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Chapter

Health Disparities at the Intersection of Gender and Race: Beyond Intersectionality Theory in Epidemiologic Research

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Abstract

Racial disparities in health have long been one of the primary foci of health inequity research in the United States, yet the use of theoretical frameworks outside of biological determinism has generally been minimal within this literature. Only recently has epidemiology begun to incorporate Intersectionality and other social theories in the study of racial health inequities. Even still, the majority of this research base neglects to engage deeply the theoretical complexity that such frameworks demand, often leaving unanswered the important question of whether and why any observed race effects vary across other social group categories. The limited body of epidemiologic work grounded in Intersectionality Theory indicates that race can be further divided into meaningfully disparate categories with important implications for accurately assessing health and health disparities. Yet, Intersectionality Theory, as it is frequently applied, is only one lens with which to appraise disparate health outcomes at these social junctures. This chapter provides an overview of current evidence for racial differences in health, which vary across gender, building support for the necessity of wholistic identity approaches that move beyond current understandings of Intersectionality Theory.

Keywords: Intersectionality Theory, health disparities, gender and race, identity pathology, social group identity, social determinants of health

1. Introduction

1.1 Brief overview of health disparities research in the United States

Throughout the history of the United States, disparities in health outcomes between racial groups and individuals of differing ethnic backgrounds have been well documented [1, 2]. Consistently, black and Indigenous persons, and those of Hispanic ethnicity have had poorer overall health, higher rates of both chronic and infectious disease, and increased risk of mortality compared with persons of European ancestry [1–4]. For decades, investigations into the causes of these unequal health outcomes largely operated under an implicit—and at times explicit—biological determinism framework [5]. Because of this narrow theoretical scope,

important contributors—the most important contributors, one could argue—to these racial and ethnic gaps in health status were often overlooked in epidemiologic research [5].

Even as epidemiologists increasingly consider the causal role of the social conditions in which individuals live and work [1, 2, 4, 6], a lack of well-developed theoretical context to health disparities research frequently yields data, results, and interpretations that obfuscate the complex mechanisms underlying social group disparities in health [7]. Wide-spread assumptions of racial homogeneity [8, 9], for example, echo biological essentialism, masking important within-race gender or socioeconomic differences in disease risk, pathogenesis, prognosis, and treatment efficacy, even in those studies which acknowledge social determinants of health.

Theory emerging from the social science and social psychological disciplines is often borne of extensive grappling with these complex causal webs. Intersectionality Theory [10] and Multidimensional Identity Theory [11] are among many theoretical frameworks which outline compelling social and psychological explanations for disparate health outcomes along what are frequently conceptualized as "dimensions" of identity—race, gender, socioeconomic status, and other social group designations. From studies of genetic risk, heath service utilization, and health behaviors to the health-impact of identity processes and coping responses, research grounded in these intersectional theories indicates that socially constructed categories such as race can be further divided into meaningfully distinct categories such as "gendered race" with important implications for accurately assessing the causes of, and solutions for, health disparities [12–14].

Despite being one of the most increasingly used social theories in epidemiologic research, much of the theoretical nuance of Intersectionality Theory is often lost in application. The frequent oversimplification of theory manifests, in part, as a growing trend in efforts to decompose the relative contributions of intersecting exposures such as race and socioeconomic status [15]. Aside from the uncertain utility of such findings in addressing social group disparities in health, these methods reflect an interpretation that is in many ways counter to the central claims of Intersectionality Theory—that the effects of such exposures can somehow be separated [16]. The relationship between epidemiologic research and social psychological theory is even more tenuous. Very few studies even consider the population-level health implications of internal identity processes, whose effects on health can in many ways be even more difficult to capture than the consequences of compounded external social processes such as racial, gender, and socioeconomic inequity.

To address the challenge of improving the utility of theory in understanding gender differences in health, I advocate for a conceptualization of social and psychosocial exposures that moves beyond "intersection" toward a "wholistic" identity approach. This approach emphasizes how the external social factors that shape health are experienced by individuals who not separately gendered, raced, or classed, but who each have a wholistic identity developed out of the unique social experience determined by these constructs which influences how external contexts are internally mediated and manifest in health. I argue that such an approach could circumvent the temptation of attempting to quantify the relative contributions of specific dimensions of oppression when far more integral to understanding social group health differences is characterizing the internal and external barriers and resources unique to different social groups. A wholistic identity approach not only aids in elucidating how the lived experience of one's social status as determined by a unique combination of race, gender, etc., affects health, but would also allow for a more ethical and scientifically sound conduct of epidemiologic research if employed with greater frequency.

1.2 Why gender and race?

A question that often arises in epidemiologic research drawing from Intersectionality Theory is how the selection for study of any two identity categories, frequently gender and race, can be justified under the concept of inseparability of identity. In this chapter, I use gender and race as one example to explore the validity of a wholistic identity approach in epidemiologic research. Importantly, of the many designations which demarcate social groups in the U.S., there is strong evidence that gender and race exert a substantial, unique influence on health [14, 17, 18]. Despite the ongoing debate of whether race or socioeconomic status contributes more to poorer health outcomes among black persons in the USA [3], the social categories of gender and race share a number of factors not characteristic of other social constructs.

First, they are arguably two of the most visible and socially reinforced demographic traits. Whether approaching from an external social resource framework or an internal identity framework, the readily recognizable nature of race and gender means that individuals are more frequently subject to interpersonal discrimination based on these characteristics than other types of discrimination [18–22]. Such experiences can have far-reaching consequences for health, from chronic physiological dysregulation associated with toxic stress [22, 23] to receipt of subpar medical care [23] and increased barriers to protective social factors such as stable, safe housing or occupations [24]. With the structurally rooted, pervasive nature of racial and gender discrimination, the visibility of race and gender ultimately yields an overrepresentation of black persons and women among the poor and disadvantaged [3]. In this way, regardless of the magnitude of the effect of socioeconomic status on health, race and gender are determinants of socioeconomic status and therefore can be conceptualized as further upstream on the causal pathway from structural to health inequity.

Secondly, many social psychological and anthropological theories of race, as well as feminist theory, argue that cues for race and gender are more prevalent in the social environment than cues for any other social designation [19, 25, 26]. That is, more cultural elements, whether dress styles or styles of worship, are racialized and gendered than given any other social group categorization. In the context of hierarchical racial and gender structures, the prevalence of these constant reminders of what constitutes blackness and whiteness, or femininity and masculinity, renders race and gender particularly influential on how susceptible individuals are to the health consequences of their groups' perceived inferiority or superiority [17, 18, 27–29].

As will be discussed further in the following section, the majority of theories rejecting biological determinism describe the central, causal role for structural inequity in the poorer health outcomes of racial and ethnic minorities and women [4, 10, 18, 20, 24]. Health outcomes can be neither ethically nor rigorously examined outside of the sociopolitical and sociocultural contexts in which the populations of interest are located. Arguably, in the U.S. as elsewhere, the long history of violence against racial and ethnic minorities and women is unapparelled in its pervasiveness and brutality [26, 30], violence executed with the express purpose of establishing and maintaining white and male supremacy [10, 26, 30, 31]. Given this history and the degree to which racial and gender violence became embedded within the structure of U.S. social institutions [10, 26, 30, 31], it can be reasonably inferred that race and gender will have a more measurable impact on the health outcomes attributed to such inequity than other social designations.

Throughout this chapter, I will use the increasingly popular term "gendered race", therefore, to reinforce the need for a wholistic identity approach in epidemiologic research on health disparities. The term captures the concomitant elements of

socially assigned gender and race categories that cannot be decomposed, neither within an individual's self-concept nor in the manner which social inequities operate to structure privilege and marginalization based on these characteristics.

1.3 Chapter goals

Far from being the first to advocate for the increased use of theory in epidemiologic research, the primary objective of this chapter is to argue for a wholistic identity approach that moves beyond concepts of intersecting social forces of oppression as determinants of health. Examining health disparities across gendered race groups through the rich perspectives emerging from the social science and social psychological disciplines, I contribute a novel interdisciplinary interpretation which underscores the need for considering both external social processes and internal identity processes in understanding and addressing the causes of gender differences in health. This chapter provides an overview of current evidence for gender differences in health which vary across race, outlining support for one wholistic identity framework, Identity Pathology theory, and its utility in the optimal execution of ethical epidemiologic research. The chapter concludes with recommendations for the inclusion of a wholistic identity approach in epidemiologic and statistical methods, as well as health intervention development.

It is important to note that the research explored in this chapter is based on cisgender identities (gender identities which are consistent with sex assigned at birth), and do not address how gendered race operates in the lived experiences of trans or gender non-conforming individuals to impact on health.

2. Theoretical frameworks for social group differences in health

2.1 Biologically driven vs. socially constructed differences in health

The distinction between biologically and socially defined categories, while rarely considered theoretically or analytically in epidemiologic research, is integral to understanding how the wholistic effect of gender and race on health extend beyond the individual contributions of either construct. Sex, a biological category, influences physiological processes through the accumulation of hormones, gene expression, and reproduction determined by the presence or absence of the X chromosome. Gender, a sociocultural category, informs identity concepts, exposure to and appraisal of stressors, behaviors, and access to care—all factors whose physiological significance is also dependent on the external contexts in which identity is experienced.

As sex and gender are often conflated in health research [32], it is all the more difficult to tease out the individual contributions of each to specific patterns of health, disease, and mortality, particularly given their interdependent nature. For example, testosterone, a naturally produced hormone is present on average at higher concentrations in males [33], is associated with aggressive behavior. Culturally designated masculinity often reinforces aggression in males [31], leading to increased production of testosterone [33]. In this way, the interplay between sex and gender renders efforts to disentangle their individual effects on population-level health differences particularly challenging.

Despite the difficulty of distinguishing health outcome differences attributable to gender and sex, focusing on eliminating those differences that are unnecessary—and therefore unjust—can serve as a useful target for epidemiologic research. Because gender is a sociocultural construct, gender differences in health are also

largely a function of the social meaning assigned to gender. These definitions carry value constructed by structural, institutionalized gender prejudice as well as cultural traditions that are heavily based in binary, hierarchical concepts of gender [19, 25]. The value assigned to gender is also dependent on other social designations such as race. In fact, perhaps contributing to persistent conflicting evidence on the magnitude and causes of gender differences in health is an assumption of consistency in gender effects on health across racial groups. Any efforts to clarify the causes of gender-related differences in health must necessarily engage the historical contexts in which these health-determining social designations are constructed. These analyses may yield additional, accessible intervention targets on gendered health disparities.

2.2 Social, anthropological, and social psychological perspectives on social group differences in health

A vast store of sociological and anthropological literature describes the racial, gender, and economic inequity inherent to the hierarchical social structure of the United States (e.g. [10, 19, 25, 26, 34–36]), as well as the ways in which such social environments are inextricably linked with health [1, 6, 7, 37, 38]. Intersectionality Theory [10], Ecosocial Theory [2], and the Environmental Affordances model [39] specifically emphasize the compound effect of multiple forms of structured inequity intersecting to influence the disproportionate distribution of social, material, and natural resources across dominant status and marginalized populations. Social dominance theory [40] further suggests that structured inequity is supported through "legitimizing myths", or consensually shared ideologies which position certain groups as beneficiaries of these health-impacting resources while also promoting narratives that influence the political practices which deprive other groups of access.

Social Identity [41] and Multidimensional Identity [11] theories exist in parallel with these frameworks, describing how the construction of social group identity, likewise informed by intersecting axes of structured oppression, designates advantage and disadvantage across social groups. Identity triggers, what the Jedi Public Health framework [42] terms the overt and covert cues embedded within the social environment which reinforce shared social ideologies, connect structural-level identity outputs with individual-level identity inputs. The Social Signal Transduction Theory of Depression [43] proposes specific ways in which these individual-level identity signals are transduced through psychoneuroendocrinological pathways that ultimately lead to disease. In this way, these frameworks each provide important but distinct elements of the larger machination by which structural inequity shapes the external social processes and internal identity processes that yield social group differences in health.

To truly engage these theories in a manner that is meaningful for researchers to understand the health significance of social hierarchies, the historical contexts which have defined concepts such as gender and race, and the ways in which these constructs become biology [38], must be carefully analyzed. Legal sanction of rape, physical assault, and other forms of wide-spread abusive behavior against women has deep historical roots in the U.S. as globally [18, 19, 30]. These practices are only one element of a social environment in which female persons have been subject to gender-based dehumanization so pervasive and persistent as to appear not only unavoidable but a product of nature [18, 19, 30].

Beyond sanctioning violence in ways that left little possibility for reprise or protection, legal disempowerment of women occurred in a variety of other ways. Among them include inheritance and land ownership prohibitions, and denial of voting rights. Historical analyses also reveal the ways in which the practice of

medicine has been used to subjugate female persons [44, 45], not in the least by employing psychiatric diagnoses to discredit resistance to oppressive social and cultural norms [44, 46]. Decades of research have accumulated substantial evidence [10, 18, 30, 34] that like black persons, women's exposure to violence, exploitation, and abuse by men while being simultaneously deprived of the physical, political, or legal means to defend themselves or prevent misogynistic violence has resulted in a greater propensity for appraising trauma through a lens of powerlessness.

The structural violence employed to enforce female subordination also conditions females to see violence committed against them as consequences of their own behaviors [19, 30], which can promote the learned helplessness that drives internalization. The widely practiced tradition of female denigration following experiences of gender-based violence increases the likelihood of females' perceptions of their own complicity in traumatic experiences, and likely contributes to the increased vulnerability to internalization observed in this group [18, 19, 30]. The sense of helplessness fostered by a lack of social and legal repercussions for males' physical and sexual aggression toward females, as well as pervasive denigrating responses to female victims of gender-based violence, has promoted internalization in a space where any outward expression of discontent might yield further abuse.

These experiences of gendered dehumanization carry compelling implications for gender differences in health. As has long been argued, there is strong evidence that socially reinforced gender hierarchies directly influence female susceptibility to internalizing psychopathologies like depression and post-traumatic stress disorder (PTSD) [18, 47]. Research highlights a key role for chronic exposure to negative circumstances, or "strain", in women's predisposition toward depressive symptoms [18, 19]. The source of this gender-specific strain is often identified in unique experiences associated with lack of social power as well as societal norms and expectations of women [18, 19]. Accordingly, the IP framework argues that the experience of being a female person in a male-dominated society [rather than solely inherent biological traits of the female sex], increases susceptibility to specific types of psychological and physical disorders associated with the physiological conditioning of subordinate status. Similar to the manner by which race becomes biology [38], so too, does gender become biology.

Growing literature on the role of inflammation in depression [48] supports this argument. The Social Signal Transduction Theory of Depression [43] offers a comprehensive framework for understanding the processes through which chronic stress associated with social identity threat can lead to depression. The framework outlines how inflammatory processes chronically triggered in response to social isolation, rejection, and marginalization stemming from subordinate social status can increase risk for several conditions, including rheumatoid arthritis, asthma, obesity, and depression. As a function of their subordinate social status, those who identify (and are identified) as female are therefore at increased risk for the identity threat which can cause sustained activation of the immune system's inflammatory response. In accordance with this theory, women and black persons are consistently found to have higher levels of inflammation than men and white persons, respectively [49, 50]. These racial and gender disparities in inflammation may underlie the increased risk for conditions such as depression, which has been increasingly linked to chronic inflammation [43, 48, 49, 51], among women (see **Table 1**). In conjunction with increasing psychological susceptibility through socialized helplessness, socially constructed subordinance can thus also act physiologically to increase female vulnerability to disorders that may be better classified as internalizing symptomatology.

While the association between subordinate status and toxic stress is well-documented, less attention is devoted to the impact of higher-status on the

Biomarker	Black women	White women	Black men	White men
Systolic BP	1.2 (0.6, 2.5)	1.1 (0.6, 2.0)	1.7 (1.1, 2.7)*	1.4 (0.8, 2.5)
Diastolic BP	1.1 (0.6, 2.1)	1.3 (0.8, 2.2)	1.2 (0.8, 1.9)	1.3 (0.8, 2.1)
Pulse	1.1 (0.7, 1.6)	1.5 (1.1, 2.2)*	1.2 (0.6, 2.4)	1.8 (1.1, 2.9)*
BMI	0.8 (0.5, 1.2)	1.1 (0.7, 1.7)	1.1 (0.6, 2.0)	0.9 (0.6, 1.3)
Total cholesterol	1.6 (1.0, 2.7)*	1.1 (0.8, 1.5)	1.0 (0.5, 2.0)	0.8 (0.4, 1.3)
HDL cholesterol	1.2 (0.6, 2.3)	1.1 (0.7, 1.7)	1.7 (0.9, 3.4)	1.3 (0.8, 1.9)
Glyco-hemoglobin	1.1 (0.8, 1.7)	1.0 (0.6, 1.7)	0.9 (0.5, 1.6)	0.8 (0.5, 1.4)
Serum Albumin	0.9 (0.6, 1.3)	1.0 (0.7, 1.6)	1.7 (1.0, 2.9)*	1.3 (0.7, 2.5)
CRP	0.8 (0.6, 1.1)	1.7 (1.1, 2.6)*	0.9 (0.5, 1.5)	1.8 (1.1, 2.8)*
High-risk AL[d]	1.1 (0.6, 2.0)	2.1 (1.5, 3.0)*	1.7 (1.0, 2.9)*	1.4 (0.8, 2.5)

Abbreviations: BP, blood pressure; BMI, body mass index; HDL, high-density lipoprotein; CRP, c-reactive protein.
[a]Models adjusted for PIR (ratio of household income to the US poverty threshold), age, and all biomarkers.
[b]PHQ-9 scores of ≥10.
[c]Results are from four separate regression models. The reference category for the biomarkers in each model is "low-risk".
[d]AL scores of ≥4 were considered "high-risk".
**p <0.05*

Table 1.
Adjusted[a] odds of depression[b] with high-risk allostatic load and biomarker levels by gendered race in National Health and Nutrition Examination Survey 2005–2010, OR (95% CI)[c].

experience of chronic strain. Still, previous research has identified stress correlates of perceived dominance, showing increased pituitary–adrenal responsiveness to psychological stressors in socially dominant males [52]. This limited evidence suggests that those in dominant positions can also experience higher levels of stress as a function of their status. However, chronic exposure to dominant-status stress likely acts to shape brain physiology and manifestations of identity-based trauma in ways distinct from the chronic stress generated by subordinate social status [53–55].

Where subordinate social status promotes internalizing disorders, dominant status promotes externalizing disorders characterized by antisocial behavior. Social dominance orientation (SDO) measures the degree of preference for inequality among social groups, a personality trait that negatively correlates with empathy, tolerance, and altruism [40], and promotes reduced activity in the brain regions associated with the ability to feel concern for the pain of others [53]. In studies measuring variation in SDO, both male and white persons were found to have significantly higher orientation, suggesting that white males are more likely than either female or black persons to both promote and subscribe to legitimizing myths (rape myths of victim culpability, for example) that enable justification of their dominance-reinforcing behavior [40]. Because high SDO also correlates with low empathy, it is likely that experiences of inequity among those whose race and gender are ranked as superior can yield a proclivity for antisocial behaviors. This propensity is manifest in gendered racial disparities in suicide risk, perpetration of rape, pedophilic child molestation, and mass violence, and risk for Anti-Social Personality disorder.

2.3 Beyond Intersectionality: identity pathology, a wholistic identity approach

Intrinsic to the concept of intersection is the existence of distinction that two distinct elements, at some point, intersect. This is the basis on which Intersectionality Theory is built. As described, the structural institutions that distribute

social resources do so along specific axes—inequitably, across races, genders, socioeconomic positions, abilities, sexual orientations, etc. Groups at the junctions of these axes are multiply advantaged or disadvantaged; disparities in health outcomes manifest at social intersections are, according to Intersectionality Theory, testament to the existence of a synergistic effect [10]. Internal identity processes that influence health in myriad ways, from stressor appraisal to behavior, have similarly been conceptualized in intersectional terms. The concept of dimensions outlined in Multidimensional identity theory also conjures an axial formulation of distinct identities. The intersection of these identities shapes how the external environment is experienced internally [11, 41], and subsequently, how social exposures impact on health.

A wholistic identity approach challenges the notion of both intersecting identities and intersecting axes of oppression. Intersectionality Theory calls attention to the locations where the distinct mechanisms of social hierarchies such as racism and patriarchy overlap to dictate social and health outcomes [10]. A wholistic identity approach instead argues that because racism is inherently gendered and sexism is inherently racialized, any effects of racism or sexism on health cannot be decomposed into distinct measurable units. The impact of either on the outcomes of individuals targeted by these systems therefore has less to do with the number of marginalized social group categories under which individuals fall and more so with the sociocultural paradigms unique to specific social groups which shape the internalization of, and response to, adverse social experiences. As such, while acknowledging the influence of social context and experience on the content and salience of specific identities, the argument put forth here is that individuals do not experience oppression in an axial fashion, but rather through a wholistic identity lens. That is, experiences of inequity are filtered simultaneously through each element of self-concept which predominates an individual's identity in a manner with direct implications for how such experiences will impact on health. While perhaps appearing problematically theoretical, this concept of wholistic identity as a determinant of health can be readily applied to improve current understandings of gender differences in health.

One newly emerging wholistic identity approach is the Identity Pathology (IP) model, an infectious disease framework for the effects of structural inequity on health. The triad paradigm of disease causation which sits at the foundation of infectious disease epidemiology describes interactions between an environment, a host, and a pathogenic agent [56]. The IP framework incorporates the three elements of the infectious disease triad to conceptualize the health-impacting interaction between structural inequity, individuals subscribing to socially constructed identities, and pathogenic identity beliefs. Conceptualizing identity beliefs as a pathogen that spreads through social interaction over time in a contagious manner to cause specific disease patterns across socially defined groups is an innovative approach to characterizing the causal pathways from structural inequity to disease. Contagion modeling of social determinants of health has been useful in explaining and predicting the effects of other social exposures, such as gun violence [57]. The IP model goes beyond identifying the contagious nature of socially driven health outcomes to directly characterizing the fundamental infectious elements underlying the spread of pathogenic social exposures.

According to IP theory, structural inequity serves as a breeding ground for the multilevel processes which yield unequal health outcomes. Through the construction and hierarchical organization of race and gender, as well as the disproportionate distribution of social and material resources across these categories, the ubiquitous nature of structural inequity lends itself to the nourishment of belief systems and associated behaviors which produce population-level disease patterns.

Transgenerational effects of trauma [58, 59] ensure that even prior to socialization, individuals are vulnerable to particular kinds of identity imprintation, making identity an ideal vector for pathogenic beliefs. Within an environment of structural inequity, what begins as an involuntary process of gendered racial socialization eventually leads to the development of identity paradigms capable of housing the pathogenic beliefs which infect and predispose individuals to various manifestations of pathology. The IP framework argues that the interconnectedness of social, psychological, neurological, and physiological processes renders every individual susceptible to the disruptive effects of identity on biological homeostasis. Whether through transgenerational epigenetic pathways [59], direct neurological pathways [43], or indirect behavioral pathways [60, 61], identity beliefs can chronically disrupt homeostasis and produce disease, a phenomenon which the framework terms *identity pathology*.

The IP model is distinct from Intersectionality theory in that it hypothesizes the concept of identity pathology, which describes a disease-prone state characterized by certain acquired beliefs about individual or group identity that are inherently pathological. Constructed in the context of structured inequities such as institutional gendered and classed racism, these identity beliefs are informed by unique experiences of individuals defined simultaneously by multiple social group designations and may partially account for the types of chronic diseases prevalent among different socially defined groups. The IP concept applies infectious disease modeling to the integration of the aforementioned theoretical frameworks in situating the adoption of socially constructed identities as a mediator of the disease patterns observed across different socially defined groups.

3. Evidence supporting the necessity of a wholistic approach to health disparities research

3.1 Current evidence supporting both intersectional and wholistic identity approaches

Extant epidemiological literature in accordance with an intersectional framework has identified gendered racial differences among black and white women and men in lung cancer treatment and mortality [12]; in the protective effects of income on depression [62]; in the association of depression with mortality [63]; and in the link between chronic stress and depression [64] among other exposure-health combinations. Yet, the results of these studies may also be viewed as evidence for the need of a wholistic identity approach.

The IP model builds on Intersectionality Theory in asserting that gendered racialization yields identity pathologies distinct to different gendered race groups, even among those not dually marginalized. Social hierarchies act to create unequal access to health-impacting resources, but it the convergence of each element of an individual's wholistic identity that accounts for the unique manifestations of disease caused by identity pathology across different social groups. In this section, I present evidence for the concomitantly protective and harmful effects of these gendered race-specific identity pathologies that can partially explain patterns of disease observed across gendered race groups in the United States.

For the purposes of demonstrating the application of IP theory to the epidemiology of gendered racial health disparities, I use the example of the socially constructed identity which has been the focus of this chapter thus far, gendered race, among four groups who occupy different tiers within a historically grounded social hierarchy: U.S.-born black and white women and men. Taking a snapshot of the pathologies (which fit accepted notions of disease) endemic to each of these

groups at middle age (45–55), we see that black women are more likely to be afflicted by cardiometabolic conditions such as obesity and uncontrolled hypertension than the other groups [65]; black men, prostate cancer (compared with white men) and cardiovascular disease [65–67]; white women, prescription opioid abuse and depressive symptomatology (MDD) [17, 68]; and white men, alcoholism and suicide [17, 69, 70]. The IP framework asserts that these disease patterns result directly from the ways in which each of these group experience identity pathology as dictated by their gendered race.

Most individuals are categorized as possessing at least one privileged and one marginalized identity. Queer or poor white men, for example, experience discordant social identities, as heteronormativity and classism rank these statuses as inferior [19, 71] even as their race and gender grant certain privileges. However, as previously noted, the immutable physical attributes assigned to gender and race lend a permanence and identifiability that make social processes particularly susceptible to discrimination based on these characteristics. Gendered racial identities are therefore particularly influential in shaping the manifestation of identity pathology.

As the theories covered in this chapter outline, female and black persons occupy subordinate social positions in the U.S. gendered racial hierarchy. Those who are both female and white, or male and black, however, occupy both subordinate (female and black) and dominant (white and male) positions and therefore can experience a particular kind of dissonance associated with simultaneous disempowerment and privilege. Because of this incongruence between the socially constructed racial and gender identities of white females and black males, these groups likely share underlying identity pathologies distinct from those of black women and white men. However, sociocultural influences as well as the influences of other centralized identities on coping can lead to distinct manifestations of identity pathology even among white women and black men.

Shared identity pathologies in which self-worth is predicated on an unattainable, but desired social status underlies prevalent diseases among white women and black men. The increased cardiovascular disease risk (as well as other chronic inflammatory diseases like prostate cancer) [72] in black men has been shown to correlate with John Henryism, a type of goal-striving stress caused by a refusal to succumb to racial or economic barriers to the practice of a socially defined masculinity among members of this group [73]. Similarly, the IP model asserts that white women's increased risk for inflammatory-based internalizing disorders such as depression are caused by an increased likelihood of self-blame and denial of social inequity. As the social value granted by whiteness is diminished through gender marginalization, opioid addiction becomes a method of avoidant coping consistent with the socialized internalization of female persons.

In this manner, black men and white women can perceive similar barriers to the benefits of their advantaged social positions. For some black men, racism prevents the full practice of socialized concepts of masculinity, leading to social deprivation, identity threat, and the cognitions and health behaviors that increase risk for cardiovascular disease [27, 73]. Likewise, for some white women, gender discrimination impedes access to the full perceived benefits of whiteness, leading to social marginalization, identity threat, and the cognitions and health behaviors that predispose members of this group to inflammatory-based depressive disorders. These hypotheses are supported by emerging evidence of a link between cardiovascular disease and depression [48, 74, 75], making a case for the assertations that (a) black men and white women may share identity pathologies that manifest distinctly based on sociocultural contexts, and (b) ostensibly dissimilar symptoms of illness may stem from shared disease origins [49, 76].

The identity pathologies of white males and black females differ from those of black males and white females, and present with different symptomatology. White males occupy both racially and gender superordinate social positions, and are therefore more likely to be socialized to adopt identity paradigms which rely primarily on socially constructed relational self-worth. Without exposure to the subordinate status that conditions adoption of identity paradigms embedded with increased risk of internalization and chronic inflammation, white males are more likely to exhibit symptoms of recurrent identity stress through externalized control-reinforcing behaviors, which have been discussed at length. Subscribing to socially constructed white male identity paradigms not only increases susceptibility to anti-social tendencies attributable to pathologized whiteness, including lack of empathy, feelings of entitlement, and behaviors to reinforce feelings of control, but also externalizing disorders driven by pathogenic masculinity that manifest through violent or aggressive behavior toward self and others. As the practice of white male identity also requires the perpetuation of the structural violence that enables members of this group access to a disproportionate share of social and material resources, white males subscribing to these identity paradigms are also less susceptible to the physical disorders such as cardiovascular disease promoted by material deprivation.

On the other hand, dominant narratives of white and male identity which distance whiteness from poverty [71] ensure that certain groups of white men are particularly susceptible to the health consequences of identity pathology. With increased dependence on superior status for a sense of self-worth [17, 28], not being afforded the expected privileges of white male membership can exacerbate the negative health effects of poverty. Poor white men, for example, face increased risk of depression, and substance abuse may serve as a form of coping [17, 77] for those white men not succumbing to other self-destructive compulsions of identity pathology such as suicide [17, 77, 78].

In contrast, the dual occupancy of subordinate social positions may reduce the risk of psychopathology among black women, while conferring an increased risk for cardiometabolic disorder. As both female and non-white, their dually marginalized positions might predict that black female identity paradigms manifest a propensity for disorders such as depression as an expression of chronic stress. Identity pathology theory, however, contrasts the external social circumstances in which black women are situated with the internal resources characteristic of this group. The necessity for adapting to multi-faceted forces of structural violence may have enabled the development of psychological durability within black female identity paradigms that is protective against psychological symptoms of toxic stress. So, while occupying both racially and gender subordinate tiers might predict higher risk of psychological manifestations of depression among black women, the greater necessity for the development of effective coping strategies may actually act to confer psychological resilience and reduce risk in this group [64, 79].

Not permitted access to social privilege or higher social status as a result of their race or gender, evidence suggests that black women have been compelled to develop alternative standards of value in order to build self-worth. In this way deprived of access to sources of socially constructed self-esteem, black women subscribing to dominant black female identity paradigms are likely to appraise potential identity threats in a manner distinct from other groups. Specifically, acute, interpersonal experiences of identity threat may be perceived as less threatening. Previous research grounded in IP theory indicates, which will be later examined in more detail, that reported lifetime gender and racial discrimination in certain settings is associated with poorer cardiovascular health among black men, white women, and white men, but not black women (**Table 2**) [80].

Setting[c]	Black women	Black men	White women	White men
In public/on the street	+0.2 (0.0, +0.5)*	0.0 (−0.3, +0.3)	+0.1 (−0.2, +0.5)	−0.5 (−1.0, −0.1)*
Getting a job	0.0 (−0.3, +0.3)	−0.3 (−0.6, 0.0)*	−0.6 (−1.3, +0.1)	−0.5 (−1.1, +0.1)
Getting housing	−0.1 (−0.3, +0.2)	−0.2 (−0.5, +0.1)	−1.5 (−2.5, −0.4)*	−0.4 (−1.5, +0.7)
At work	+0.1 (−0.2, +0.4)	−0.4 (−0.7, −0.1)*	−0.4 (−0.9, +0.1)	−1.0 (−1.6, −0.3)*
At school	+0.3 (0.0, +0.6)*	−0.4 (−0.8, 0.0)*	−0.1 (−0.6, +0.5)	−0.3 (−1.1, +0.4)
Receiving medical care	−0.5 (−0.9, −0.1)*	−0.7 (−0.9, −0.1)*	−1.5 (−3.8, +0.7)	−1.1 (−2.7, +0.5)
By the police or courts	−0.1 (−0.2, +0.4)	−0.1 (−0.4, +0.2)	−1.1 (−2.0, −0.3)*	−0.3 (−0.9, +0.4)
At home	+0.1 (−0.3, +0.4)	−0.1 (−0.6, +0.4)	−0.2 (−0.5, +0.1)	−0.2 (−0.7, +0.3)

[a]*All models are adjusted for age and study center.*
[b]*Health scores are calculated based on data collected in year 30 or the last follow-up after year 7, using six components with a total possible 12 points: body mass index, total cholesterol, systolic blood pressure, fasting glucose, smoking status, and physical activity. Higher scores indicate better health.*
[c]*At year 7, discrimination "at home" was excluded from the race or color scale; "by the police or courts" and "getting housing" were excluded from the gender scale.*
*$p < 0.05$

Table 2.
Adjusted[a] difference in cardiovascular health Score[b] at year 30 of the CARDIA study across settings of simultaneously reported racial and gender discrimination at year 7, ß (95% CI): 1992–2016.

This psychological resilience among black women may be grounded in an ability to redefine standards of value in a manner that challenges the very notion of socially constructed subordinance. Contrary to what many psychological theories once predicted, members of stigmatized groups tend to have comparable levels of self-esteem with non-stigmatized groups [81]. Researchers attributed these surprising findings to the use of self-protecting mechanisms by members of stigmatized groups such as "selectively devaluing, or regarding as less important for their self-definition, those performance dimensions on which they or their group fare(s) poorly, and selectively valuing those dimensions on which they or their group excel (s)" [81]. Dominant sociocultural narratives rank black women at the bottom in most highly regarded social dimensions—physical beauty, intellectually capability, etc. [82], but celebrate their caregiving, selfless, mothering natures [83, 84]. However, rather than devaluing the dimensions in which society ranks them poorly as identity stigma predicts, many black women appear to have developed alternative social rating systems which do not predicate socially valued traits on dominant group standards [79, 82].

Furthermore, their professions, voting patterns, and activism demonstrate that black women have identified a source of self-worth inherent to the practice of caring for and about others [85]. In this way, by enabling a greater sense of self-efficacy in which black women feel capable of determining for themselves standards against which their value will be measured [82], multifaceted forces of disempowerment may confer individuals subscribing to dominant black female identity paradigms a measure of protection against the psychological manifestations of the very chronic identity threat they cause.

In support of this hypothesis, research demonstrates that allostatic load, a measure of cumulative physiological dysregulation stemming from chronic stress that precedes and correlates highly with many chronic diseases [86], is associated with depression among black men and white women, but not black women or white men

(**Table 3**) [64]. Furthermore, there is indication that the underlying neurobiology of depression differs among black women compared with black men, white women, and white men (**Table 1**) [49, 87]. The depressive response to deprivation among black women, rather than being a function of a perceived threat to deeply held self-concepts that promotes sustained inflammation, as IP theory argues is more likely to be the case among black men and white women, may be based more in a situation-appropriate response to the uniquely disadvantaged social conditions in which black women are disproportionately situated. So, while the prevalence of depressive symptoms may be substantial among black women, these symptoms may be indicative of a response that is distinct from the pathology manifest in depressive symptoms among other groups. Evidence that adjusting for socioeconomic status eliminates the gender disparity in depression among black persons but not white [88] further supports this theory.

These potential psychological benefits do not come without physical costs, however. Where black women may be psychologically resilient, they are likely to be physically vulnerable; high rates of obesity, hypertension, and poor maternal/neonatal outcomes in this group reflect a unique adaptation to structural inequity—metabolically, rather than psychologically, exhibiting pathology. In addition to the structural racism and sexism that concentrates economic deprivation and limits the capacity for health-promoting behaviors within black female populations [10, 82, 85], black female identity paradigms demand what could be argued as a pathological minimization of self-care in efforts to be valued as caregiver [84, 89]. As Superwoman Schema theory suggests, in prioritizing the needs of others, black women often bear an extensive familial and community burden without complaint at the cost of their own emotional and physical needs [84]. Adherence to these gendered race-specific identity paradigms predisposes black women to automated coping such as emotional eating [90, 91], other risk-factors for obesity such as postpartum weight retention [92], and other health-impacting behaviors such as low health services utilization [93]. Furthermore, another form of identity pathology characterized by a failure to acknowledge the existence, or negative psychological impacts, of structural inequity can lead to denial and internalization which may lead to premature disease and mortality [29, 94].

	All (*n* = 6431)	**Black women (*n* = 980)**	**White women (*n* = 2147)**	**Black men (*n* = 1028)**	**White men (*n* = 2276)**
Depression, % (SE)	7.3 (0.5)	14.6 (1.3)	8.5 (0.7)	7.1 (0.8)	4.9 (0.6)
Low AL[b] (0–3)	6.1 (0.5)	13.4 (1.4)	6.9 (0.7)	6.1 (0.9)	4.3 (0.6)
High AL (4–9)	11.8 (1.2)	17.1 (3.0)	15.3 (1.9)	10.1 (2.0)	7.4 (1.5)
Crude	2.1 (1.6, 2.7)**	1.3 (0.8, 2.2)	2.4 (1.7, 3.4)**	1.7 (1.0, 3.0)*	1.8 (1.0, 3.0)*
Adjusted[c]	1.7 (1.3, 2.2)**	1.1 (0.6, 2.0)	2.1 (1.5, 3.0)**	1.7 (1.0, 2.9)*	1.4 (0.8, 2.5)

[a]*Results are from five separate logistic regression models; one for the total sample and one for each gendered race group.*
[b]*Allostatic load, calculated as a composite of nine cardiovascular, metabolic, and immune biomarkers.*
[c]*Adjusted for five age groups and five groups of ratio of household income to the US poverty threshold.*
*$p < 0.05$.
**$p < 0.0001$.

Table 3.
Depression in relation to Allostatic load by gendered race Group[a] among Black and white US adults: National Health and Nutrition Examination Survey, 2005–2010.

Importantly, the IP framework does not assert that compounded inequities necessarily translates to greater likelihood of a specific disease outcome among multiply marginalized groups. Instead, the framework argues that the lived experience of race and gender in a society which advantages some groups in certain ways while disadvantaging others in different ways [12, 95] based on these identities yields variation in the efficacy of health-protective factors. This variation in turn manifests as a differential vulnerability to disease across gendered race groups.

3.2 Application of IP theory to investigating gendered racial differences in cardiovascular health

Recently published work applies the IP framework to the study of gendered racial variation in the association of discrimination with cardiovascular health (CVH). This emerging body of research makes a compelling case for considering the role of wholistic identity in assessing the manner by which structural inequity contributes to unjust and unnecessary gender differences in health. Persistent gendered racial differences in the prevalence and severity of cardiovascular disease (CVD) in the U.S. highlight the necessity for a stronger theoretical foundation in understanding the role of discrimination in yielding social group disparities in CVD [21, 22, 80].

The age-adjusted likelihood of a CVD diagnosis is approximately equal for black and white men [65, 66], but black women are nearly twice as likely as white women in the same age group to develop CVD [65, 66]. Black women are also more likely than white women or black men to develop cardiometabolic precursors to CVD [96]. Among other risk factors [7, 10], researchers frequently attribute this increased risk among black women to a greater likelihood of experiencing racial and gender discrimination [12, 95]. Unlike the large gender disparity among whites, however, black women and men report comparable exposure to interpersonal gender and racial discrimination [21, 97] even as black men develop CVD at a faster rate than black women [65, 66].

Due to these prominent disparities in cardiovascular outcomes between black and white women and men, researchers have examined social group-specific exposures as potential contributors to these inequities [98]. Consistent with the dominant biomedical, individual-level orientation of epidemiological research [7], the literature has largely focused on interpersonal racial discrimination as a driver of poorer CVH within these groups [7, 22, 98, 99]. Often conceptualized as a proxy for structural discrimination, or, alternatively, as a mechanism through which structural discrimination acts on health, interpersonal discrimination provides an accessible method for investigating social determinants of health [7]. The underlying assumption for the majority of studies examining interpersonal discrimination appears to be that the stress associated with experiencing discriminatory interactions has a detrimental effect on CVH, directly through chronic activation of the stress response system, or indirectly through promoting poor health behaviors, which in turns increases risk for cardiovascular morbidity and mortality [7, 22, 100]. As such, populations more likely to encounter these experiences (e.g. women compared with men in the case of gender discrimination) will exhibit poorer health behaviors, experience higher rates of cardiometabolic dysfunction, and necessarily have a greater burden of disease.

In line with this reasoning, previous studies have linked reported racial discrimination to sedentary behavior, smoking, hypertension, obesity, and incident CVD within black and white populations [21, 100–103]. Because the prevalence of reported interpersonal racial discrimination is substantially higher among black persons than whites [7, 23] these findings have generally been interpreted through

the lens of differential exposure rather than vulnerability [16]. That is, a higher prevalence of disease theorized to correspond with a higher prevalence of exposure, rather than with differential vulnerability to the effects of exposure [7, 99]. Consequently, consensus has leaned toward an association of reported racial discrimination with the disproportionate rate of cardiovascular morbidity and mortality among blacks [7, 98, 99].

Admittedly, researchers have emphasized relevant differences in the effects of exposure depending on the basis of discrimination [98, 102] (racial versus weight, for example), the frequency of discrimination [98], demographic characteristics such as the age or gender of the individual to whom the discrimination is directed [103], and how individuals respond to stress [21, 104]. Even still, few have theoretically considered the nature of these differences and whether the reasons for these differences have implications for the exposure-disease relationship; even fewer have taken these potential implications into account during analysis.

Further, while the consequences of structural and interpersonal discrimination are documented more frequently among women [20, 95], recent evidence showing no association of reported gender discrimination with incident CVD [21], along with other recent findings inconsistent with previous evidence [94], calls into question unidimensional conceptualizations of discrimination as a cause of poorer CVH. A focus on differential exposure to interpersonal discrimination as underlying gendered racial disparities in CVH may prevent identification of other relevant group-specific characteristics such as varying *susceptibility* to the health effects of perceiving discrimination [7, 12, 62, 63, 105]. For example, a recent study assessing the effect of cumulative unfair treatment on subclinical CVD among a multi-ethnic sample of women found an association only among white women [106]. Such evidence supports the argument that while women and black persons are more likely to experience both structural and interpersonal gendered racial discrimination, men and white persons may be more susceptible to the health consequences of perceiving interpersonal discrimination as a result of group-specific internal resources [29, 80, 107].

Previous findings also suggest that the magnitude of stress discriminatory experiences cause and whether responses to these experiences exacerbate or reduce the risk of CVD depends on the context in which they occur [100, 104, 106]. Therefore, in addition to the challenge of capturing variation in the subjective identity characteristics that might render interpersonal discrimination detrimental to CVH, as well as the complex psychological processes by which individuals attribute discriminatory experiences, it is also necessary to consider how the setting in which discrimination is reported reflects access to both internal and external psychosocial resources that may independently relate to CVH differently for different gendered race groups. Everyday experiences of discriminatory treatment not only encompass individual acts but also the complex relation of acts that will be specific to specific social contexts, as argued by some critical race theorists [12]. The particular relationship between individual and context bears important implications for the physiological impact of perceived discriminatory interactions.

Further, the context of reported discrimination, such as at school, at work, by the police or courts, or while seeking healthcare, may provide insight into distinct effect pathways operating among different gendered race groups. While discrimination may act directly on CVH through repeated activation of the stress response system for some, others may be more susceptible to the indirect effects of interpersonal discrimination such as barriers to quality health care [98, 100, 108].

The complex relationships of these psychosocial exposures with CVD among black and white women and men connoted in the literature point to a need for further consideration of how and in whom discrimination operates to affect risk for disease [7].

Evidence suggests that the multifaceted nature of the interpersonal discrimination experience operates within distinct social groups to differentially influence CVH in a manner not frequently captured in epidemiologic studies [22, 98, 99, 103]. Inconsistencies in the literature may be attributable, in part, to an inadequate conceptualization, measurement, and analysis of interpersonal discrimination in relation to CVH across demographically diverse populations.

The Identity Pathology framework provides a useful model for investigating these inconsistencies in the relationship of discrimination with CVD (see **Figure 1**). While not solely applicable to CVD, the model is useful for clarifying inconsistencies in the literature on interpersonal discrimination and CVD because it specifies the conditions under which—and in whom—reported experiences of interpersonal discrimination will be measured as damaging to CVH and lead to the development of disease. As applied to CVD disparities and interpersonal discrimination, the model makes three central assertations.

First, that in order to more accurately capture the effects of interpersonal discrimination on cardiovascular health and health disparities, multiple aspects of the discrimination experience must be considered in the design, analysis, and interpretation of health-related studies. Secondly, the IP framework posits that experiences of interpersonal discrimination are fundamentally based in historically structured inequities that impact on each dimension of the discrimination process in health-relevant ways. Finally, the model purports that the precision with which reported experiences map onto perceptions and intentionally or implicitly driven acts of discrimination depend on a variety of psychosocial characteristics, one of the most important of which is an individual's beliefs about their gendered racial identity. In other words, the contribution of discrimination to disparities in CVH may extend beyond gendered racial variation in *exposure* to gendered racial differences in the effect of perceiving interpersonal discrimination.

The IP model argues that this variability in effect across gendered race groups can be attributed to differing manifestations of identity pathology. Due to the relationship between identity pathology and the experience of interpersonal discrimination, the experience being captured in reported discrimination among different gendered race groups must necessarily be different. For men reporting frequent experiences of gender discrimination, these experiences are less likely to reflect objective encounters with discrimination as traditionally conceptualized and are more likely to signify that these men feel they are being deprived of the

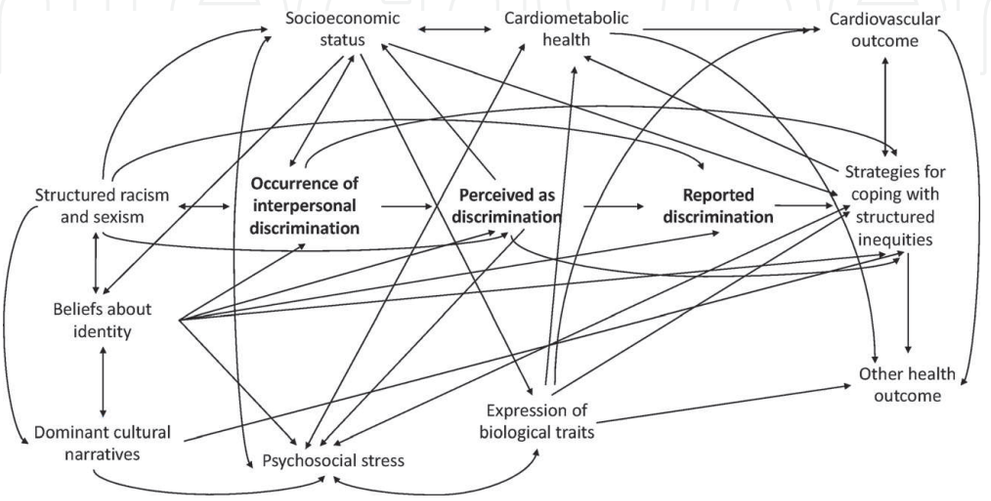

Figure 1.
Application of the emerging identity pathology framework to describe potential pathways from intersecting axes of structured racism and sexism to cardiovascular disease.

entitlements they believe they are due as a result of their manhood. Similarly, reporting of multiple encounters with racial discrimination by white persons likely indicates encounters in which these individuals believe they were deprived of entitlements due to them as white persons. Regardless of the accuracy of their reporting, the perception of what members of dominant status groups consider discrimination can be stressful enough to have a measurable impact on their cardiovascular health. This effect may be exacerbated by their recognizing the inconsistencies of their perceptions with the way that society defines experiences of discrimination.

Moreover, even among those whom the occurrence, perception, and reporting of discrimination overlap with high accuracy, differences in beliefs about the significance of being perceived and treated as inferior by another group will influence the stressfulness of perceiving discrimination. Finally, identity beliefs associated with gendered race also shape how individuals will cope with the reality of being perceived and treated as inferior, thereby creating another source of variability in the effect of reported interpersonal discrimination on CVH. Because increased exposure to social stressors among marginalized groups may yield an array of adaptive coping strategies that are protective against the health consequences of psychosocial adversity, the IP model predicts, perhaps counterintuitively, that the association between reports of racial and gender discrimination and declining CVH to be stronger among members of dominant status groups. The IP framework also posits that the susceptibility to direct versus indirect effects of discrimination on CVH are primarily a function of an individual's cumulative social experiences and will therefore manifest differentially across gendered race groups.

Given these hypotheses, two studies [29, 80] examined the relationship of reported interpersonal discrimination with CVH among black and white women and men using 30 years of longitudinal data from the Coronary Artery Risk Development in Young Adults study. The first study evaluated whether the associations of reported interpersonal experiences of racial and gender discrimination simultaneously compared with racial or gender discrimination alone, or no discrimination, with cardiovascular health 23 years later was stronger among white men than other groups. The second study explored variation in the relationship between simultaneously reported racial and gender discrimination and future CVH across eight social settings.

The studies identified important characteristics of the relationships between reported racial and gender discrimination and cardiovascular health (CVH) in black and white women and men. The first study identified differences in the associations between reported gender and racial discrimination and CVH, suggesting differential vulnerability (**Table 4**). Compared with reporting no discrimination, reporting any racial discrimination predicted higher CVH scores among black women, while no statistically significant associations were found among black men. Among white women, reporting any gender discrimination predicted higher CVH scores than reporting no discrimination. For white men, predicted CVH scores were higher for those reporting any racial discrimination, and lower for those reporting racial and gender discrimination in at least two settings, than in those reporting no discrimination.

These findings contrasted with those describing a link between racial discrimination and poorer cardiovascular health among black persons [7, 99, 101]. Though inconsistent, the literature has demonstrated associations of reported racial discrimination with CVD risk factors including diet, hypertension, smoking, sedentary behavior, obesity, and inflammation [50, 101, 103, 109], as well as social predictors of CVD such as marital status, socioeconomic position, and education, in both black women and men [7, 23]. In this study, we did not find a statistically significant association between racial discrimination and poorer CVH within these groups.

	Black women	Black men	White women	White men
Discrimination (year 7)	ß (95% CI)	ß (95% CI)	ß (95% CI)	ß (95% CI)
None	ref.	ref.	ref.	ref.
Any racial only	0.4 (0.0, 0.8)*	−0.1 (−0.5, 0.4)	−0.3 (−1.2, 0.6)	0.4 (0.1, 0.8)*
Any gender only	−0.3 (−0.8, 0.2)	0.2 (−0.6, 1.0)	0.3 (0.0, 0.6)*	0.0 (−0.4, 0.3)
Any racial or gender, in <2 settings	0.1 (−0.3, 0.5)	0.0 (−0.5, 0.5)	0.2 (−0.2, 0.6)	−0.2 (−0.6, 0.1)*
Both racial and gender, in ≥2 settings	0.2 (−0.1, 0.6)	−0.3 (−0.7, 0.1)	0.0 (−0.4, 0.4)	−0.6 (−1.1, −0.1)

*$p < 0.05$.
[a]*Cardiovascular health scores are calculated based on data collected in year 30 or the last follow-up using six components: body mass index, total cholesterol, systolic blood pressure, fasting glucose, smoking status, and physical activity. Higher scores indicate better health.*
[b]*Models are adjusted for age and geographic location.*

Table 4.
Adjusted difference in cardiovascular health Score[a] for categories of reported racial and/or gender discrimination by gendered Race[b]: CARDIA, 1992–2016.

Other cross-sectional analyses [100, 110] and the only study prospectively examining the relationships of racial discrimination with incident CVD exclusively among black women and men have also failed to find a connection [94]. Taken together, these findings offer evidence that traditionally accepted risk factors may be poorer predictors of CVD among black persons. Accordingly, while interpersonal racial discrimination may increase the likelihood that black women and men develop cardiometabolic risk factors for CVD, other factors integral to the experience of multiply marginalized identities may have a much more substantial impact on the development of CVD in these groups. As these other potential risk factors remain under studied [7, 95], the long history of investigating interpersonal discrimination as a cause of poorer health has done little to expand an understanding of CVD disparities between black and white women and men.

In addition to suggesting alternative causes of higher CVD morbidity and mortality among marginalized groups, the IP model theorizes that discrepancies between the occurrence, perception, and reporting of interpersonal discrimination contribute to the observed variability in the associations of reported racial and gender discrimination with CVH among black and white women and men (see **Figure 1**). The model suggests that for some gendered race groups in certain places and settings, reported discrimination is more likely to reflect interactions that meet objective standards of inequitable treatment. In these cases, acknowledging experiences that actually occur may be beneficial for health, while denying may lead to increased stress and stress-related pathology regardless of one's gendered race group [50, 111]. From building social networks based on shared experiences to enabling the development of healthier coping behaviors [27, 109], recognizing and acknowledging the discrimination one encounters may allow for chronic stress relief that reduces risk for CVD associated with discrimination exposure [50, 111]. Reported experiences of racial and gender discrimination may thus be measured as protective among those against whom such experiences actually occur.

To fully account for the results of this study in the context of IP theory, it is important to note that across the four gendered race groups, reporting or not reporting exposure likely signify different health-relevant psychological and

emotional states [27, 112]. The relatively low percentage of black women who reported experiencing no racial or gender discrimination did so despite a considerable body of evidence to the contrary, indicating a measure of denial or "tough it out" mentality in this group [27] distinct from the evidence-based reasons that a much greater proportion of white men would report no exposure. Even within gendered race groups, the meaning of reported exposure to discrimination may vary. As proposed in the IP framework, white men reporting few experiences of racial discrimination may subscribe to identity paradigms distinct from those in their group reporting both racial and gender discrimination in multiple settings. The framework posits that among white persons, reported experiences of racial discrimination in only one setting (e.g. at school) may be more likely to meet objective standards of discriminatory treatment. Accordingly, better CVH scores among white men who reported only racial discrimination would not be inconsistent with a protective effect of reporting interpersonal experiences of discrimination that meet objective measures. That is, white men who reported only exposure to racial discrimination were likely the white men for whom the overlap of the occurrence, perception, and reporting of discrimination was relatively accurate. As the IP model predicts, in such cases, there is likelihood that reported discrimination will be measured as protective of CVH. That the positive effect on CVH among white men reporting only racial discrimination persisted even after adjusting for SES further supports this assertation.

Study 2 revealed that simultaneously reported racial and gender discrimination were differentially associated with CVH depending on gendered race and setting (**Table 2**). Among black women, with one exception, reported instances of interpersonal discrimination were not associated with CVH or were associated with a higher CVH score while the opposite findings were observed among the three other gendered race groups. For black men, simultaneously reported discrimination in four of the eight settings was significantly associated with poorer CVH. Associations across settings also differed between white women and men. For white women, reported racial discrimination by the police or courts or while seeking housing was associated with lower CVH scores, while among white men, self-reports of racial and gender discrimination in public or at work were associated with a lower CVH score. For all groups, reporting discriminatory experiences while receiving medical care had a negative impact on future CVH, although effect estimates did not reach statistical significance among white women and men.

That the settings in which reported racial and gender discrimination were associated with poorer CVH differed among black and white women and men is consistent with disparate effect pathways for these groups that may be linked to gendered race-specific external and internal characteristics. The findings suggest that for black women, interpersonal experiences of discrimination are more likely to act indirectly on CVH by deterring access to health-influencing resources such as medical care, a mechanism that has been demonstrated in previous research [113]. Black women who reported racial and gender discrimination while receiving medical care were the only individuals of their gendered race group to experience a decline in CVH associated with reported discrimination; reported exposure in other settings was measured as either protective or had no influence on CVH. Rather than yielding a greater vulnerability to the negative health consequences of psychosocial stress as might be intuitively concluded, these findings suggest that black women may more readily adapt to hostile social environments such that the effects of recurrent interpersonal discrimination on the stress response system [64], or on certain health-related behaviors that preempt cardiovascular disease [103], are minimized in comparison to other gendered race groups. These results do not suggest that black women are immune to the physiological impacts of the

discrimination they report. Rather, these findings indicate that structural barriers, such as reduced access to high-quality medical care, may have a much more compelling effect on the cardiovascular health of black women than stress stemming from encounters with interpersonal discrimination, as has been previously argued [4, 98].

The settings in which reported discrimination impacted CVH among black men in this study indicate that members of this group may be more susceptible to the direct physiological impact of perceived subordinate status than black women. This may be because racism targeted at black men has historically been more ostensibly violent [97], or due to other psychosocial and cultural factors influencing the distinct coping methods of these groups [10, 64, 97]. The observed patterns in the associations of reported discrimination with CVH indicate that reminders of marginalized status may be experienced as more stressful among black men than black women and therefore may be more likely to act on CVH through direct physiological mechanisms in addition to creating barriers to health and social resources in this group.

One explanation for the patterns observed in this study is that interpersonal discrimination may act as an "identity trigger" consistent with claims of the Jedi Public Health framework [42]. The authors suggest that identity triggers, or elements of the social environment that trigger awareness of one's social status, are one mechanism through which structured inequities act to differentially impact on health and lead to health disparities. The unequal social conditions in which black and white women and men are situated influence the type and saturation of identity triggers each of these groups will encounter, as well as available coping resources [39, 111], within and across various social settings [42]. According to this framework, experiences of discrimination pose a setting-specific disease risk for each gendered race group. We suggest further that perceived experiences of interpersonal discrimination can act as identity cues, even in the absence of actual occurrences of discrimination, which might partially explain the associations we found among white women and men. Identity triggers and the perceived coping resources [39] particular to black and white women and men may act to specify conditions under which experiences of interpersonal discrimination will have a measurable impact on CVH.

Hierarchical social conditions create power dynamics between marginalized and dominant status groups which influence how inequity will be experienced on a personal basis by members of both types of groups [7, 10, 97, 114]. Experiences of discrimination based on gendered race that occur in the context of medical care, education, or in interactions with law enforcement, for example, can bring to bear historically structured power imbalances through heightened awareness of one's stigmatized status in the form of race consciousness [99, 115]. Instances of interpersonal discrimination in these specific settings may be uniquely stressful for marginalized persons both because of the likelihood of recurrence and a perceived lack of opportunities for retribution [39, 42, 111].

On the other hand, the settings in which awareness of unequal social status might be triggered among dominant group members—whether or not a discriminatory interaction actually occurred—and the resources they believe are available for coping with the accompanying stress, likely differ. These perceptions of social status triggered by interpersonal discrimination lead to between-group differences in the types of social contexts in which experiencing discrimination will contribute to deteriorated CVH. This interplay is consistent with our findings that although a higher percentage of black men reported encountering discriminatory treatment in public or on the street than in any other setting, this setting was the only one in which exposure was not associated with poorer CVH within this group. In contrast,

"in public or on the street" was one of the two settings in which white men who reported experiencing racial and gender discrimination experienced declining CVH. Given the historical contexts in which white men's social status afforded a measure of public and occupational deference, for some white men instances when this deference is absent or challenged in settings such as on the street or at work may be more likely to be perceived as discriminatory and more stressful than encounters perceived as discriminatory in other settings, an explanation that is consistent with the findings of this study.

4. The role of valid theory in ethical and scientifically sound research

Despite the detailed theoretical focus of this chapter, a wholistic identity approach to epidemiologic research, of which the IP framework is one example, has practical application for clarifying gender-related differences in health. One of the most significant assertions of the IP framework is that epidemiologic research should embrace a more nuanced approach to social determinants of health and health disparities research, specifically as related to assumptions of homogeneity in social group differences in health. This paper has presented strong evidence that concepts of gender and race are conceptually far more complex than is often operationalized in many epidemiologic analyses. Furthermore, the health implications of adverse social experiences associated with gender and race are heavily dependent on psychosocial characteristics that are rarely measured in epidemiologic studies.

The inclusion of sound theoretical foundations is necessary to ethically and rigorously address these concerns. The IP framework calls for reconsidering some standard methodologies of epidemiologic research. Because the white male referent presents a number of conceptual problems, using stratified analyses can circumvent many of the biases to which research questions based on multi-gender, multi-racial, or multi-ethnic samples are vulnerable. Stratified analyses can also avoid the pitfall of including variables for complex social constructs such as gender or race in regression models, as recent literature has described notable limitations and conceptual inconsistencies in this approach [15, 16].

In addition to implications for improving the rigor of scientific research, the increased application of well-developed theory to research into the causes of social group differences in health has ethical implications as well. Outside of the academic settings in which health research frequently occurs, epidemiologic findings have significant impact of the health and lives of real people. What we discover about the causes of gender differences in health informs the policies and societal changes intended to alleviate unnecessary and unjust suffering. A failure to fully consider all available evidence is a failure to meet the lofty ideals of epidemiology as a discipline —to identity the causes of disease in order to eradicate.

5. Conclusions

In this chapter, I have addressed the necessity and challenge of incorporating sound theory into epidemiologic research on the causes of gender differences in health. Intersectionality Theory has in many ways served as a springboard for the growing collaboration of epidemiology with social science. Still, although the use of interdisciplinary theory in epidemiologic research has increased substantially within the last decade, there is much room for improving the application of theory to everything from developing research questions to the selection of confounders to the interpretation of results. Moreover, much of the research employing an

intersectional approach struggles to deeply engage the health implications of concepts like socially constructed gendered race. While there is acknowledgement of the external social processes which shape the health of groups in different socially defined categories, little attention is given to how internal identity processes also play a pivotal role in determining health. This oversight is largely due to a resistance within the field of epidemiology to grapple with complex social psychological phenomena such as the influence of social group identity on population-level differences in health.

For this reason, I have argued in this chapter for the necessity of moving beyond intersectional approaches to health disparities research. The use of a wholistic identity approach to understanding social group differences in health requires the engagement of wide array theories which each provide important but distinct elements of the larger mechanisms by which structural inequity produces social group differences in health. One wholistic identity theory, the Identity Pathology model, is built on such an interdisciplinary conceptualization of health disparities.

According to Identity Pathology theory, embedded in socially constructed identities are beliefs that moderate whether and how exposure to chronic adverse social conditions, for example experiences of interpersonal discrimination, will generate disease. When individuals are socialized with identities built on pathogenic identity beliefs, they are more susceptible to a number of physical and mental illnesses. Pathologized identities act to foster disease through dictating cognitive and behavioral practices—stressor appraisal, health behaviors, etc.—that yield distinct pathologies in the context of unequal social conditions. The IP framework argues that gendered racial identities constructed in the context of inequitable social conditions create unique manifestations of health and disease among black and white women and men, contributing to gender differences in health that will vary across race in a manner that may not be adequately captured in current interpretations of Intersectionality Theory.

Fundamentally, many theories on social determinants of health, including Intersectionality Theory, predicate the health of the socially marginalized—whether that be on the basis of gendered race, socioeconomic status, etc.—on a set of resources of which they are systematically deprived. In some ways, these theories carry undercurrents of an adversarial tone by situating the "disadvantaged" as those who have everything to gain from social change against the "privileged" who are at risk for a corresponding loss. As such, any improvements in the health of members of marginalized groups are necessarily dependent on the decisions of those who retain power over the distribution of these resources, individuals who have little incentive to relinquish their positions of authority (perceived and actual) or enact more inclusive policies [28].

In *The Health Gap*, Marmot observes: "Being at the wrong end of inequality is disempowering, it deprives people of control over their lives. Their health is damaged as a result. And the effect is graded–the greater the disadvantage the worse the health" [116]. This observation, while not incorrect, seems to suggest, almost tacitly, unintentionally perhaps, that at the "right" end of inequality, individuals are artificially empowered. I consider whether the focus on external resources to the exclusion of internal resources of health is another form of disempowering marginalized persons. To act on the idea that the marginalized many cannot even enjoy health without the permission of the advantaged few feels, at its core, like another practice of structural violence.

If structural inequities and the unequal health outcomes such conditions cause are to be truly deconstructed, intervention must entail more than efforts to change social and economic policies which were intentionally established to ensure that power and resources remain under the control of white men [10, 26, 28, 34].

The persistence of documented health disparities over the last century despite long-standing calls for social, economic, and political reform as well as substantial advances in our understanding of the role of social determinants in health indicates, as the IP framework theorizes, that these policies and the decision-makers behind them are resistant to change. Reservoirs of infection, source populations which stubbornly harbor pathogenic identity beliefs even as changing discourse variably decreases or increases the acceptability of social prejudice, ensure that interventions focusing only on shifting policy will do little to yield lasting social equity.

In light of these observations, the IP framework suggests that because pathogenic identity beliefs perpetuate the pathogenic social environments in which they flourish, interventions must target the environment, agent, and host simultaneously. Eradicating health disparities therefore requires an additional approach that acts in conjunction with efforts to deconstruct problematic institutions and policies, and efforts to create identity-safe cultures. In the case of identity pathology, environmental interventions, which have been well-described in extant theory [1, 37], involve abolishing the policies and practices which maintain and promote inequity within social institutions and the inequitable distribution of health-impacting resources. Agent interventions require shifting the cultural and social norms in which pathogenic beliefs flourish and are transmitted, as proposed by the Jedi Public Health Framework [42]. Host interventions, which the IP framework newly proposes, target the identity beliefs which make individuals particularly susceptible to the effects of inequitable social conditions on the cognitions and behaviors that directly and indirectly influence their own health as well as the health of others.

Although the IP framework uses the example of race and gender hierarchy among black and white women and men, application of the framework extends well beyond these particular groups and examples of structural inequity. As the objective of the framework is to highlight the substantial role of identity processes in health outcomes, the principles of the IP framework can be adapted to describe the effects of any inequitable social contexts on the physical and psychological well-being of any populations exposed to those contexts. The IP framework may be particularly useful for examining the understudied health impacts of structural inequity among groups such as those with varying physical abilities or native populations whose suffering has been systematically made invisible.

The framework is densely theoretical and draws from a number of disciplines in outlining complex mechanisms from structural inequities to health inequities. Despite its ambitious reach, the core concepts of the framework are readily applicable to health research. Through suggesting adjustments to analytic methods, outlining testable causal mechanisms, and proposing an evidence-based intervention, the IP model orients health researchers toward another channel for more ethical and rigorous investigation the causes of and solutions to unjust gender disparities in health.

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