SFP=Still Face Paradigm; SGA=small for gestational age; SHCN=special health care need; SLE=Stressful life events; SLEI=Lobel and Zambrana Stressful Life Events Inventory; SRI= serotonin reuptake inhibitor; STAXI-2=State-Trait Anger Expression Inventory-2; STD=

sexually transmitted disease; STRAIN=Stress and Adversity Inventory; TL=telomere length; VLBW=very low birth weight (< 1500g); VPTB=very preterm birth (<34 weeks gestation)

Note: Brunst et al. (2017) and Sternthal et al. (2011) were produced from the same Asthma Coalition on Community Environment and Social Stress (ACCESS) project. Cheng et al. (2016), Witt et al. (2014a,b), Witt et al. (2015), and Witt et al. (2016) were all produced from the same the Early Childhood Longitudinal Study-Birth Cohort. Cammack et al. (2019), Flagg et al. (2014), Ihongbe (2018), and Strutz et al. (2014) were all produced from the National Longitudinal Study of Adolescent to Adult Health (Add Health)

Appendix F: Newcastle Ottawa Scale for Quality Assessment for Cohort Studies Criteria

Selection (5 maximum total points):
Representativeness of the exposed cohort *Enter 0 or 1:* 1 = truly representative of the average _____ in the community 1 = somewhat representative of the average _____ in the community 0 = selected group of users (e.g., nurses, volunteers) 0 = no description of the derivation of the cohort
Selection of the non-exposed cohort *Enter 0 or 1:* 1 = drawn from the same community as the exposed cohort 0 = drawn from a different source 0 = no description of the derivation of the non-exposed cohort
***Ascertainment of exposure** *Enter 0 or 1:* 1 = biological test (e.g., blood/urine) 1 = structured interview 1 = written self-report that characterizes dose (current or cumulative) 0 = written self-report without quantification of exposure 0 = no description
***Ascertainment of exposure done prospectively or retrospectively** *Enter 0 or 1:* 1 = Prospectively 0 = Retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment *Enter 0 or 1:* 1= yes 0 = no
Comparability (2 maximum total points):
Comparability of cohorts on the basis of the design or analysis *Add points: Minimum 0, Maximum 2* 1 = study accounts/controls for _____ (most important factor) 1 = study controls for any additional factor 0 = no adjustment for potential confounders
Outcome (3 maximum total points):
***Assessment of outcome** *Enter 0 or 1:* 1 = objective measure 1 = validated self-report measures 0 = no information or non-validated measures

Was follow-up long enough for outcomes to occur? *Enter 0 or 1:* 1 = yes (select an adequate follow up period for outcome of interest) 0 = no
Adequacy of follow-up of cohorts *Enter 0 or 1:* 1 = complete follow-up; all subjects accounted for 1 = subjects lost to follow-up unlikely to introduce bias - small number lost → ___% (select an adequate %) or description was provided of those lost 0 = follow-up rate < ___% (select an adequate %) and no description of those lost 0 = no statement

*Modified based on Portland, V. A., Kansagara, D., O'Neil, M., Nugent, S., Freeman, M., Low, A., Kondo, K., Elven, C., Zakher, B., Motu'apuaka, M, Paynter, R., & Morasco, B. J. (2017). Benefits and harms of cannabis in chronic pain or post-traumatic stress disorder: A systematic review.

Appendix G: Newcastle Ottawa Scale for Quality Assessment for Cohort Studies

CRITERIA CATEGORIES	Astone et al. (2007)	Brunst et al. (2017)	Cammack et al. (2019)	Flagg et al. (2014)	Freeman (2014)
Representativeness of the exposed cohort	0 – select group of mothers (convenience sampling)	0 – select group of pregnant women (convenience sampling)	1 – truly representative of the average U.S. school (stratified sampling)	1 – truly representative of the average adolescent in the U.S. (probability sampling)	1 – truly representative of the average adolescent in the U.S. (probability sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – structured interview (face-to-face interview)	1 – written self-report that characterizes dose (validated self-report measure [R-CTS])	1 – written self-report that characterizes dose	1 – written self-report that characterizes dose	1 – written self-report that characterizes dose
Ascertainment of exposure done prospectively or retrospectively	1 – prospectively	0 – retrospectively	0 – retrospectively	1 – prospectively	1 – prospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	1 – yes	1 – yes	1 – yes	1 – yes	1 – yes
Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal prenatal health, SES, smoking)	1 – study controls for any additional factors (e.g., child sex, maternal age, education, race, sex & birthweight)	1 – study controls for any additional factors (e.g., maternal race & childhood SES)	1 – study controls for any additional factors (e.g., grandparental education, maternal birthweight, age, substance use)	1 – study controls for any additional factors (e.g., maternal health, prenatal environment, current SES)
Assessment of outcome	0 – non-validated measure (maternal report)	1 – objective measure (maternal report of clinician-diagnosed asthma)	0 – non-validated measure (maternal report)	0 – non-validated measure (maternal report)	0 – non-validated measure (maternal report)

Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	1 – subjects lost to follow-up unlikely to introduce bias (< 3% of offspring lost)	1 – subjects lost to follow-up unlikely to introduce bias (< 4% lost)	1 – subjects lost to follow-up unlikely to introduce bias (< 20% lost)	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost)	0 – no statement on % of subjects lost to follow-up
Risk of Bias (ROB):	Moderate ROB; the primary source of bias was the non-representative sample used; maternal report of offspring health was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity; maternal report of offspring health was also used	Relatively low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity; maternal report of offspring health was also used	Moderate ROB; the primary source of bias was the lack of information about participants' response rate; maternal report of offspring health was also used

CRITERIA CATEGORIES	Hillis et al. (2004)	Ihongbe (2018)	Kerkar et al. (2021)	Strutz et al. (2014)	Hilmert et al. (2014)
Representativeness of the exposed cohort	0 – select group of women (convenience sampling)	1 – truly representative of the average adolescent in the U.S. (stratified random sampling)	0 – select group of women (convenience sampling)	1 – truly representative of the average adolescent in the U.S. (probability sampling)	0 – select group of pregnant women (convenience sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – written self-report that characterizes dose (validated self-report measure [ACEs Questionnaire])	1 – written self-report that characterizes dose	1 – written self-report that characterizes dose (validated self-report measure [ACEs Questionnaire])	1 – written self-report that characterizes dose	1 - structured interview
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	0 – no	1 – yes	0 – no	1 – yes	1 – yes

Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal age, race, education, & adolescent pregnancy)	1 – study controls for any additional factors (e.g., maternal age, education, receipt of prenatal care, prenatal substance use)	1 – study controls for any additional factors (e.g., maternal age at pregnancy, BMI, education, smoking)	1 – study controls for any additional factors (e.g., maternal preconception BMI, substance use, marital status)	1 – study controls for any additional factors (e.g., maternal BMI, SES, exposure to SLEs)
Assessment of outcome	0 – non-validated measure (maternal report)	0 – non-validated measure (maternal report)	0 – non-validated measure (maternal report)	0 – non-validated measure (maternal report)	1 - objective measure (medical records)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	0 – response rate < 70% (68%)	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost at each wave of data collection)	0 – no statement on % of non-respondents	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost at each wave of data collection)	1 – subjects lost to follow-up unlikely to introduce bias (~7% lost)

Risk of Bias (ROB):	High ROB; the primary sources of bias were the non-representative sample used and the inadequate participant response rate obtained; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity; maternal report of offspring health was also used	High ROB; the primary sources of bias were the lack of information about participants' response rate and the non-representative sample used; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity; maternal report of offspring health was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used

CRITERIA CATEGORIES	Sealy-Jefferson et al. (2019)	Dominguez et al. (2008)	Gray et al. (2017)	Margerison-Zilko et al. (2017)	Masho et al. (2015)
Representativeness of the exposed cohort	0 – select group of women (convenience sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – written self-report that characterizes dose (validated self-report early-life neighborhood social control & social disorder scales)	1 – structured interview	1 – written self-report that characterizes dose (validated self-report measure [ACEs Questionnaire])	1 – structured interview (detailed in-person & self-recorded interview)	1 – written self-report that characterizes dose (validated self-report measures [SLEI & PSS])
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	0 – no	1 – yes	1 – yes	1 – yes	1 – yes
Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal age, marital status, educational attainment, income)	1 – study controls for any additional factors (e.g., maternal medical & sociodemographic risk factors, gestational age at delivery, spontaneous labor)	1 – study controls for any additional factors (e.g., gestational age, maternal education, infant sex)	1 – study controls for any additional factors (e.g., maternal education, parity, marital status)	1 – study controls for any additional factors (e.g., maternal age, education, adequacy of prenatal care)

Assessment of outcome	1 - objective measure (medical records)	1 - objective measure (medical records)	1 - objective measure (EEG)	1 - objective measure (medical records)	1 - objective measure (medical records)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	1 – non-respondents unlikely to introduce bias (29% of participants approached declined participation)	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost)	0 – no statement on % of subjects lost to follow-up	1 – subjects lost to follow-up unlikely to introduce bias (< 1% lost)	0 – no statement on % of subjects lost to follow-up
Risk of Bias (ROB):	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	High ROB; the primary sources of bias were lack of information about participants' follow-up rate and the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	High ROB; the primary sources of bias were the lack of information about participants' follow-up rate and the non-representative sample used; a retrospective measure of maternal preconception adversity was also used

CRITERIA CATEGORIES	Seng et al. (2011)	Blackmore et al. (2016)	Cheng et al. (2016)	Mersky & Lee (2019)	Noll et al. (2007)
Representativeness of the exposed cohort	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)	1 – truly representative of the average child born in the U.S. (probability sampling)	0 – select group of women (convenience sampling)	0 – select group of women (convenience sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort
Ascertainment of exposure	1 – written self-report that characterizes dose (validated self-report measure [Life Stressor Checklist])	1 – written self-report that characterizes dose (validated self-report measure [PTSD section of the SCID])	1 – structured interview	1 – written self-report that characterizes dose (validated self-report measure [ACEs Questionnaire])	1 – structured interview (referral by CPS agencies)
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	1 – prospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	1 – yes	1 – yes	0 – no	0 – no	1 – yes
Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal poverty, chronic condition, antepartum complication, substance use, adequate prenatal care)	1 – study controls for any additional factors (e.g., maternal age, BMI, prenatal substance use)	1 – study controls for any additional factors (e.g., maternal chronic conditions, parity, age, SES)	1 – study controls for any additional factors (e.g., maternal age, race/ethnicity, and education)	1 – study controls for any additional factors (e.g., maternal minority status, offspring number of siblings in sample)

Assessment of outcome	1 - objective measure (medical records)	1 - objective measure (medical records)	1 - objective measure (birth certificate)	1 - objective measure (archival program records)	1 - objective measure (hospital records)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	0 – follow-up rate < 70% (~53%)	1 – subjects lost to follow-up unlikely to introduce bias (< 5% lost)	0 – no statement on % of non-respondents	0 – no statement on % of non-respondents	1 – subjects lost to follow-up unlikely to introduce bias (~4% lost)
Risk of Bias (ROB):	High ROB; the primary sources of bias were the non-representative sample used and the inadequate % of participants retained at follow up; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity used	High ROB; the primary sources of bias were the lack of information about participants' response rate and the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used

CRITERIA CATEGORIES	Smith et al. (2016)	Sternthal et al. (2011)	Witt et al. (2014a)	Witt et al. (2014b)	Witt et al. (2015)
Representativeness of the exposed cohort	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)	1 – truly representative of the average child born in the U.S. (probability sampling)	1 – truly representative of the average child born in the U.S. (probability sampling)	1 – truly representative of the average child born in the U.S. (probability sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – written self-report that characterizes dose (modified validated self-report measure [ETI-SF])	1 – written self-report that characterizes dose (self-report of binary measure)	1 – structured interview	1 – structured interview	1 – structured interview
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	1 – yes	1 – yes	0 – no	0 – no	0 – no

Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal race/ethnicity [other additional factors were mediators (e.g., maternal smoking, education, marital status)])	1 – study controls for any additional factors (e.g., maternal atopy, nativity status, race/ethnicity, child sex)	2 – study controls for most important factor (prenatal adversity) and any additional factors (e.g., maternal sociodemographic & health factors, prenatal stress)	2 – study controls for most important factor (prenatal adversity) and any additional factors (e.g., maternal sociodemographic & health factors, prenatal stress)	2 – study controls for most important factor (prenatal adversity) and any additional factors (e.g., maternal sociodemographic & health factors, prenatal stress)
Assessment of outcome	1 - objective measure (medical records)	1 - objective measure (enzyme immunoassay)	1 - objective measure (birth certificate)	1 - objective measure (birth certificate)	1 - objective measure (birth certificate)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	1 – subjects lost to follow-up unlikely to introduce bias (~14% lost)	1 – subjects lost to follow-up unlikely to introduce bias (~23% lost)	1 – non-respondents unlikely to introduce bias (< 24% of participants approached declined participation)	1 – non-respondents unlikely to introduce bias (< 24% of participants approached declined participation)	0 – no statement on % of non-respondents
Risk of Bias (ROB):	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity used

CRITERIA CATEGORIES	Witt et al. (2016)	Cowell et al. (2021)	Gavin et al. (2011)	Gillespie et al. (2017)	Jones et al. (2019)
Representativeness of the exposed cohort	1 – truly representative of the average child born in the U.S. (probability sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of elementary school children (convenience sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of pregnant women (convenience sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – structured interview	1 – written self-report that characterizes dose (validated self-report measure [CTQ])	1 – written self-report that characterizes dose (validated self-report measure [CTQ])	1 – written self-report that characterizes dose (validated self-report measure [STRAIN])	1 – written self-report that characterizes dose (validated self-report measure [ACEs Questionnaire])
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	0 – no	1 – yes	1 – yes	1 – yes	1 – yes

Comparability of cohorts on the basis of the design or analysis	2 – study controls for most important factor (prenatal adversity) and any additional factors (e.g., maternal sociodemographic & health factors)	1 – study controls for any additional factors (e.g., maternal age, parity, education, smoking)	1 – study controls for any additional factors (e.g., maternal BMI, prenatal substance use)	1 – study controls for any additional factors (e.g., maternal adulthood stress, sleep quality, hours awake prior to venipuncture)	1 – study controls for any additional factors (e.g., infant sex, maternal race, prenatal smoking)
Assessment of outcome	1 - objective measure (birth certificate)	1 – objective measure (medical records)	0 – non-validated measure (maternal report)	1 – objective measure (prenatal and labor & delivery records)	1 – objective measure (ECG, placental TL)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)	1 – yes (offspring was born)
Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	1 – non-respondents unlikely to introduce bias (< 24% of participants approached declined participation)	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost)	1 – description of subjects lost was provided (no differences between subjects retained and lost)	1 – subjects lost to follow-up unlikely to introduce bias (1% lost)	0 – no statement on % of subjects lost to follow-up
Risk of Bias (ROB):	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	High ROB; the primary sources of bias were the lack of information about participants' follow-up rate and the non-representative sample used; a retrospective measure of maternal preconception adversity was also used

CRITERIA CATEGORIES	Miller et al. (2017)
Representativeness of the exposed cohort	0 – select group of pregnant women (convenience sampling)
Selection of the non-exposed cohort	1 – drawn from the same community as the exposed cohort (same sample)
Ascertainment of exposure	1 – written self-report that characterizes dose
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively
Demonstration that outcome of interest was not present at start of study, OR baseline assessment	1 – yes
Comparability of cohorts on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal demographics, education, and obstetrical confounders [e.g., nulliparity])
Assessment of outcome	1 - objective measure (maternal and neonatal charts)
Was follow-up long enough for outcomes to occur?	1 – yes (offspring was born)

Adequacy of follow-up of prospective cohorts/Adequacy of response of retrospective cohorts	1 – subjects lost to follow-up unlikely to introduce bias (< 30% lost)
Risk of Bias (ROB):	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used

Appendix H: Newcastle Ottawa Scale for Quality Assessment for Cross-Sectional Studies Criteria

Selection (5 maximum total points):
Representativeness of the sample *Enter 0 or 1:* 1 = truly representative of the average in the target population (all subjects or random sampling) 1 = somewhat representative of the average in the target population (non-random sampling) 0 = select group of users (e.g., nurses, volunteers) 0 = no description of the sampling strategy
Non-respondents *Enter 0 or 1:* 1 = comparability between respondents and non-respondents characteristics is established and the response rate is satisfactory 0 = the response rate is unsatisfactory, or the comparability between respondents and non-respondents is unsatisfactory 0 = no description of the response rate or the characteristics of the respondents and non-respondents
Sample size *Enter 0 or 1:* 1 = justified and satisfactory 0 = not justified
Ascertainment of exposure *Enter 0 or 1:* 1 = validated measurement tool 1 = non-validated measurement tool that is available or described 0 = no description of the measurement tool
***Ascertainment of exposure done prospectively or retrospectively** *Enter 0 or 1:* 1 = Prospectively 0 = Retrospectively
Comparability (2 maximum total points):
The subjects in different outcome groups are comparable, based on the study design or analysis - confounding factors are controlled *Add points: Minimum 0, Maximum 2* 1 = study accounts/controls for the most important factor (select one) 1 = study controls for any additional factor 0 = no adjustment for potential confounders
Outcome (3 maximum total points):
Assessment of outcome *Enter 0 or 1:*

2 = independent blind assessment 1 = record linkage 1 = self-report 0 = no description
Statistical test *Enter 0 or 1:* 1 = the statistical test used to analyze the data is clearly described and appropriate, and the measurement of the association is presented, including confidence intervals and the probability level (p value) 0 = the statistical test is not appropriate, not descried or incomplete

*Adapted for cross-sectional studies by Herzog, R., Álvarez-Pasquin, M. J., Díaz, C., Del Barrio, J. L., Estrada, J. M., & Gil, Á. (2013). Are healthcare workers' intentions to vaccinate related to their knowledge, beliefs and attitudes? A systematic review. *BMC Public Health, 13(1)*, 1-17. doi: 10.1186/1471-2458-13-154

Appendix I: Newcastle Ottawa Scale for Quality Assessment for Cross-Sectional Studies

CRITERIA CATEGORIES	Jovanovic et al. (2011)	Rowell (2020)	Chen et al. (2017)	Daniels et al. (2020)	Stein et al. (2000)
Representativeness of the exposed cohort	0 – select group of children (convenience sampling)	0 – select group of pregnant women (convenience sampling)	0 – select group of children (convenience sampling)	0 – select group of women (convenience sampling)	1 - somewhat representative of the average homeless woman in LA (stratified sampling)
Non-respondents	0 – no description of the response rate	0 – no description of the response rate	1 – the response rate is satisfactory (~76%)	0 – no description of the response rate	1 - the response rate is satisfactory (81%)
Sample size	0 – not satisfactory (36 children)	0 – not satisfactory (31 pregnant women)	1 – satisfactory (150 children)	1 – satisfactory (208 women)	1 - (237 homeless women with live births in the last 3 years)
Ascertainment of exposure	1 – validated measurement tool (self-reported CTQ)	1 – validated measurement tool (self-reported ACEs Questionnaire)	1 – non-validated measurement tool that is available or described (self-reported childhood home ownership)	1 – non-validated measurement tool that is available or described (adolescent and childhood exposure to direct & vicarious racial discrimination)	1 - non-validated measurement tool that is available or described (self-reported yes or no to rape or sexual abuse and assault before age 18)
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively	0 – retrospectively
Comparability: The subjects in different outcome groups are comparable, based on the study design or analysis - confounding factors are controlled	1 – study controls for any additional factors (e.g., child trauma exposure, child sex & age, maternal PTSD & depression)	1 – study controls for any additional factors (e.g., maternal distress, trimester, BMI, systolic & diastolic blood pressure)	1 – study controls for any additional factors (e.g., child age, sex, ethnicity use of beta agonists, use of inhaled corticosteroids)	1 – study controls for any additional factors (e.g., maternal parity, household income, educational attainment, employment status, marital status)	1 – study controls for any additional factors (e.g., maternal age, nulliparity, antenatal complications)

Assessment of outcome	2 – independent or blind assessment (EMG & ECG)	2 – independent or blind assessment (doulas present at delivery)	2 – independent or blind assessment (blood samples)	1 – self-report (maternal report)	1 – self-report (maternal report)
Statistical test	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (ANOVAs & hierarchical regressions, coefficients & F statistics, $p < .05$)	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (linear regressions, coefficients, $p < .05$)	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (ANCOVAs & multiple regressions, coefficients & F statistics, 95% CIs, $p < .05$)	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (logistic regression, ORs, 95% CIs, $p < .05$)	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (SEM, coefficients, $p < .05$)
Risk of Bias (ROB):	High ROB; the primary sources of bias were the small and non-representative sample used, and lack of information about participants' response rate; a retrospective measure of maternal preconception adversity was also used	High ROB; the primary sources of bias were the small and non-representative sample used, and lack of information about participants' response rate; a retrospective measure of maternal preconception adversity was also used	Moderate ROB; the primary source of bias was the non-representative sample used; a retrospective measure of maternal preconception adversity was also used	High ROB; the primary sources of bias were the non-representative sample used and the lack of information about participants' response rate; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used	Low ROB; the primary source of bias was the retrospective measure of maternal preconception adversity; maternal report of offspring health was also used

CRITERIA CATEGORIES	Lê-Scherban et al. (2018)
Representativeness of the exposed cohort	1 - somewhat representative of the average resident of Philadelphia & its surrounding counties (stratified sampling)
Non-respondents	0 – the response rate is unsatisfactory (67%)
Sample size	1 – satisfactory (350 parents & their children)
Ascertainment of exposure	1 - validated measurement tool (self-reported adapted ACEs Questionnaire & BRFSS ACE module)
Ascertainment of exposure done prospectively or retrospectively	0 – retrospectively
Comparability: The subjects in different outcome groups are comparable, based on the study design or analysis - confounding factors are controlled	1 – study controls for any additional factors (e.g., parent age & sex, child age & sex)
Assessment of outcome	1 – self-report (parent report)
Statistical test	1 - the statistical test used is clearly described and appropriate & the measurement of the association is presented (logistic regression, ORs, 95% CIs, $p < .05$)
Risk of Bias (ROB):	Moderate ROB; the primary source of bias was the inadequate participant response rate obtained; a retrospective measure of maternal preconception adversity and maternal report of offspring health were also used

Appendix J: Newcastle Ottawa Scale for Quality Assessment for Case-Control Studies Criteria

Selection (A study can be awarded a maximum of one star for each numbered item):
1) Is the case definition adequate? Representativeness of the exposed cohort a) yes, with independent validation* b) yes, e.g., record linkage or based on self-reports c) no description
2) Representativeness of the cases a) consecutive or obviously representative series of cases* b) potential for selection biases or not stated
3) Selection of controls a) community controls* b) hospital controls c) no description
4) Definition of controls a) no history of disease (endpoint)* b) no description of source
Comparability (A study can be awarded a maximum of one star):
1) Comparability of cases and controls on the basis of the design or analysis a) study controls for _____ (Select the most important factor.)* b) study controls for any additional factor* (These criteria could be modified to indicate specific control for a second important factor.)
Exposure (A study can be awarded a maximum of one star for each numbered item):
1) Ascertainment of exposure a) secure record (e.g., surgical records)* b) structured interview where blind to case/control status* c) interview not blinded to case/control status d) written self-report or medical record only e) no description
2) Same method of ascertainment for cases and controls a) yes* b) no
3) Non-Response rate a) same rate for both groups* b) non respondents described c) rate different and no designation

Appendix K: Newcastle Ottawa Scale for Quality Assessment for Case-Control Studies

CRITERIA CATEGORIES	Freedman et al. (2017)
Is the case definition adequate?	1 – yes, with independent validation (medical records)
Representativeness of the cases	1 – consecutive or obviously representative series (population-based study with stratified random sampling)
Selection of controls	1 – community controls (same birth hospitals as cases)
Definition of controls	1 – index delivery did not result in stillbirth
Comparability of cases and controls on the basis of the design or analysis	1 – study controls for any additional factors (e.g., maternal education, age, time between index delivery & follow-up interview)
Ascertainment of exposure	0 – written self-report (CTQ)
Same method of ascertainment for cases and controls	1 – yes (CTQ)
Non-response rate	0 – rate different for each group (17% non-response for cases vs. 25% non-response for controls)
Risk of bias (ROB):	Moderate ROB; the primary source of bias was the different participant response rates in the case and control groups; a retrospective measure of maternal preconception adversity was also used

Appendix L: Hierarchical generalized linear model examining associations between maternal general adversity by timing and self-rated health (*n* = 56)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	0.00(-0.02, 0.02)	.849	0.00(-0.02, 0.02)	.868	0.00(-0.02, 0.02)	.823
Income during year child was born[b]						
$25k-$49,999	-0.56(-1.18, 0.05)	.076	-0.55(-1.18, 0.07)	.082	-0.41(-1.08, 0.24)	.213
$50k-$74,999	-0.56(-1.18, 0.05)	.074	-0.55(-1.18, 0.06)	.079	-0.47(-1.09, 0.15)	.139
$75k or more	**-0.71(-1.34, -0.09)**	**.025**	**-0.74(-1.37, -0.10)**	**.022**	-0.57(-1.23, 0.08)	.086
Adversity experiences						
Childhood	0.00(-0.04, 0.04)	.971	-0.00(-0.06, 0.04)	.775	-0.00(-0.06, 0.04)	.747
Preconception	—	—	0.01(-0.04, 0.08)	.614	-0.00(-0.08, 0.06)	.800
Post-conception	—	—	—	—	0.05(-0.02, 0.14)	.172
Constant	2.79(1.46, 4.13)	<.001	2.80(1.47, 4.14)	<.001	2.55(1.12, 3.98)	<.001
Model Statistics (AIC, BIC)	(4.08, -195.09)		(4.12, -191.09)		(4.15, -187.30)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix M: Hierarchical generalized linear model examining associations between maternal law enforcement adversity by timing and self-rated health (*n* = 50)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	0.00(-0.02, 0.02)	.773	0.00(-0.02, 0.03)	.728	0.00(-0.02, 0.03)	.578
Income during year child was born[b]						
$25k-$49,999	-0.59(-1.26, 0.08)	.088	-0.57(-1.26, 0.11)	.103	-0.56(-1.26, 0.12)	.110
$50k-$74,999	-0.63(-1.31, 0.03)	.065	-0.70(-1.44, 0.02)	.058	**-0.82(-1.62, -0.02)**	**.042**
$75k or more	**-1.01(-1.72, -0.31)**	**.005**	**-1.05(-1.78, -0.32)**	**.005**	**-1.13(-1.90, -0.37)**	**.004**
Adversity experiences						
Childhood	-0.03(-0.14, 0.07)	.532	-0.08(-0.29, 0.13)	.452	-0.13(-0.37, 0.09)	.254
Preconception	—	—	0.05(-0.16, 0.27)	.613	0.05(-0.16, 0.26)	.643
Post-conception	—	—	—	—	0.09(-0.11, 0.29)	.399
Constant	2.81(1.49, 4.13)	<.001	2.77(1.42, 4.11)	<.001	2.58(1.14, 4.02)	<.001
Model Statistics (AIC, BIC)	(4.09, -166.42)		(4.13, -162.54)		(4.17, -158.72)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix N: Hierarchical generalized linear model examining associations between maternal law enforcement adversity by timing and waist to height ratio (*n* = 48)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Age[a]	0.00(-0.00, 0.00)	.958	0.00(-0.00, 0.00)	.956	0.00(-0.00, 0.00)	.994
Income during year child was born[b]						
$25k-$49,999	-0.01(-0.09, 0.06)	.641	-0.01(-0.10, 0.06)	.660	-0.01(-0.10, 0.06)	.651
$50k-$74,999	-0.00(-0.08, 0.07)	.856	-0.00(-0.09, 0.07)	.847	-0.00(-0.09, 0.08)	.914
$75k or more	-0.04(-0.14, 0.04)	.304	-0.04(-0.14, 0.04)	.308	-0.04(-0.14, 0.05)	.392
Adversity experiences						
Childhood	-0.00(-0.01, 0.00)	.550	-0.00(-0.03, 0.02)	.700	-0.00(-0.03, 0.02)	.898
Preconception	—	—	0.00(-0.02, 0.02)	.942	0.00(-0.02, 0.02)	.890
Post-conception	—	—	—	—	-0.00(-0.02, 0.01)	.676
Constant	0.56(0.39, 0.72)	<.001	0.56(0.39, 0.72)	<.001	0.56(0.39, 0.73)	<.001
Model Statistics (AIC, BIC)	(-1.56, -162.13)		(-1.52, -158.26)		(-1.48, -154.39)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix O: Hierarchical generalized linear model examining associations between maternal racial discrimination by timing and number of physician-diagnosed ailments (*n* = 55)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics				
Age[a]	**0.97(0.95, 0.99)**	**.043**	**0.97(0.95, 0.99)**	**.045**
Income during year child was born[b]				
$25k-$49,999	**0.39(0.21, 0.73)**	**.003**	**0.33(0.16, 0.64)**	**001**
$50k-$74,999	0.77(0.47, 1.25)	.301	0.72(0.43, 1.19)	.207
$75k or more	0.75(0.44, 1.25)	.272	0.69(0.38, 1.26)	.236
Adversity experiences				
Childhood	1.00(0.99, 1.02)	.448	1.00(0.97, 1.03)	.815
Preconception	—	—	1.00(0.98, 1.02)	.696
Constant	8.05(2.71, 23.86)	<.001	9.43(2.88, 30.87)	<.001
Model Statistics (AIC, BIC)	(4.67, -66.15)		(4.87, -38.51)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix P: Hierarchical generalized linear model examining associations between maternal racial discrimination by timing and self-rated health (*n* = 55)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics				
Age[a]	0.00(-0.01, 0.03)	.525	0.00(-0.02, 0.03)	.658
Income during year child was born[b]				
$25k-$49,999	-0.49(-1.10, 0.10)	.107	-0.59(-1.25, 0.06)	.078
$50k-$74,999	**-0.66(-1.27, -0.06)**	**.031**	**-0.71(-1.40, -0.02)**	**.041**
$75k or more	-0.60(-1.21, 0.01)	.055	-0.72(-1.45, 0.00)	.052
Adversity experiences				
Childhood	0.01(-0.01, 0.04)	.234	0.01(-0.02, 0.05)	.476
Preconception	—	—	0.00(-0.02, 0.03)	.918
Constant	2.38(1.13, 3.63)	<.001	2.50(1.02, 3.97)	.001
Model Statistics (AIC, BIC)	(4.11, -190.86)		(4.17, -158.21)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix Q: Hierarchical generalized linear model examining associations between maternal racial discrimination by timing and waist to height ratio (*n* = 53)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics				
Age[a]	0.00(-0.00, 0.00)	.988	0.00(-0.00, 0.00)	.855
Income during year child was born[b]				
$25k-$49,999	-0.01(-0.09, 0.06)	.715	-0.02(-0.10, 0.05)	.595
$50k-$74,999	0.00(-0.07, 0.08)	.897	0.00(-0.07, 0.08)	.949
$75k or more	-0.02(-0.10, 0.05)	.493	-0.04(-0.13, 0.05)	.376
Adversity experiences				
Childhood	-0.00(-0.00, 0.00)	.899	0.00(-0.00, 0.00)	.697
Preconception	—	—	-0.00(-0.00, 0.00)	.428
Constant	0.55(0.38, 0.72)	<.001	0.54(0.36, 0.73)	<.001
Model Statistics (AIC, BIC)	(-1.60, -186.10)		(-1.52, -158.26)	

[a] Used as continuous variable

[b] Reference group = less than $25,000

Appendix R: Hierarchical generalized linear models examining associations between offspring adversity by timing and waist to height ratio (*n* = 56)

	General Adversity				Law Enforcement			
	Model 1		Model 2		Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics								
Age[a]	**0.00(0.00, 0.00)**	**.009**	0.00(-0.00, 0.00)	.284	**0.00(0.00, 0.00)**	**.007**	**0.00(0.00, 0.00)**	**.028**
Female gender[b]	0.04(-0.01, 0.11)	.160	0.04(-0.02, 0.11)	.212	0.03(-0.04, 0.10)	.432	0.03(-0.04, 0.10)	.441
Adversity experiences								
General childhood	0.00(-0.00, 0.00)	.330	0.00(-0.00, 0.00)	.776	__	__	__	__
General adulthood	__	__	0.00(-0.00, 0.01)	.507	__	__	__	__
Law enforcement childhood	__	__	__	__	-0.00(-0.03, 0.01)	.595	-0.00(-0.03, 0.02)	.603
Law enforcement adulthood	__	__	__	__	__	__	0.00(-0.02, 0.02)	.918
Constant	0.33(0.23, 0.43)	<.001	0.36(0.22, 0.49)	<.001	0.36(0.25, 0.47)	<.001	0.36(0.25, 0.48)	<.001
Model Statistics (AIC, BIC)	(-1.79, -208.84)		(-1.76, -204.82)		(-1.78, -208.83)		(-1.74, -204.81)	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix S. Hierarchical generalized estimating equations examining associations between maternal law enforcement adversity by timing and number of offspring physician-diagnosed ailments (*n* = 51)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics						
Female gender[a]	1.71(0.89, 3.25)	.101	1.50(0.77, 2.93)	.232	1.50(0.76, 2.93)	.235
Age[b]	1.02(0.99, 1.04)	.068	**1.02(1.00, 1.04)**	**.046**	**1.02(1.00, 1.04)**	**.046**
Offspring adversity	**1.02(1.00, 1.03)**	**.011**	**1.01(1.00, 1.03)**	**.047**	**1.01(1.00, 1.03)**	**.048**
Adversity experiences						
Childhood	0.95(0.84, 1.08)	.501	0.87(0.74, 1.03)	.129	0.87(0.71, 1.06)	.187
Preconception	—	—	1.12(0.96, 1.31)	.139	1.12(0.96, 1.32)	.144
Post-conception	—	—	—	—	1.00(0.85, 1.18)	.973
Constant	0.36(0.14, 0.88)	.027	0.38(0.15, 0.95)	.040	0.38(0.15, 0.95)	.040
Model Statistics	Wald's χ^2 (4, 51) = 15.83, p = .003		Wald's χ^2 (5, 51) = 18.39, p = .002		Wald's χ^2 (6, 51) = 18.40, p = .005	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix T. Hierarchical generalized estimating equations examining associations between maternal racial discrimination adversity by timing and number of offspring physician-diagnosed ailments (*n* = 56)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**IRR (95% CI)**	***p***	**IRR (95% CI)**	***p***
Demographics				
Female gender[a]	1.78(0.94, 3.37)	.073	1.76(0.91, 3.38)	.090
Age[b]	**1.03(1.01, 1.05)**	**<.001**	**1.03(1.00, 1.05)**	**.012**
Offspring adversity	1.01(0.99, 1.02)	.110	1.01(0.99, 1.03)	.123
Adversity experiences				
Childhood	1.01(0.99, 1.03)	.163	1.02(0.98, 1.06)	.157
Preconception	—	—	0.98(0.95, 1.01)	.379
Constant	0.24(0.10, 0.58)	.002	0.25(0.09, 0.66)	.005
Model Statistics	Wald's χ^2 (4, 56) = 26.05, $p < .001$		Wald's χ^2 (5, 50) = 18.24, $p = .002$	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix U. Hierarchical generalized estimating equations examining associations between maternal general adversity by timing and offspring self-rated health (*n* = 57)

	General Adversity					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	**0.83(0.38, 1.29)**	**<.001**	**0.82(0.36, 1.28)**	**<.001**	**0.84(0.38, 1.30)**	**<.001**
Age[b]	0.00(-0.02, 0.03)	.743	0.00(-0.02, 0.03)	.760	0.00(-0.02, 0.03)	.677
Offspring adversity	0.00(-0.01, 0.01)	.590	0.00(-0.01, 0.02)	.520	0.00(-0.00, 0.02)	.441
Adversity experiences						
Childhood	0.01(-0.03, 0.05)	.607	0.01(-0.03, 0.07)	.564	0.01(-0.03, 0.07)	.560
Preconception	—	—	-0.01(-0.06, 0.04)	.674	-0.00(-0.07, 0.05)	.785
Post-conception	—	—	—	—	-0.01(-0.07, 0.05)	.700
Constant	1.45(0.66, 2.23)	<.001	1.46(0.67, 2.25)	<.001	1.41(0.62, 2.21)	<.001
Model Statistics	Wald's χ^2 (4, 57) = 14.92, p = .004		Wald's χ^2 (5, 57) = 14.90, p = .010		Wald's χ^2 (6, 57) = 15.60, p = .016	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix V. Hierarchical generalized estimating equations examining associations between maternal racial discrimination adversity by timing and offspring self-rated health (*n* = 56)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics				
Female gender[a]	**0.87(0.42, 1.31)**	**<.001**	**0.84(0.37, 1.31)**	**<.001**
Age[b]	-0.00(-0.03, 0.02)	.808	0.00(-0.03, 0.04)	.801
Offspring adversity	0.01(-0.00, 0.02)	.171	0.01(-0.00, 0.02)	.229
Adversity experiences				
Childhood	-0.01(-0.03, 0.00)	.241	-0.01(-0.04, 0.01)	.248
Preconception	—	—	-0.00(-0.02, 0.01)	.779
Constant	1.64(0.82, 2.45)	<.001	1.55(0.58, 2.53)	.002
Model Statistics	Wald's χ^2 (4, 56) = 16.43, p = .002		Wald's χ^2 (5, 50) = 16.07, p = .006	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix W. Hierarchical generalized estimating equations examining associations between maternal law enforcement adversity by timing and offspring waist to height ratio (*n* = 51)

	Law Enforcement					
	Model 1		**Model 2**		**Model 3**	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics						
Female gender[a]	0.03(-0.01, 0.08)	.191	0.04(-0.01, 0.09)	.155	0.04(-0.01, 0.09)	.155
Age[b]	0.00(-0.00, 0.00)	.086	0.00(-0.00, 0.00)	.095	0.00(-0.00, 0.00)	095
Offspring adversity	0.00(-0.00, 0.00)	.224	0.00(-0.00, 0.00)	.184	0.00(-0.00, 0.00)	.186
Adversity experiences						
Childhood	-0.00(-0.01, 0.00)	.492	0.00(-0.01, 0.01)	.948	**0.00(-0.01, 0.01)**	**.894**
Preconception	—	—	-0.00(-0.02, 0.01)	.563	-0.00(-0.02, 0.01)	.604
After conception	—	—	—	—	-0.00(-0.01, 0.01)	.861
Constant	0.36(0.28, 0.45)	<.001	0.36(0.28, 0.44)	<.001	0.36(0.28, 0.45)	<.001
Model Statistics	Wald's χ^2 (4, 51) = 7.62, p = .106		Wald's χ^2 (5, 51) = 8.01, p = .155		Wald's χ^2 (6, 51) = 8.04, p = .235	

[a] Female = 1; Male = 0

[b] Used as continuous variable

Appendix X. Hierarchical generalized estimating equations examining associations between maternal racial discrimination by timing and offspring waist to height ratio (*n* = 55)

	Racial Discrimination			
	Model 1		Model 2	
Variables	**ß (95% CI)**	***p***	**ß (95% CI)**	***p***
Demographics				
Female gender[a]	0.05(-0.01, 0.11)	.104	0.03(-0.01, 0.09)	.186
Age[b]	0.00(-0.00, 0.00)	.062	0.00(-0.00, 0.00)	.188
Offspring adversity	0.00(-0.00, 0.00)	.054	0.00(-0.00, 0.00)	.175
Adversity experiences				
Childhood	-0.00(-0.00, 0.00)	.454	-0.00(-0.00, 0.00)	.461
Preconception	__	__	0.00(-0.00, 0.00)	.852
Constant	0.33(0.23, 0.43)	<.001	0.37(0.28, 0.46)	<.001
Model Statistics	Wald's χ^2 (4, 55) = 13.68, p = .008		Wald's χ^2 (5, 50) = 7.42, p = .191	

[a] Female = 1; Male = 0

[b] Used as continuous variable