MOLECULARIZATION AT THE INTERSECTIONS: TESTOSTERONE, PROSTATE CANCER AND THE CONSTRUCTION OF RACIAL DIFFERENCE

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ABSTRACT OF THE DISSERTATION

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Prostate Cancer and the Construction of Racial Difference

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In this dissertation, I examine how race has been molecularized through the hormone testosterone and the impact that this process has had on the construction of racial disparities in prostate cancer between black and white men. While testosterone is widely conceptualized as a molecular marker of masculinity by both scientific and popular accounts, feminist science studies scholars have documented how these claims are both misleading and dangerous. Building from this foundational work, this project explores how testosterone has been both gendered *and* racialized by scientists over the course of the 20th-century. More specifically, biomedical researchers claim that racial differences in testosterone help explain racial disparities in prostate cancer between black and white men. This discourse endorses the theory that higher levels of testosterone, which has historically been a marker used to designate prostate cancer risk, must differ between black and white men and thus explain the persistence of prostate cancer disparities between these two groups.

To examine the validity of these racialized claims, I use insights from science & technology studies, critical race theory, and social network analysis to critically evaluate the triangular linkages between testosterone, race, and prostate cancer in biomedical research. In my first analytic chapter, I examine the relationship between testosterone and race. To do this, I conduct a content analysis of 147 studies that evaluate population dif-

ferences in testosterone. I find that, despite widespread claims that testosterone varies between racial groups, the literature provides scant evidence to support these assertions. To demonstrate how population differences are enacted and reproduced, I use social network analysis to visualize a citation network of these data and trace how the racialization of testosterone circulates through scientific research. I identify three mechanisms – ambiguity, absence and data recycling – to help explain how racial difference testing has contributed to preserving the cultural myth of testosterone as a molecular marker of racialized masculinity.

Second, I evaluate the linkage between race and prostate cancer, looking to understand how racial disparities in this disease are constructed. Biomedical and epidemiological researchers widely claim that African American men suffer from prostate cancer at two to three times the rate of white Americans. However, a critical review of this literature finds that racial disparities in prostate cancer-specific mortality are largely explained by socioeconomic differences between the two groups. While scientists do discuss the effects of socioeconomic inequalities in their work, researchers also suggest that black men's more "aggressive biologies," propelled by testosterone and other biological differences, help explain why this group is more likely to experience earlier onset of disease, faster growing tumors and worse overall survival. These racialized claims shape group-differentiated patterns in the use of prostate-specific antigen screening, racially-specific treatment guidelines, and variability in the distribution of hormone-based pharmaceuticals and surgical interventions. Ultimately, I argue that biomedical researchers' focus on explaining prostate cancer through racial differences in biology (i.e. testosterone) ulti-

mately leads to the misallocation of funding away from addressing the structural causes that drive these disparities in the first place.

In the final analytic chapter, I investigate the linkage between testosterone and prostate cancer to see how this association has changed over time. By examining changes in scientific consensus over time, I demonstrate that the association between testosterone and prostate cancer has undergone a radical paradigm shift over the past 25 years. While *high* levels of testosterone were considered a robust indicator of prostate cancer risk for more than seven decades, most biomedical experts now argue that clinically *low* levels of testosterone may, in fact, be a more accurate risk factor for diagnosing this disease. Although prostate cancer researchers have moved towards consensus, this paradigm shift has not carried over to impact all domains of research in the same way. For example, today's most widely used clinical guidelines on testosterone replacement therapies explicitly advise clinicians *not* to prescribe testosterone to African American men because of their increased risk of prostate cancer. These guidelines are not only predicated on the assumption that black and white men have different testosterone levels, but also on the antiquated theory that higher levels of testosterone contribute to prostate carcinogenesis, which unjustly withholds testosterone therapies from black men. In response to these findings, I call for the Endocrine Society to reassess their guidelines to reflect a more equitable policy on testosterone replacement therapies, which relies on the best available evidence on the topic.

Together, these chapters demonstrate that molecularization and racialization are co-constitutive processes that shape the contours of today's biomedical markets, including the "gold-standards" of evidence-based medicine and access to hormone-based phar-

maceuticals for black and white men across the United States. In the conclusion, I discuss how my work speaks to scholars working in science & technology studies as well as the literatures on the medicalization and pharmaceuticalization of race. Furthermore, I outline how my work affects biomedical research on testosterone. Most notably, I argue that those responsible for constructing the Endocrine Society's clinical guidelines on testosterone replacement therapies need to revise their recommendations to remove race as a basis for withholding pharmaceuticals from black consumers. While it is crucial to recognize and assess the potential risks of hormone-based pharmaceuticals, using race as a basis for (not) disseminating medical treatment perpetuates the legacy of scientific racism and unjustly bars important resources from patients who may benefit from the use of testosterone replacement therapies.

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Chapter 1: Molecularizing Race through Testosterone

After the completion of the Human Genome Project in the early-2000s, many social scientists thought that the search for a molecular basis to race had finally met its end. Although critical race scholars, social scientists and historians had already argued for decades that race is more appropriately conceptualized as a social and political classification rather than a biological essence, the Human Genome Project corroborated these claims by showing that no biological markers could reliably distinguish between racial groups (see Bliss 2012; D. Roberts 2011). And yet, over the past 20 years, the use of race and other population classifications have only proliferated in genetics and genomics research (Panofsky and Bliss 2017), as scientists continue to use these terms to help explain the molecular underpinnings of health disparities as well as a myriad of other social and behavioral outcomes (Bliss 2012, 2018). Though social scientists have done well to document how race is used in the context of genetics and genomics, few have focused on how specific biomarkers have become racialized in biomedical research.

In this dissertation, I examine the ways that the hormone testosterone has become simultaneously gendered *and* racialized in scientific studies. By most popular accounts, testosterone is conceptualized as a molecular marker of masculinity. These proclamations may make intuitive sense to some, especially given that testosterone has the capacity to produce hair and muscle on the body - characteristics that are commonly encoded as "masculine" traits (Giltay and Gooren 2000; Hembree et al. 2017; Huo et al. 2016). However, as feminist science studies scholars have argued for more than three decades, and as I detail more below, reducing testosterone to a "sex hormone" can be both misleading and dangerous (Fausto-Sterling 1985, 2000; Fine 2017; R. Jordan-Young and

Karkazis forthcoming; Oudshoorn 1994; C. Roberts 2007). Despite these critical efforts, testosterone is still widely essentialized in and outside of scientific literature today (R. Jordan-Young and Karkazis forthcoming).

While past scholarship has done well documenting how the hormone is sexed/gendered (Fausto-Sterling 1985, 2000; Fine 2017; R. Jordan-Young and Karkazis forthcoming; Oudshoorn 1994; C. Roberts 2007), researchers have only recently directed their attention to the ways that cultural assumptions about race shape how testosterone is conceptualized in scientific studies (Carlin and Kramer 2018; Gill-Peterson 2014; R. Jordan-Young and Karkazis forthcoming; Karkazis and Jordan-Young 2018). This dissertation adds to this burgeoning literature by documenting how testosterone has been racialized and the impact that this has had on the scientific construction of racial disparities in prostate cancer. Today, biomedical experts advance claims that black men suffer from prostate cancer at two to three times the rates that white men do in the United States (Siegel, Miller, and Jemal 2017). To help explain this disparity, researchers advance what I call the "racial testosterone theory" - a discourse that suggests racial differences in testosterone help to explain, at least in part, why black men (supposedly) develop and die from prostate cancer more often than their white counterparts (Richard et al. 2014; R. Ross et al. 1986). But do these claims hold up to scientific scrutiny and, if not, what are the implications for the production and dissemination of biomedical interventions for black and white men being treated for prostate cancer?

In this dissertation, I examine how race is molecularized *through* testosterone and the impact this has on health disparities research by focusing on the linkages between three entities: race, testosterone and prostate cancer. As I explain more in the forthcoming

pages, I conceptualize these three entities as a set of triangular relations that are connected by both scientific evidence as well as the claims that researchers deploy to characterize their findings. Because what scientists claim and what scientific evidence actually demonstrates do not always align, one core motive of this project is to reveal the discordances between evidence and claims in this context by focusing, more specifically, on how cultural assumptions about race and gender seep into testosterone research.

To do this, I employ insights of feminist science & technology studies, critical race theory, and intersectional theory to detail how testosterone is not *just* imbued with cultural stereotypes that perpetuate its mischaracterization as "male sex hormone," but also to show that testosterone is racialized in a way that it alters how scientists conceptualize the biological risk profiles of black and white men diagnosed with prostate cancer. The lens of intersectionality helps to reveal the ways that testosterone is enacted in distinct and even contradictory ways depending on the context that it circulates through (R. Jordan-Young and Karkazis forthcoming). Most importantly, this may also help to illuminate how preconceptions about specific racialized and gendered groups lead to outdated or discriminatory biomedical practices.

Leaning on the triangle to link race, testosterone and prostate cancer together as a conceptual framework, each analytical chapter interrogates the linkages between two of these entities. In my first analytic chapter, I start by examining the connection between race and testosterone, showing that the racial testosterone theory (i.e. the discourse that there are racial differences in testosterone) is unsupported by extant scientific literature. Still, researchers broadly claim that racial differences in testosterone help to explain why

black and white men tend to exhibit different tendencies in both biosocial (e.g. patterns of crime and aggression) and biomedical outcomes (e.g. disparities in prostate cancer).

In the second analytic chapter, I conduct a critical content analysis of epidemiological research in order to explore the widespread claims researchers make about black men having a higher incidence *and* mortality of prostate cancer. My overview of this literature shows, however, that racial gaps in mortality are robustly explained by group differences in socioeconomic status. While scientists do discuss the potential effects of socioeconomic status in their work, I find that researchers still tend to deploy and sometimes even prioritize molecular discourses when explaining racial disparities in prostate cancer. Most notably, prostate cancer researchers argue that black men have "more aggressive" biologies than their white counterparts. More specifically, black men are thought to have higher levels of testosterone, prostate-specific antigen and shorter androgen receptors, which are thought to lead to earlier onset of disease, more aggressive tumors and worse overall survival. These discourses not only reflect how cultural stereotypes about black bodies (and testosterone) are imbued into biomedical knowledge, but also lay a false foundation for scientists to deem black men (in)eligible for receiving various biomedical interventions to treat prostate cancer.

Building on the findings from these first two chapters, I analyze how scientists conceptualize the relationship between testosterone and prostate cancer and how it has changed over time. In the world's leading clinical guidelines on testosterone replacement therapies, the Endocrine Society explicitly advises clinicians *not* to prescribe African American men testosterone-based pharmaceuticals because of their higher risk of prostate cancer (Bhasin et al. 2010, 2018). This advice is predicated on the assumptions (1) that

black men have higher levels of testosterone and (2) that testosterone contributes directly to a higher risk of developing prostate cancer. Thus, by tracing historical patterns of scientific consensus in prostate cancer research, I outline a radical paradigm shift in how experts conceptualize testosterone as a biomarker underpinning this disease. While *high* levels of testosterone were thought to be a marker of prostate cancer risk for more than seven decades, researchers now suggest that clinically *low* levels of testosterone may, in fact, be a better risk factor for diagnosing that disease. The disjuncture between my structural analysis of scientific consensus and the recommendations advanced in the clinical guidelines suggests that the decision to withhold testosterone therapies from black men is predicated on an historical myth, which carries over to impact their access to these drugs throughout the United States (Jasuja et al. 2017).

Before moving into these analyses, this introductory chapter will give a brief overview of two literatures that I draw from extensively in this dissertation. In the first section, I outline how feminist science studies scholars have examined testosterone in past work. I begin by emphasizing that testosterone has long been mischaracterized as a so-called sex hormone, which leads to misleading claims about behavioral and structural differences between men and women. I then outline the histories of hormone-based pharmaceuticals, touching on how they have contributed to various biomedical controversies for almost a century. In the second section, I summarize literature on the molecularization of race in biomedical and pharmaceutical research. In doing so, I hope to contextualize how this project complements other work focusing on racialized biomedical contexts, including the infamous case of BiDil.

A Brief History of Testosterone in Scientific Research

Since their synthesis in the 1920's, "sex hormones" have played an integral role in shaping medical knowledge. By tracing the social networks of bench scientists, clinicians, and pharmaceutical companies, Oudshoorn (1994) has shown that what scientists claim about these hormones do not reflect "natural facts," but instead demonstrates that these chemicals *became* masculine or feminine when pre-scientific ideas about male/female differences were imbued into them over time (see also Gaudillière 2003, 2004, 2005). Perhaps, most famously, Brown-Séquard (1889) touted that chemicals extracted from animal testicles could be injected into the body to rejuvenate lost physical stamina and intellectual capacities that men lose as they grow older. While Brown-Séquard was later discredited by the scientific community, the work of other researchers, especially Viennese endocrinologist Eugen Steinach's, would eventually bolster testosterone's reputation for having the potential to rejuvenate lost masculine qualities through much of the early 20th century (Oudshoorn 1994). Since this time, testosterone has been widely conceptualized as *the* male hormone by the media, public, and even by some scientific experts (Baron-Cohen 2003; Dabbs and Dabbs 2000; Hoberman 2005). Popular discourses often proclaim that men's higher levels of testosterone help to explain various differences between men and women - from sex differences in spatial and mathematical reasoning to men's propensity to engage in aggression, crime and other forms of deviance at higher rates than women (Baron-Cohen, Knickmeyer, and Belmonte 2005; Booth et al. 2006; Hines 2006; Mazur and Booth 1998).

These discourses persist despite a number of compelling arguments that suggest gendering hormones is both misleading and dangerous (Fausto-Sterling 1985, 2000; Fine

2017; R. M. Jordan-Young 2010; R. Jordan-Young and Karkazis forthcoming; Oudshoorn 1994; C. Roberts 2007). For one, testosterone circulates through *all* human bodies, regardless of sex/gender, and is essential to many core physiological functions, including liver metabolism. While exogenous testosterone does have the capacity to produce hair and muscle when administered to the body (Giltay and Gooren 2000; Hembree et al. 2017; Huo et al. 2016), these are not physical traits that occur exclusively in men. In fact, recent meta-analysis has even found that more testosterone in the body does not necessarily correspond to the amount of hair on the body (Amiri et al. 2017). While men do tend to have higher levels of testosterone than women, on average, some women do have higher testosterone levels than some men (Stanton 2011; Handelsman et al. 2015; Healy et al. 2014; Karkazis and Jordan-Young 2015), which further undermines the notion that testosterone is essentially masculine.

Yet, the ideology that testosterone can explain gender differences in behavior or gendered disparities in income, wealth or occupational success has been exploited by scientific researchers and the popular media for some time. For example, at the height of the financial crisis, a slew of researchers argued that men's higher levels of testosterone help to explain the rampant plunge in the stock market in the mid-2000s (Coates and Herbert 2008; Apicella et al. 2008; Cueva et al. 2015). These authors suggest that men's higher levels of testosterone, which supposedly drives them to be "more successful" than women in occupational and evolutionary terms, also propels them to engage in the same risk-taking behaviors that ultimately contributed to the stock market crashing.

Beyond the biological essentialism that this view endorses, feminist scholars have also pointed out that the concepts used in this work are inherently biased. Fine (2017) cri-

tiques these researchers for conceptualizing measures like "risk-taking" in gendered terms, focusing, for example, on the risks of stock-trading over the inherent dangers of pregnancy. When researchers suggest that testosterone differences between men and women help to explain why men have been more successful in the financial industry, they also ignore the larger cultural and historical systems that prevent women from entering into this arena. Despite these counterpoints, testosterone is still imbued with cultural meanings of masculinity and, in turn, our knowledge about this hormone becomes distorted by the assumptions that follow it around in and outside of the scientific literature (R. Jordan-Young and Karkazis forthcoming).

To be fair, hormones are not a simple subject to study. As Jordan-Young and Karkazis (forthcoming) note, testosterone is a multiplicity that is enacted in different ways across scientific and cultural settings, which means the hormone has a variety of distinct ontologies. For example, testosterone can be both an *endogenous* hormone, found circulating in the body at birth, as well as an *exogenous* hormone that enters into the body via various methods of administration. In this dissertation, I mostly focus on how testosterone is conceptualized as an endogenous hormone, which I argue *becomes* racialized through scientist's assumption that hormonal differences arise "naturally" between racial groups. While each chapter examines how endogenous testosterone is conceptualized as a risk factor in biomedical research, the broader implications of my work speak more to issues surrounding access to testosterone as an exogenous hormone administered to the body to treat various medical conditions. To better understand the implications of those findings, I now turn to a brief history of hormone-based pharmaceuticals.

Over the course of the 20th-century, the distribution of so-called sex hormones has impacted men and women in dramatically different ways. Hormone replacement therapies (HRTs) and contraceptives have been widely used by women since the 1930's with HRT consumption only declining over the past 20 years (Krieger et al. 2005; Krieger, Chen, and Waterman 2010; Verkooijen et al. 2009). On the other hand, testosterone-based pharmaceuticals had only a brief period of success during the 1930's before sales fizzled out when a number of technical, industrial, and political factors led to this market collapsing during the interwar period (Gaudillière 2003, 2004, 2005; Laveaga 2005; Oudshoorn 1994). As a result, clinical research on testosterone fell out of favor after the mid-1950's until a resurgence in the 1990's made TRTs relevant once again (Watkins 2007, 2008, 2012). Most argue that the pronounced difference in the distribution of hormone-based pharmaceuticals is due to the political fixation with controlling women's bodies, particularly in the domain of sexual reproduction (Oudshoorn 1994; Preciado 2013; C. Roberts 2007). As this work suggests, cultural preoccupations about gender not only muddle biomedical knowledge, but also affect the economic landscape of the global pharmaceutical industry.

As feminist science studies scholars argue, conceptualizing so-called sex hormones as "natural" remedies can negatively shape health outcomes. While HRTs were widely distributed to women during the 20th-century, a major controversy arose in the 1990's when the Women's Health Initiative found that these drugs put women at a dramatically higher risk of developing breast cancer and heart disease (Rossouw 2002; Writing Group for the Women's Health Initiative Investigators 2002). Krieger and colleagues (2005) argue that HRT distribution was driven by a negligence to consider how history

and socio-cultural factors, like gendered assumptions about "sex hormones," misled bio-medical scholars about the capacities of these drugs. More specifically, scientists assumed that estrogen and progesterone were "feminine" substances that would promote better health in women after the hormonal changes that occur during menopause. By ignoring that hormones do more than just produce sex-linked characteristics, scientists and clinicians dismissed the fact that these drugs could also proliferates cell growth and carcinogenesis, which harmed millions of women over the course of four decades. The point here is that reducing hormones to being essentially sexed/gendered misled researchers, obscuring them from seeing the more far-reaching dangers of what these drugs had the capacity to do.

While HRT use has dwindled since the early-2000s, the testosterone replacement therapies (TRT) industry has grown exponentially during that time. Since 2000, the TRT industry has grown from $300 million to a $2 billion global market in 2011 (Handelsman 2013). TRTs provide a robust example of what sociologists call *pharmaceuticalization* or the process by which social, behavioral, or bodily conditions are deemed to be treatable through drugs (Abraham 2009). To date, most social scientists argue that the expansion of the TRT market resulted from the sexual medicine industry reworking the diagnostic criteria of men's health conditions like erectile dysfunction and andropause in order to promote testosterone as a "lifestyle drug" (Lexchin 2001; Marshall 2009a; Watkins 2007, 2008, 2012). In recent years, however, TRTs have also been prescribed to treat health conditions as diverse as depression, metabolic syndrome, diabetes, insulin resistance, and/or cardiovascular disease (Abdulmaged M Traish, Guay, et al. 2009; Abdulmaged M Traish, Saad, et al. 2009; Abdulamaged M Traish, Saad, and Guay 2009), which is a

point that sociologists have largely failed to recognize in their attempts to explain the growth of the TRT industry. By focusing on how TRTs are talked about in prostate cancer research, I hope to add to the sociological literature by showing that testosterone is not just a drug talked about in the sexual medicine literature. Instead, debates about TRTs and their potential risks require researchers to expand into other domains of knowledge like oncology and cardiology.

Few sociologists have focused on the parallels between HRTs and TRTs. While HRTs have largely fallen out of favor for female consumers around the world, emerging evidence suggests that TRTs may increase the risks of cardiovascular disease (Huo et al. 2016; Lin Xu et al. 2013), leading the Food and Drug Administration to mandate warning labels on these products (FDA 2015). In a sense, it seems that history is repeating itself – when HRTs began to decline because of the health risks they posed for women, TRT use began to increase with the same deleterious health risks now arising in studies conducted on men (Huo et al. 2016; Lin Xu et al. 2013; but see Morgentaler et al. 2016; Morgentaler 2016). On the other hand, the potential benefits of using TRTs in men who have clinically low levels of testosterone (i.e. "low T") in addition to a number of well-documented symptoms have been widely documented by emerging gold-standard studies (Bhasin et al. 2010, 2018; Corona et al. 2014, 2016; Ponce et al. 2018). In this dissertation, my work grapples with both the potential benefits and risks of testosterone therapies while also showing the distinct ways that they play out to impact different racial groups.

Molecularizing Race in the Sociogenomic Era

The *molecularization* of masculinity is part of a broader historical shift in the 'ethopolitics' of scientific research (Rose 2007). Today, biomedical and epidemiological scientists

employ frameworks that conceptualize social processes, like health disparities, in biological and genetic terms. As a result of this shift, the management of "risk" at the population level, which was previously a concern of public health initiatives like nationalized health care, has been displaced back onto the individual and their genetic inheritance. In turn, individuals are forced to take responsibility for managing their own health by eating well, exercising, engaging in the proper regimens of self-care, and, more pertinent to my interests, by attending to their biological risk profiles. Testosterone is incorporated into this ethopolitical shift in that men are now encouraged to consider whether their "low T" might contribute to their underwhelming sex lives, reduced energy, or recent weight gain (Watkins 2012). By taking up TRTs, many men believe they can take control over their lives by using pharmaceuticals to manipulate their molecularized selves.

In the same sense, race too has become increasingly molecularized over the past two decades. While many believed that racially motivated biological analysis would come to an end after the Human Genome Project found that no biological markers could reliably distinguish between racial groups, the postgenomic era has actually led to proliferation in research of this kind (Bliss 2012, 2018; Bolnick et al. 2007; Duster 2004; D. Roberts 2011; TallBear 2013; Wailoo, Nelson, and Lee 2012). Social scientists note that "race" can come to signify many things in genomics and biomedical research. As Shim (2002) suggests, race can be included in routinized ways that lack any theoretical foundation whatsoever. In turn, social scientists tend to focus on the ways that race is used to reinscribe group differences largely or entirely in genetic terms (Fujimura and Rajagopalan 2011; Fullwiley 2007; Montoya 2011).

Alternatively, Bliss (2018) has outlined the recent growth of the sociogenomic paradigm. In this view, many aspects of the world, like race and health disparities, are seen as the result of an interdependent mix of social *and* biological factors. While race is often molecularized within this framework, researchers do not necessarily make strong claims about what race "is," but instead focus more on using racial differences to fulfill various heterogeneous objectives from mobilizing funding for future research to justifying racially-specific biomedical interventions (Chun 2013; Shim et al. 2014). In this sense, race is both a *multiplicity*, in that it can have multiple meanings depending on the context it is mobilized within (M'charek 2013), and a *technology* that can be used to 'do things' (Chun 2013) like creating racialized pharmaceutical products.

Undoubtedly, the most infamous example of these interventions is BiDil - a drug that is marketed by NitroMed to treat heart failure in and only in African American patients (Kahn 2013; Pollock 2012). BiDil was originally tested in a large, multi-racial cohort, but failed to garner regulatory support because the original data were not designed to present to the Federal Drug Administration (FDA). After the FDA denied the original application, NitroMed ran sub-group analyses on the clinical trial data and later published a paper arguing that the drug was efficacious for treating heart disease in African American patients (Carson et al. 1999). In turn, the company ran trials with only African American patients, finding that the drug reduced mortality rates by 43% (A. L. Taylor et al. 2004). While NitroMed never tested whether BiDil worked better in black consumers compared to patients in other racial groups. the company did eventually garner a patent for BiDil as a racially-specific drug that allowed them to obtain an additional 13 years of market monopoly (Kahn 2008). As Kahn (2003) documents, one of the arguments for re-

branding this drug for a racially-specific market was predicated on the notion that black people suffer from heart disease at higher rates than their white counterparts, despite the statistics underlying this claim being unsubstantiated by epidemiological research. What Kahn shows in his work is that the discourse of health disparities, regardless if they are grounded in strong evidence, can be exploited by researchers in order to motivate research activities, including the development of racialized pharmaceuticals.

In this dissertation, I work from the basis that race is not "biological" in the sense that some genetic marker can accurately distinguish the "race" of an individual or group, but instead that biomarkers become racialized over the course of time. First, the Human Genome project has shown clearly that biological markers cannot reliably distinguish between racial groups (see Bliss 2012; D. Roberts 2011). Yet, researchers still uncover racial differences in biological outcomes when conducting their research. Thus, I believe that social scientists must develop theories that explain why racial differences can and do arise in scientific research, which requires re-conceptualizing how biological differences that emerge between groups are shaped by either research practices or dynamic structural processes. Following this logic, biological differences could be "real" in the sense that they are produced by inequalities in one's social environment. For example, group differences in testosterone may arise as the result of disparities in socioeconomic status, environmental exposure to endocrine disrupting chemicals, or diet and other lifestyle factors that alter the hormone's production over the life course (Dabbs and Morris 1990; Gore et al. 2015; Hall et al. 2008).

The reality is that many testosterone researchers still believe that racial differences are static traits that contribute to stable group-differentiated tendencies in hormonal

production and/or polymorphic variation in genes (R. K. Ross et al. 1998; R. Ross et al. 1986). I stand opposed to this perspective, not only because extant evidence demonstrates that biomarkers do not distinguish racial groups, but also because the very notion of "race" is culturally, socially, and historically contingent (Morning 2011; Omi and Winant 2014; D. Roberts 2011). Because race and other population distinctions continue to change based on the social and political systems that enact them over time, it would be impossible to empirically distinguish genetic differences between racial groups because the basis of those distinctions are not static in the first place.

Working from this premise, I focus instead on the *racialization* of testosterone as a scientific object. Racialization has many meanings, which race and ethnicity scholars have detailed in their historical accounts of this concept (Barot and Bird 2001; Hochman 2018; Murji and Solomos 2005; Omi and Winant 2014). My use of the term aligns most closely with that of Omi and Winant (2014) who argue that racialization can be understood as the "the extension of racial meaning to a previously racially unclassified relationship, social practice, or group" (13). By focusing on racialization as a *process*, rather than reifying "race" as foundational concept to predicate difference-making, we also open up the possibility of de-racializing scientific classification systems (Hochman 2018).

Importantly, the molecularization of race and the racialization of testosterone are different, but overlapping, processes. The molecularization of race is a more comprehensive framework focused not only on how "race" is constructed in biological terms, but also interested in how institutions, procedures, instruments, and forms of capitalization are organized around the efforts to enact race as a biological entity (Rose 2007). This framework does not depend on one specific biomarker, but extends more broadly to a constel-

lation of practices, labs and other processes – typically in the field of genomics (Bliss 2012; Fullwiley 2007). My focus on the racialization of testosterone is different in that I examine how researchers work to establish group differences in testosterone over time though the use of scientific evidence and/or theoretical frameworks (see Chapter 2). It is only once these differences are demonstrated (in at least a subset of studies) and have gained some level of institutional backing that testosterone *becomes* racialized (or is imbued with meaning that varies between racial groups). Once testosterone is racialized, the discourse becomes a mechanism through which race can become molecularized, meaning that a broader set of behaviors and/or risks can be explained through the notion that testosterone is a biological vector that animates perceived racial differences. While race can still be molecularized without testosterone, the racialization of biomarkers, more generally, is necessary for race to become molecularized. Thus, I hope to focus on how testosterone was originally racialized with the hope that it can also be de-racialized moving into the future, especially since existing evidence offers no support that the hormone varies between racial groups (see Chapter 2).

Focusing on the racialization of testosterone is important because of the way that this hormone is gendered. Throughout this dissertation, I lean on the orienting framework of intersectionality to guide my analyses (Crenshaw 1989; McCall 2005). The insights offered by intersectional theory are imperative to understand testosterone as a multiplicity (R. Jordan-Young and Karkazis forthcoming), as its plenitude in the body often depends on sex, age, social class and body composition, among a myriad of other factors (see Chapter 1). Intersectionality is also important for interpreting extant scientific findings, especially since gender and racial essentialism often bias the interpretation of empirical

results in important ways. In this project, I focus primarily on the ways that researchers conceptualize testosterone as a biomarker that poses differentiating risks to black and white men. While I hope my future work will proffer insights that span beyond this corner of the intersectional spectrum, the implications of my findings on these two groups ultimately became so important that it I focused my efforts here for now. As I demonstrate in the second and third chapters, the way that testosterone is both gendered *and* racialized in scientific research affects the distribution of various biomedical interventions in ways that harm black men's health.

When looking at nexus of epidemiological and biomedical research, I found that racialization operates through the exploitation of "gaps" in biomedical research. Here, racial differences in health disparities, hormonal outcomes, or genetic markers are all "gaps" that researchers can act upon to mobilize research activities. For example, the disparity between black and white men's risk of developing heart disease was used as one of the justifications for rebranding and marketing BiDil as a racially-specific pharmaceutical (Kahn 2003). Without the motive to address this gap in mortality, there is no clear need to develop a racialized pharmaceutical for treating that disease. These gaps are not exploited solely to carry out racial projects, through this undoubtedly does happen, but also to advance the broader political and financial goals that a researcher may be invested in. Finding molecular differences between racialized groups provides a rationale to argue for more research funding or, in some cases, the distribution of interventions in racially-specific ways. Addressing disparities is not simply about finding out what causes them to persist, but also finding a solution - preferably one that a group of researchers can benefit from and profit on at a later date.

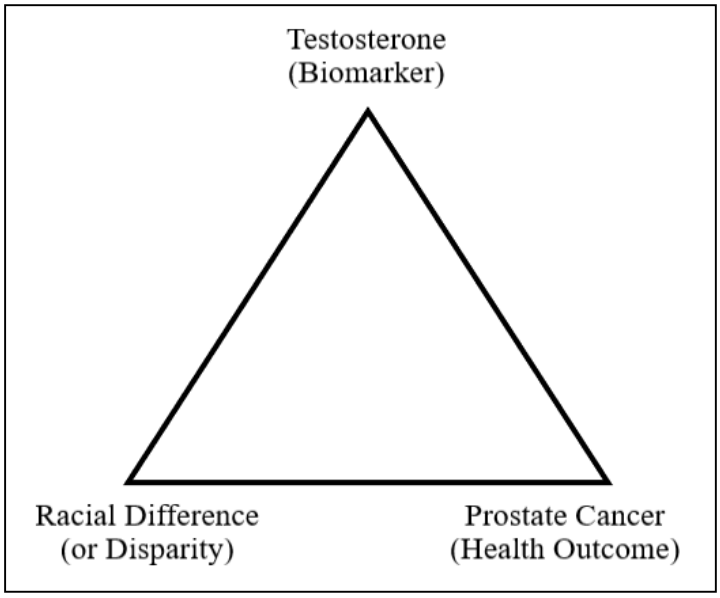

Figure 1. Triangle of Relations between Testosterone, Racial Difference and Prostate Cancer

In this framework, molecularization and racialization are co-constitutive processes, which, in turn, affect the contours of pharmaceuticalization in that specific drugs are directed towards consumers on the basis of their racial background. As I show in this dissertation, the molecularization of race *through* testosterone shapes how black men are deemed ineligible pharmaceutical consumers. More specifically, the Endocrine Society's clinical guidelines on TRTs explicitly "recommend against starting testosterone therapy in patients… at a high risk for prostate cancer such as African Americans" (Bhasin et al. 2010, 2536, see also Bhasin et al. 2018). While it is difficult to show that these claims causally influence prescription practices, it is the case that black men do receive TRTs at significantly lower rates than white men in equal access healthcare settings (Jasuja et al. 2017). While African American men's access to testosterone is undercut by these policies, it is important to note that these drugs are linked to an increased risk of cardiovascular disease in some studies (Huo et al. 2016; Lin Xu et al. 2013), suggesting that more access does not necessarily mean better care or long-term outcomes.

In a sense, this dissertation is a challenge to those responsible for constructing these guidelines and disseminating testosterone-based pharmaceuticals. After realizing the role that the Endocrine Society's guidelines play in shaping of the testosterone market, I began to examine the evidence underlying these claims. The recommendation to withhold TRTs from black men is predicated on three assumptions: (1) that black and white men have differing levels of testosterone, (2) that testosterone contributes to prostate carcinogenesis, and (3) that racial disparities in prostate cancer not only exist, but exposure to testosterone plays some role in exacerbating that disparity. The three analytical chapters in this dissertation follow directly from these claims.[1]

A Short Note on Data and Methodology

Throughout this manuscript, I lean on two primary methods of inquiry – content analysis and network analysis. Content analysis is a strategy widely used throughout science & technology studies, including in work that examines the use of race and ethnicity in biomedical and genomics literature (Bliss 2013; Lee 2009; Shim and Thomson 2010). Following similar procedures to these exemplar studies, I generated datasets of existing scientific publications at the intersection of testosterone and prostate cancer using the ISI Web of Science and Google Scholar databases. While Web of Science was used for all my original queries (search terms listed in the Data and Methodology sections for each chapter), I used Google Scholar to snowball larger, more comprehensive samples in Chapter 2 (on the racialization of testosterone) and Chapter 3 (on the construction of ra-

[1] The original use of this triangulation methodology was developed while working as a research assistant for Rebecca Jordan-Young and Katina Karkazis (Jordan-Young and Karkazis forthcoming). I have also taken a similar methodological approach in a co-authored article with Liz Carlin (Carlin and Kramer under review).

cial disparities in prostate cancer) in order to strengthen the quality of my analysis. My goal in both cases was to aggregate the largest sample I could in order to minimize the effects of sampling bias on claims related to population differences and disparities.

My approach to content analysis was based on grounded theory and abductive principles (Charmaz 2006; Timmermans and Tavory 2012). This means that in Chapter 2, for example, I sought to first examine how testosterone was racialized by looking at the most common comparisons conducted in the literature and evaluating whether evidence existed to support those claims. As an iterative response to these initial findings, I developed theoretical mechanisms that simultaneously explain inconsistencies in the literature while also showing how these mechanisms contribute to the enactment of racial differences in scientific studies. In Chapter 3, my approach was similar in that I was initially focused on the core claims about racial disparities in prostate cancer before I focused more specifically on how those disparities were simultaneously molecularized and racialized in the prostate cancer literature. In both cases, I worked to validate my theoretical framework while also using abductive principles to refine my theoretical contributions based on surprising and/or counterintuitive findings.

In Chapter 2 and Chapter 4, I also employed aspects of network analysis to examine scientific publications acquired from Web of Science and Google Scholar. Network analysis does have a long history in science studies (Garfield 1972; Price 1965), but this method, to my knowledge, has not been used to examine racialization in scientific studies. My implementation of network analysis in Chapter 2 contributes to the molecularization of race literature by offering a way to conceptualize racialization as a discursive process that unfolds over time while simultaneously demonstrating that most studies do not

support theories about racial differences in testosterone. While I opted to focus on the visual and descriptive aspects of these data for this dissertation, my future work will likely employ inferential network techniques to make claims about what propels racialization in scientific studies. On the other hand, Chapter 4 employs a methodology developed by Shwed and Bearman (2010) to examine patterns of scientific consensus over time. This procedure helps to establish a quantitative metric for establishing when consensus forms, which I used to inform my qualitative analyses that informed how these patterns intersected with racialization and molecularization more specifically.

Outline

In this dissertation, I examine how race is molecularized through testosterone and the impact this has on prostate cancer research. To do this, I interrogate the linkages between: (1) testosterone and race, (2) testosterone and prostate cancer, and (3) race and prostate cancer. As Figure 1 demonstrates, these relationships can be represented as a triangle where each vertex corresponds to (1) the hormone testosterone, (2) racial difference, or (3) prostate cancer. On the other hand, the three edges represent the discursive and evidential relationships that link these three entities together. My goal throughout this manuscript is to treat these associations as tenuous - relationships that are contingent on a diverse body of scientific practices and publications that do not always align. By critically examining both the discourses and evidence that circulate through scientific publications, I show that this triangle, in contrast with what biomedical researchers tend to claim, is actually quite fragile; the links of this triangle unravel when aggregating the evidence together as a whole. As I detail below, this unraveling has the potential to influence a wide

range of biomedical best practices, including those that follow from the Endocrine Society's clinical guidelines on testosterone replacement therapies.

To start, I take on the first link in Chapter 2, examining how researchers construct racial differences in testosterone. By conducting a content analysis of 147 studies that evaluate population differences in testosterone, I find that, despite widespread claims that testosterone varies between racial groups, the literature provides scant evidence to support these assertions. To demonstrate how population differences are enacted and reproduced, I use social network analysis to visualize a citation network of this data and trace how the racialization of testosterone proliferated from early 20th-century eugenics research to the contemporary "gold-standard" papers that biomedical experts use today. In turn, I propose three mechanisms to help explain how this myth has been perpetuated. First, I outline how the ambiguity of population classifications function across the literature, within individual papers, and through citation practices. Second, I show how the practice of data recycling allows researchers to enact testosterone as a racial construct and proliferate racial difference testing. Third, I trace how the absence of covariates, widely used in the broader testosterone literature, but omitted in large subsets of population comparisons, contribute to misleading claims about racial differences in testosterone. Together, these mechanisms provide a basis for biomedical researchers construct population differences, preserving the cultural myth of testosterone as a molecular marker of racialized masculinity.

In Chapter 3, I evaluate the linkage between race and prostate cancer, seeking to understand how racial disparities in this disease are constructed and how testosterone becomes incorporated within this broader project. Biomedical and epidemiological re-

searchers widely claim that African American men suffer from prostate cancer at two to three times the rate as white American men (Siegel, Miller, and Jemal 2017). These claims, however, ignore the fact that racial disparities in prostate cancer mortality are consistently explained by group differences in socioeconomic status. While scientists do discuss socioeconomic inequalities, they rarely advance recommendations to promote policy interventions to address these issues directly. Instead, researchers often argue that black men's "aggressive biologies" explain disparities in incidence and mortality between the two groups. More specifically, black men are thought to have higher levels of testosterone, prostate-specific antigen and shorter androgen receptors, which are thought to lead to earlier onset of disease, more aggressive tumors and worse overall survival. Following from these theories, prostate cancer researchers propose various racialized biomedical interventions to treat black white men in different ways in the clinic, including through the use of group-differentiated prostate-specific antigen testing, racially-specific treatment guidelines, and variability in the distribution of pharmaceutical and surgical interventions. Drawing from critical race theory and science & technology studies, I suggest this racialization of prostate cancer treatments is propelled by a broader structure of (white) ignorance that fails to address the structural inequalities that (poor) black men tend to face. As a result, the policy interventions that are advanced in epidemiological research do little to address the fundamental causes that drive these disparities in the first place.

In Chapter 4, I investigate the linkage between testosterone and prostate cancer. Using Shwed and Bearman's (2010) quantitative method to examine patterns of scientific consensus in citation networks from 1980-2017, I demonstrate that the association be-

tween testosterone and prostate cancer has undergone a radical paradigm shift over the past 25 years. While *high* levels of testosterone were once considered a robust indicator of prostate cancer risk for more than seven decades, a group of biomedical experts now argue that clinically *low* levels of testosterone may, in fact, be a more accurate risk factor for diagnosing this disease. While the Endocrine Society's clinical guidelines advise clinicians *against* prescribing testosterone to men with prostate cancer (as well as African American men because they supposed have an increased risk of this disease), the paradigm shift undermines these recommendations. The logic by which black men are denied access to testosterone therapies is predicated on the historical myth that testosterone contributes to prostate cancer risk. In turn, I call for those responsible for penning these clinical guidelines to reassess the evidence underlying these recommendations in order to redress the differences in how these pharmaceuticals are distributed between black and white men. Of course, these recommendations would also be wise to note the potential dangers of testosterone therapies on cardiovascular disease to ensure that these drugs are not prescribed solely because race is removed from the exclusion criteria.

In the conclusion, I outline the implications that this dissertation has on two broad domains of research. First, I discuss how my work speaks to the sociological literature on medicalization and pharmaceuticalization. More specifically, I show that the growth testosterone replacement therapy industry is not only predicated on research and marketing efforts within sexual medicine, but also that this market's growth intersects with other biomedical contexts like prostate cancer research. By investigating this context, I show that the testosterone market has been racialized and, in turn, that the expansion of the testosterone market has impacted men of different racial groups in distinct ways. My work

provides social scientists with a complementary case to that of BiDil by documenting how race and pharmaceuticalization intersection in complex ways in testosterone research.

Second, I outline how my work affects scientific research on testosterone. I ultimately argue that those responsible for constructing the Endocrine Society's clinical guidelines on testosterone therapies need to revise their recommendations to advance a new policy that puts an end to race-based testosterone distribution practices. Using race to (not) disseminate biomedical treatments to patients may perpetuate social inequalities between white and black men. In this case, the Endocrine Society's recommendations unjustly deny important resources from black patients who may benefit from the use of testosterone therapies, despite the potential risks of using these drugs.

Chapter 2: The Racialization of Testosterone in Scientific Research

While popular accounts typically deem testosterone a molecular marker of masculinity, feminist science studies scholars argue that reducing testosterone to a "sex hormone" overlooks its capacity to influence a number of functions beyond sex-linked characteristics and, as I explain in more detail below, may even lead to dangerous models of human health and behavior (Fausto-Sterling 1985, 2000; Oudshoorn 1994; C. Roberts 2007; Fine 2017; R. Jordan-Young and Karkazis forthcoming). And though these patterns of gender essentialism are well-documented, very little research to date has outlined how testosterone is racialized in scientific research. Drawing from feminist science studies and literature on the molecularization of race, this chapter outlines how scientific constructions of racial differences in testosterone play a harmful role in contemporary biomedical and biosocial research. Based on a content analysis of 147 publications that evaluate population differences in testosterone, I demonstrate that, despite widespread claims that racial differences in testosterone exist among black, white and Asian populations, there is very scant evidence to support these assertions. This undermines the theory that racial differences in testosterone may help to explain discrepancies in various behavioral and biomedical outcomes, including racial disparities in prostate cancer.

My analysis also outlines the mechanisms that contribute to the racialization of testosterone, perpetuating the myth that race is biological. First, I describe how ambiguity in the application of population classifications function across the overall testosterone literature, within individual papers, and through citation practices. Second, I show how researchers' recycling of scientific datasets enacts testosterone as a racial construct through repeated testing of population differences. Lastly, I suggest that the absence of covariates,

widely used in the broader testosterone literature, but omitted in large subsets of racial difference testing, contribute to misleading claims about population differences in testosterone. Together, these mechanisms provide a basis for researchers to enact racial differences in testosterone scientific research, which perpetuate cultural myths about testosterone being a molecular marker of racialized masculinity.

The Molecularization of Race in Biomedical Research

In recent years, social scientists have detailed how race has become entrenched in biomedical research. While the Human Genome Project demonstrated that human beings are "99.9 percent" similar in genetic terms (Bliss 2012; D. Roberts 2011), a faction of the biomedical community still maintains that racial groups can be reliably distinguished through the use of genetic variables (Rosenberg et al. 2002). In part, this enduring use of race as a marker of group difference is motivated by state-sponsored mandates for medical research to be more inclusive of women as well as racial and ethnic minorities (Epstein 2007), but leading biomedical scholars, academic organizations, and corporations also play a vital role in perpetuating and proliferating the reification of race as a biological concept (Bolnick et al. 2007; Duster 2004; Kahn 2013; Pollock 2012; Reardon 2009).

A growing subset of this literature also documents that race and related populations classifiers, while broadening in use, are employed in increasingly inconsistent and ambiguous ways. On the one hand, this is not surprising, as racial and ethnic classifications are dynamic historical processes (Morning 2011; Omi and Winant 2014; D. Roberts 2011). Still, many biomedical scientists also define populations in heterogenous ways depending on the situational properties of their research program (Shim et al. 2014). For ex-

ample, Panofsky and Bliss (2017) find that genomic researchers frequently conflate racial, ethnic, continental, regional, and linguistic labels when classifying diverse samples, garnering scientific authority by framing health disparities through these loosely-defined distinctions. Yet, this ambiguity is not always easily recognized by the scientists themselves, as many struggle to define what "race" means when questioned about it directly (Bliss 2012; Fujimura and Rajagopalan 2011; Fullwiley 2007).

Absence also plays an important part in the re-circulation of race in biomedical research. As Frickel (2014) notes, absences can become intertwined with the complex information that is available in scientific studies, which ultimately come to shape the distribution of justice, social access and equity. For one, biomedical scientists often omit a definition of race or fail to justify its use in their research (Lee 2009; Shim 2002). By neglecting to take an ontological stance of what race "is," the meaning of molecularized group differences is never entirely clear when revealed through the complex 'web of causation' of contemporary multivariate models (Krieger 1994). While biological differences may be the result of environmental factors, researchers could just as easily deploy a genetic explanation (Darling et al. 2016). Through ambiguity and absence, race becomes reified as a risk-factor-in-and-of-itself (see Shim 2002). No explanation is needed for what causes biological difference; race instead becomes the surrogate for the cause. When scientists reflexively incorporate race into health disparities research (Bliss 2011, 2012), ambiguity and absence function to re-inscribe the fiction of race as a biological entity (M'charek 2013), shaping how media outlets and their readers come to mistakenly understand race as an essential property of human difference (Phelan, Link, and Feldman 2013).

Testosterone in Feminist Science Studies

While literature on the molecularization of race illustrates how racial essentialism shapes genomics research, feminist science studies scholars highlight the ways that gender essentialism shapes hormonal research. For example, Oudshoorn (1994) documents how pre-scientific ideas about sex differences shaped early 20th-century pharmaceutical research, shifting scientific understandings of sex from organs to chemical substances. In the process, testosterone became known as *the* masculine hormone, despite the fact that it circulates through and is crucially integral the overall health of *all* human bodies (Fausto-Sterling 1985, 2000; C. Roberts 2007).

Gendering testosterone in this way imbues the hormone with a great deal of explanatory power (R. Jordan-Young and Karkazis forthcoming). Fine (2017) details how biosocial scientists use testosterone to naturalize supposed gender differences in risk-taking by framing outcomes around stereotypical forms of masculinity like stock-trading rather than recognizing pregnancy as a form of risk-taking. Testosterone, in turn, is used to justify the structures that these behaviors map onto, mobilized to explain gender disparities in the finance industry or overall wealth gap at the expense of structural and historical factors. Although some biosocial scientists have integrated insights from feminist science studies to develop models that go beyond the pre-scientific assumptions of gender essentialism (Van Anders 2013), most of this work still relies on the notion that testosterone and masculinity are one and the same.

In health research, gendering hormones can have dangerous implications. For example, Krieger and colleagues (2005) argue that the failure to consider historical and socio-cultural factors, like gendered assumptions about “sex hormones,” misled clinicians

to distribute hormone replacement therapies to women as a treatment for menopause. By focusing on how these drugs were gendered, rather than as molecules that proliferate cell growth, including carcinogenesis, medical experts put thousands of women at increased risk of breast and ovarian cancer for nearly four decades. Perhaps echoing this history, the testosterone replacement therapy (TRT) market has now grown more than 12-fold worldwide since 2000 (Handelsman 2013)—a trend bolstered by the longstanding notion that TRTs can rejuvenate men's masculinity circulating through pharmaceutical marketing, scientific research, and popularized stories about the hormone (Marshall 2009a). Yet, the Federal Drug Administration (FDA) now warns that TRTs may actually contribute to a heightened risk of cardiovascular disease among men who take these drugs (FDA 2015). Though this matter is still hotly contested today (Huo et al. 2016; Morgentaler 2016; Morgentaler et al. 2016; Lin Xu et al. 2013), gender essentialism has played an important role in shaping how health experts and consumers conceptualize the potential risks surrounding these hormonal therapeutics.

The Racialization of Testosterone in Scientific Research

In this chapter, I explore how racial differences are enacted *through* testosterone in scientific research. While extant literature shows how racial essentialism is perpetuated in genomic research and how hormones are gendered, research on the racialization of testosterone is only now beginning to mount (Carlin and Kramer 2018; Gill-Peterson 2014; R. Jordan-Young and Karkazis forthcoming; Karkazis and Jordan-Young 2018). Although testosterone is different from most genetic markers due to the gendered assumptions that circulate alongside it, these two literatures overlap in that they show how groups are es-

sentialized through biology, demonstrating how this form of knowledge production is misleading and dangerous.

To help explain this, I draw from Pollock's concept of durable preoccupations. Pollock (2012) argues that long-held concerns about racial disparities in heart disease and biomedical experts repeated, yet continuously adapting, attempts to minimize those disparities has led to re-inscription of race as a biological entity. For instance, the notion that African Americans suffer from heart disease at higher rates than white people was one of the arguments used by NitroMed while they were marketing BiDil - the first ever racially-specific pharmaceutical approved by the FDA (Kahn 2003, 2013). Here, the point is that the enduring ideology that race is biological continues to transform alongside scientific practices, altering societal beliefs and the distribution of resources in ways that may perpetuate, or even exacerbate, health disparities in the process.

Unlike past work that uses specific diseases or health outcomes as a central frame, my work highlights the racialization of testosterone as a biomedical object, situating the hormone as an entity that scientists mobilize to explain various health and behavioral outcomes that vary between populations. On the one hand, biomedical researchers utilize testosterone as an explanatory mechanism across a diverse set of health outcomes, including research on heart disease, diabetes, metabolic syndrome, osteoporosis, polycystic ovary syndrome, and various types of cancer in both men and women. On the other hand, biosocial models use testosterone to explain patterns of crime, aggression, mental illness, marital instability, and antisocial behavior (Mazur and Booth 1998). Though biosocial research is not always racialized explicitly, the cultural transport of testosterone allows it to be mobilized in malicious ways (R. Jordan-Young and Karkazis forthcoming). And while

this chapter mainly focuses on the implications that racializing testosterone has on health disparities research, my findings could also be used to interrogate work using testosterone in misinformed ways in biosocial research as well.

Following Jordan-Young and Karkazis (forthcoming), I see testosterone as a "multiplici-T" that is enacted differently across various contexts; testosterone is not a singular entity, but an object that comes to mean different things in different circumstances. Akin to Mol's application of multiplicity (2002), testosterone may be relationally contingent on a consumer's administration of a TRT, the clinical context that a medical expert uses it to designate a health risk, or can be based on the technologies researchers use to measure the hormone. Entwining this past work with intersectional theory (Crenshaw 1989), I also focus on how testosterone becomes imbued with cultural assumptions of various social categories. While this chapter mainly highlights the ways that race and gender intersect with testosterone in biomedical research, I also discuss how age, body composition, and socio-economic status factor into scientific constructions of the hormone as well.

Data and Methodology

This chapter examines how scientists construct population differences in testosterone. To generate a sample, I searched all databases on ISI Web of Science for original, peer-reviewed research reports on racial and ethnic differences in testosterone, limiting to English language articles from 1980-2016. I chose this time range because the Web of Science Core Collection provides the most reliable data extending back to 1980. Next, I combined the search terms testosterone and racial/ethnic difference (including "racial dif-

ference," "ethnic difference," "racial variation," or "ethnic variation"). This search yielded a total of 55 results, only 26 of which were retained after searching each study for population comparisons. I used these 26 papers as "seeds," tracing their citations to snowball a larger sample. This process involved reviewing each paper to find racialized (i.e. population-specific) claims about testosterone. After using qualitative coding to identify all of the racialized claims in each paper, I developed a broader citation network where these racialized claims linked to other papers in the overall literature. To do this, I created an "edge list" in a spreadsheet where the citing paper was entered in one column and the cited paper into a second column. From this edge list, I constructed the citation network where nodes represent publications and ties correspond to racialized (or population-specific) claims about testosterone (Figure 2, described below). In total, my final sample includes 147 publications ranging from 1966-2017 (see Appendix I).

In addition to understanding how testosterone has become racialized, I wanted to understand if the literature as a whole supports claims about racial differences in testosterone. In the only meta-analysis available on this topic, Richard et al. (2014) compares the testosterone of black and white men using 14 studies, suggesting that at least a subset of the literature does find significant variation on some testosterone measures. However, building on insights from critical race theory, I hypothesized that the literature as a whole would *not* reliably demonstrate racial differences in testosterone. To aggregate the outcomes of studies, I conducted a content analysis focused on (1) outcomes relating to population differences in testosterone, (2) the size and demographic characteristics of each dataset, and (3) which controls, statistical tests, and measurements techniques were used across these studies.

While coding, I noticed inconsistency in the ways that group labels were used in addition to key covariates that would affect group comparisons. Thus, following grounded theory and abductive principles (Charmaz 2006; Timmermans and Tavory 2012), my subsequent analyses focused on outlining a series of theoretical mechanisms that contribute to the enactment of populations differences in scientific research. Two of the mechanisms (ambiguity and absence) have already been explicated in past studies in the science & technology studies literature (Frickel 2014; Panofsky and Bliss 2017). Building on the insights these studies offered, I examined the (in)consistency of labeling practices related to race, ethnicity, and other population markers throughout my sample. The third mechanism (data recycling) surfaced inductively from my content analysis. Together, these mechanisms help explain how testosterone has become racialized in scientific research. More generally, these mechanisms contribute to a more comprehensive framework that provides insights into how population differences are constructed through scientific practices.

Enacting Difference through Testosterone

The search for racial differences in hormones dates back to the days when testosterone was first discovered and eugenics research was still in its prime (Marett 1935). The most common theory circulating through the literature since consists of three components: (1) that people of African ancestry have the highest levels of testosterone; (2) that people of Asian ancestry have the lowest levels of testosterone, and (3) that people of European descent have testosterone levels that fall somewhere in between African and Asian descendants (e.g. Orwoll et al. 2010; Randolph et al. 2003; R. K. Ross et al. 1992; Sowers

et al. 2003; L. Xu et al. 2014). For shorthand, I call will refer to this discourse and set of comparisons as the "racial testosterone theory."

Measuring testosterone differences between black and white subjects is by far the most frequent comparison (see Table 1), comprising nearly 70% of my data (101/147 studies). In contrast, white and Asian groups are compared in approximately 31% of studies (46/147) and comparisons of black and Asian groups make up about 13% of the data (19/147). As summarized in Table 1, researchers compare the testosterone of black and white men at slightly higher rates than black and white women. In contrast, women are tested more frequently than men when researchers contrast white and Asian populations. Studies conducted on children, which focus exclusively on white and black subjects, are generally separated from adult populations, but do not necessarily divide boys and girls because testosterone does not vary significantly by sex before puberty.

This sex/gender discrepancy corresponds closely to how testosterone is implicated in two prominent health conditions. Since the 1940s, high levels of testosterone have been used to explain the etiology of prostate cancer (PCA) (Huggins and Stevens 1940; Huggins and Hodges 1941). Because African American men are usually shown to have higher rates of PCA than white men in the US (Simon Evans et al. 2008), researchers often argue that black men's (supposedly) higher testosterone levels help explain these disparities (R. Ross et al. 1986). Since the mid-1980s, a similar logic has been advanced for racial disparities in cardio-metabolic disease and bone mineral density, among other health outcomes, but to a much lesser extent (e.g. Araujo et al. 2008; Gapstur et al. 2002).

Table 1. Most Frequent Population Comparisons of Testosterone

	Studies Comparing Men (n=79)			Studies Comparing Women (n=63)			Studies Comparing Children (n=15)		
	Black/White	Asian/White	Asian/Black	Black/White	Asian/White	Asian/Black	Black/White	Asian/White	Asian/Black
Highest in Asians		2 (8.2%)	0 (0.0%)		2 (7.4%)	1 (9.1%)		0 (0.0%)	0 (0.0%)
Highest in Blacks	8 (15.1%)		3 (27.3%)	8 (19.5%)		1 (9.1%)	1 (6.6%)		0 (0.0%)
Highest in Whites	1 (1.8%)	5 (20.8%)		2 (4.9%)	5 (18.5%)		0 (0.0%)	0 (0.0%)	
Mixed	11 (20.8%)	2 (8.2%)	0 (0.0%)	2 (4.9%)	5 (18.5%)	4 (36.4%)	1 (6.6%)	0 (0.0%)	0 (0.0%)
Null	33 (62.3%)	15 (62.5%)	8 (72.7%)	29 (70.7%)	15 (55.5%)	5 (45.4%)	13 (86.7%)	0 (0.0%)	0 (0.0%)
Total	53 (100.0%)	24 (99.9%*)	11 (100.0%)	41 (100.0%)	27 (100.0%)	11 (100.0%)	15 (99.9%*)	0 (0.0%)	0 (0.0%)

* Totals do not add to 100% due to rounding

In studies conducted on women, testosterone is one among many hormones used to evaluate differences in the onset of menopause and the prevalence of polycystic ovary syndrome (PCOS), mainly between white and Asian patients (e.g. Carmina et al. 1992; Randolph et al. 2003; Sowers et al. 2003). Here, testosterone is thought to explain differences in hair and hirsutism (i.e. "male-pattern" or "excessive" hair growth in women), which is one of the clinical criteria for PCOS. Despite scant and contradictory evidence to support their claims, clinicians often assume that testosterone and hair vary by race, which perpetuates population-specific diagnostic criteria for PCOS (Carlin and Kramer 2018).

Comparisons of black and Asian populations are conducted much less frequently, regardless of sex/gender, and these analyses almost uniformly occur when black, white, and Asian populations are juxtaposed in the same article. This suggests that white populations are considered a default or normative group. This subset of studies sometimes explores testosterone as a potential mechanism to explain PCA disparities in men of different racial groups, but, like the overall literature, may also just attempt to establish biological variation between racial groups without any clear theoretical justification. Without any rationale for what contributes to potential differences between groups, testosterone can easily be theorized as an outcome to environmental exposures *or* as the result of an inherent genetic difference. The majority of the literature either suggests differences are genetic or provides no reason at all to explain why differences arise.

The remainder of the literature, constituting 14.3% of my dataset (21/147), frames comparisons around ethnic or geographical difference rather than race per se. Of the 14 remaining studies on men, three compare African Americans to black Nigerian or black

Caribbean men, seven compare ethnic groups of "non-industrialized" participants in Africa, South America, and South Asia, and three compare white Europeans to Arab or Indian men. The remaining publications on women focus exclusively on PCOS differences between white (or Caucasian) women in the US and Mexican-American, "Moslem," Middle Eastern, and/or Italian women. While the racial testosterone theory is the most widely tested set of comparisons in my dataset, the literature does not employ consistent or reliable population classifications (see below).

Still, researchers widely claim that testosterone varies by race despite this assertion not being supported by the literature as a whole. As Table 1 summarizes, only a minority of the literature finds population differences in testosterone. Of the 147 studies in my sample, 77 (or 52.3%) yielded null results with an additional 38 (or 25.8%) offering mixed results. Here, mixed results are of two kinds: One type refers to studies that compare multiple populations, finding differences for some groups, but not for others. For example, Hill and colleagues (1976) find testosterone differences between Bantu and Caucasian girls at age nine, but then offer null results when comparing 20-30 years-old Bantu, Japanese, and Caucasian women. Mixed results also refer to studies that find contradictory results when comparing multiple types of testosterone. For instance, Richard et al.'s (2014) meta-analysis of black and white men finds free testosterone differences, but offers null results when comparing total testosterone between these groups.

Only 32/147 studies (or 21.8%) demonstrate clear population differences in testosterone for all the groups they compare. Of course, clear evidence of difference in one study might directly contradict findings from another. While the racial testosterone theory argues that all Asian groups have the lowest testosterone levels of the three groups out-

lined above, Orwoll et al. (2010) finds that both Japanese and Hong Kong samples had markedly higher testosterone than white and black comparisons in the US, Sweden, and Tobago as well as their Asian American participants. As I show below, the fluidity of these labeling practices further deteriorates any concrete basis that the racial testosterone theory has to stand on.

To build on these findings, Figure 2 more clearly demonstrates how much of the literature contains null or mixed results as well as the stature that certain studies have in the overall literature. In this network, each *node* (or bubble) represents a publication in the data while the *ties* connecting studies together correspond to racialized (i.e. population-specific) claims about testosterone. Each tie has an arrow, which corresponds to the direction of the citation (i.e. from citing article to cited article). The size of the nodes indicates the number of times the publication has been cited by others in the network (known as the in-degree centrality in the network analysis literature). In this network, colors align with outcomes of testosterone comparisons by population. Red, orange, and green nodes respectively signify null findings, mixed results, and outcomes that demonstrate population differences. White nodes stand for papers cited as evidence for population differences in testosterone without offering empirical data to back that claim.

While the majority of the studies are visibly null results, the most highly cited paper in the network (i.e. the largest green node) finds that black men have higher levels of testosterone when compared to white men (R. Ross et al. 1986). The next two largest nodes also show racial differences in testosterone on some comparisons, but not others. Ellis and Nyborg (1992) demonstrate that black men have higher total testosterone than Non-Hispanic white men, but find no differences when comparing Hispanic, Asiatic/ Pa-

cific Islander, and Native American men. Gapstur et al. (2002), on the other hand, only compares white and black participants, but run analyses to compare these groups at three

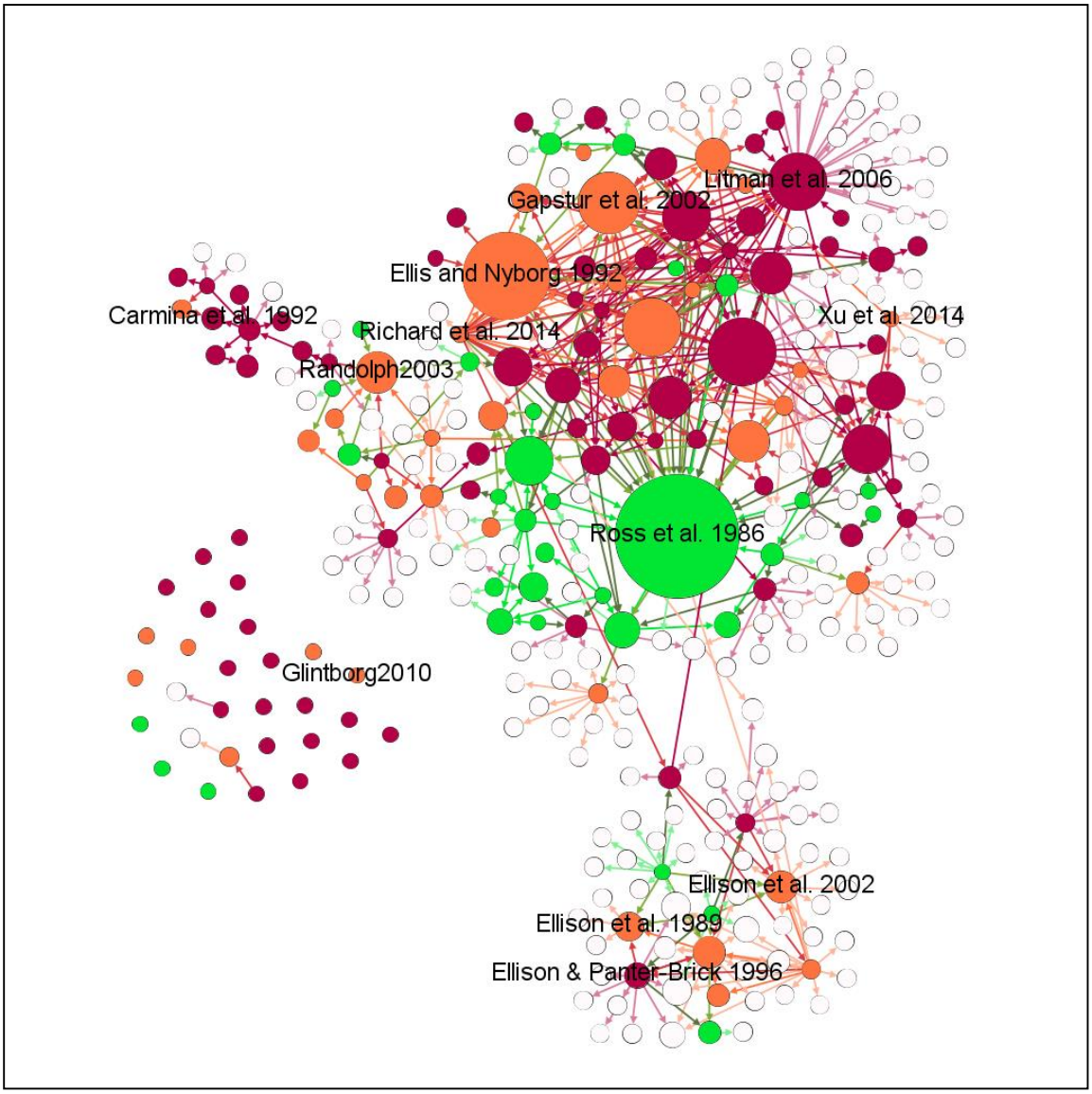

Figure 2 shows a citation network where nodes represent studies and ties stand for population-specific claims about testosterone. Red and orange nodes represent null and mixed outcomes respectively. Green nodes symbolize studies that find population differences. White nodes correspond to publications used as evidence by other studies despite offering no population comparisons of testosterone.

different time points over the life course. They find that black men have higher unadjusted free testosterone at one time point but found no other differences in free or total testosterone in any of the other five time points.

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This is one way that absence functions to reinforce the racialization of testosterone. The disproportionate citation of studies that support the racial testosterone theory relies on the selective omission of roughly 80% of the literature. Furthermore, when publications cite studies with mixed findings, like Gapstur et al. (2002), authors ignore the fact that the study proffers more evidence *against* racial differences in testosterone than it does in favor of that claim.

Here, my argument is not that researchers are aware of every study in the literature and strategically ignore those results, though it may be the case some researchers do this. Instead, I argue that the ideology that race is molecularized and, more specifically

that testosterone is the biological vector that animates racial differences, is already enough rationale to empower false positive findings (like the three studies mentioned here) to circulate at disproportionately higher rates than the null or negative findings that comprise the majority of the literature. As I outline below, there are at least three mechanisms that work alongside this ideology.

Ambiguity/Slippage

In this section, I outline how testosterone researchers use ambiguity in population labeling in the context of testosterone research. Panofsky and Bliss (2017) find that genomics research has become increasingly ambiguous through the indiscriminate blending of classification schemes at the field level, within articles, and within-population comparisons. My analyses corroborate their work by demonstrating that testosterone researchers also deploy diverse, ever-changing classifications when making population comparisons. In testosterone studies, population slippage manifests in three ways: across the literature, within articles, and through citation practices.

First, looking across the literature, Table 2 shows that testosterone researchers use more than 85 distinct population classifications that shift between racial, ethnic, continental, national, regional, linguistic, and religious designations to demarcate group differences. This means that at least one new population classification is introduced in every two articles. While concerns about racial difference endure, the basis of population differentiation is not a constant, but instead a logic that has to be continuously used to make and remake populations during analyses. Population slippage also manifests within articles. This ambiguity is most obvious when groups fall outside of the U.S. Census' stand-

ardized racial classifications. For example, in their study of a multiethnic group of PCOS patients in Sweden, Glintborg et al. (2010) categorizes Pakistani participants as Middle Easterners, excluding them from Asian participants, despite the fact that the authors themselves suggest this group is most appropriately designated as South Asian. Later in the article, the authors need to clarify that a study they cite includes Pakistani women in a comparison of South Asians and Caucasians, even though this goes against their own classification schema. Here, the authors conflate racial, ethnic, national, and regional distinctions, recreating populations anew in each sequential instance.

Slippage also happens through citation practices. Like in the previous example, the definition of a group can change when other studies highlight a different aspect of a study population's identity. For example, Carmina et al. (1992) compares the testosterone of Italian, Japanese, and U.S. women. However, papers that cite the Carmina paper often re-classify the latter two groups as either Asian or Hispanic when it suits their study's needs (Carlin and Kramer 2018). Similarly, researchers make claims about geographically-specific racial groupings like African Americans, but then cite studies that use samples outside of the US to support claims about more general racial difference. The terms that are used to define each group tend to vary study-by-study depending on the classifications that are most applicable for the publication's specific objectives.

Perhaps the best example of population slippage comes from the anthropological literature. Ellison et al. (2002) compare Lese horticulturalists from rural Congo, Tamang agro-pastoralists from Nepal, Ache foragers from Paraguay, and residents of (Boston) Massachusetts, USA. While the lead authors' past publications use the same data to com pare Lese villagers and Efe pygmies (Ellison, Lipson, and Meredith 1989), all mention of

Table 2. Population Terms Used Across Dataset (n=85)				
Ache	Chinese American	Indian	Middle Eastern	Singapore-Chinese
African/Afro-American	Chinese migrant	Indian-Trinidadian	Moslem	South Asian
African-Caribbean	Datoga	Italian	Native American	South African
African-Trinidadian	Dutch	Japanese	Native Hawaiian	South American
American Indian	East Asian	Japanese American	Nepalese	Southern European
Arab	East Indian	Jamaican	Nigerian	Sri Lankan
Ariaal	Efé	Kami	Non-Australian	Sumburu
Asian/Asiatic	Euro-Caucasian	Kavango	Non-Hispanic Asian	Swedish
Australian	European White	Kenyan	Non-Hispanic White	Tamang
Austrian	Filipino	Korean	Non/Industrial	Tsimane
Bantu	German	!KungSan	North American/US Black	Tobagonian
Black	Gujarati British	Laotian	North American/US White	Turkana
British	Gujarati Indian	Latino/a	Occidental	Vietnamese
British Asian	Hadza	Lese	Oriental	Western
Caucasian	Haitian	Maori	Pacific-Islander	Western European
Caribbean-Hispanic	Hispanic	Mestizo	Pakistani	White
Chinese	Icelandic	Mexican American	Scandinavian	Zaire

Efe heritage is removed from the 2002 article where subjects are simply deemed Congolese. On the other hand, Kami participants recruited in Ellison and Panter-Brick (1996) are excluded from the Nepalese sample in the later study. Despite the authors claiming this group is one of "four populations that are geographically, genetically, ecologically, and culturally distinct" (Ellison et al. 2002, 3252), no further demographic information is provided for the Massachusetts sample. This suggests that people from Massachusetts are a genetically distinct group living in non-segregated neighborhoods of similar socioeconomic backgrounds. This, of course, is not the case.

Researchers also engage in citation slippage by conflating multiple claims without providing evidence to support their entire argument. To claim, for example, that testosterone explains racial disparities in PCA, researchers should presumably offer: (1) evidence to support testosterone's causal role in prostate carcinogenesis; (2) evidence for racial differences in testosterone; and (3) evidence for racial disparities in PCA. I found numerous cases where claims are only partially supported by the citations provided. This tendency is especially difficult to evaluate when several citations are combined at the end of a claim. For example, Litman et al. (2006, 4326–27) claims that racial/ethnic differences in androgen levels may help explain a host of outcomes from body composition to fracture incidence, but the majority of the citations used do not even mention testosterone in the publication.

This form of citation slippage has an interesting structural feature as well, as evidenced by the various "fan structures" in Figure 2. In the bottom and top right-hand corners of the graph, shaded nodes with multiple ties pointing outward to white nodes that do not connect to other nodes in the network (i.e. fan structures) mean that the shaded

publications engage in citation slippage because their racially-specific claims about testosterone are not supported by the empirical evidence they provide. While these two clusters are the strongest examples, fan structures can be found extensively throughout the entire network.

Data Recycling

The second theme that emerged was the practice of data recycling. Of 147 publications, 55 (or 37.4%) contained data that was used in at least one other study in my sample. Oftentimes, these are multi-sited or nationally representative samples designed for use across multiple research teams. My interest in these studies is not to suggest that anything is inherently wrong with re-using these datasets. Instead, following Hatch (2016, 64–68), I mean to suggest that the public availability of these datasets allows for both health outcomes (like metabolic syndrome in Hatch's case) and biomarkers (like testosterone) to become racialized constructs. These dataset's continued re-use simply provides a mechanism to re-enact racial difference through the routinized use of population variables.

The most commonly recycled data come from the National Health and Nutrition Examination Survey (NHANES), which was used in nine studies. While the NHANES data were most often used to compare the testosterone levels of Non-Hispanic White, Non-Hispanic Black and Mexican American men and boys, the results were not always consistent across publications: four articles proffered null results, four others had mixed results, and only one established a clear difference between populations. While many disparate results likely arise because different cohorts are being compared (1988-1994, 1999-2004, 2011-2012), the NHANES also provides access to free and total testosterone

measures for various age groups that researchers can pick and choose how to analyze. For example, two studies using the same cohort end up concluding different groups have higher testosterone, presumably because they use different age ranges and sample weights in their study (Mazur 2009; Rohrmann et al. 2007). Here, the testing of multiple outcomes, a phenomenon known as "researcher degrees of freedom" (Simmons, Nelson, and Simonsohn 2011), may also lead to the proliferation of false positive findings (see more below), evidenced by the mixed findings across NHANES studies.

Seven publications recycled data from the Study of Women's Health across the Nation (SWAN)—a study that recruited over 3,000 women from various racial/ethnic minority groups from several U.S. locations. Most studies demonstrate racial/ethnic variation by showing median testosterone levels in tables, alongside other biomarkers, without elaborating on what the results mean in their discussion sections (e.g. Sowers et al. 2003). The notable exception is Randolph et al. (2003) who find, after adjustment for several covariates, Caucasian, Chinese, and Japanese women had higher levels of testosterone than Hispanic and African American women. Interestingly, the SWAN study, a particularly large dataset relative to others in my sample, contradicts the racial testosterone theory by finding that (1) white and Asian women have similar testosterone levels and (2) that African American women have testosterone levels that are significantly *lower* than the other two groups. What is concerning, however, is that three other studies which report only the unadjusted median levels of testosterone (like Sowers et al. 2003) would lead readers to think that Chinese and Japanese women actually have the lowest overall levels of testosterone of these groups. This contradiction is one clear example of

how excluding covariates can dramatically change the results of population comparisons – a point I return to later in this chapter.

In addition to inconsistency in outcomes across publications, recycling data magnifies the practice of population slippage. For example, Xu and colleagues (2014) compare U.S. men with Chinese men. In this study, U.S. subjects are the aggregation of Non-Hispanic White, Non-Hispanic Black, and Mexican American men from the NHANES dataset. Though eight other studies in my sample partition these groups, Xu et al. (2014) collapse these participants together and argue that total testosterone differs between Western and Asian populations, even though different measurement techniques (or assays) were used to evaluate testosterone for each group.

Recycling data brings to light a number of insights about how difference is enacted in biomedical research. First, these datasets provide a mechanism for researchers to enact testosterone as a racial construct while their continued re-use is the conduit through which scientists continually re-make those differences. Second, data recycling elucidates the ways that population slippage emerges across the literature by demonstrating how population labels are applied in distinct ways across publications that use the same data. This inconsistency does not necessarily end with ambiguity in population labeling, as researchers may also selectively report certain testosterone measures (free or total) and leave out others that offer null results. Of course, the repeated testing of the same outcome also increases the chance of reporting false positive findings, which Ioanniddis (2005) argues may characterize a majority of contemporary biomedical findings. These issues related to this broader phenomenon, known as the replication crisis, manifest even more clearly when conceptualized through the third mechanism: absence or omission.

Absence/Omission

Testosterone exhibits variation across a number of different timelines. For both men and women, testosterone can change over the course of the day by 20-50% with the highest levels occurring shortly after waking and the lowest levels in the late evening hours (Bremner, Vitiello, and Prinz 1983). Variation can occur over the life course in complex ways as well. Testosterone differences between boys and girls are negligible from birth until the onset of puberty (Hibel et al. 2007; Kuijper et al. 2013). At that time, adolescent boys' testosterone typically triples before peaking in their early-20s. While testosterone remains fairly constant for the next two decades of life, men's testosterone usually declines by roughly 1% each year after the age of 40 (at least in Western or industrialized countries). Although women, on average, tend to have about 1/10th the testosterone levels that men have in their 20s, women also exhibit a sharp increase in testosterone during their adolescent years before a nadir in their mid-40s and subsequent incline during menopause (Handelsman et al. 2015).

When making population comparisons, researchers do not always account for temporal variations appropriately. 36.7% of studies in my sample (54/147) failed to indicate that collection times were standardized, opening the door to systematic temporal variation between subgroups. Further, 32.7% of studies in my sample (48/147) did not measure age or found a significant difference in age between groups and opted not to adjust for this in their models. In both cases, the absence of systematic controls raises the possibility that a statistically significant population difference is actually a product of deficient study design.

Biomedical researchers also find that testosterone levels show a robust relationship with body composition. For men, total testosterone tends to decrease as body mass index increases, suggesting that testosterone declines with age as men put on weight (MacDonald et al. 2009). On the other hand, research shows "obese" or "overweight" women have significantly higher testosterone levels (Lim et al. 2013). As with the absence of controls for age and diurnal variation, scientists fail to account or control for body composition in 50.3% of my dataset (74/147).

Lastly, testosterone researchers fail to systematically account for socioeconomic status (SES) in population comparisons. Multiple studies have found that the risk of hypogonadism in men (i.e. clinically low testosterone) is highest among those in lower SES brackets (e.g. Hall et al. 2008). Hypogonadism is also associated with deleterious health outcomes like diabetes, metabolic syndrome, and cardiovascular disease (Brand et al. 2010; Corona, Rastrelli, et al. 2011; Ding et al. 2006), which disproportionately impact economically disadvantaged groups (National Center for Health Statistics 2017). Again, large subsets of the literature fail to account for SES and chronic health conditions: only 42.2% of studies (or 62/147) controlled for or excluded participants with heart disease, diabetes, and metabolic syndrome while only 7.5% of studies (or 11/147) measure SES in at least some variation (see Appendix II).

Given that most studies comparing two or more (racial or ethnic) populations find only miniscule effect sizes when comparing populations, the absence of these four covariates is remarkable because each variable could potentially account for more variance than the between-population effects observed in any given study. In this sense, absences are generative in that they increase the likelihood that false-positive population differ-

ences are reported. Seen through the lens of the replication crisis (i.e. the concern that most scientific findings are false positives and thus not reproducible), this lack of systematicity may help explain why a subset of the literature finds statistical differences in testosterone by population; poorly conducted research magnifies the chances of finding false positive results (Ioannidis 2005). Likewise, the widespread practice of subgrouping (X. Sun et al. 2012), especially when implemented to divide populations without any theoretical rationale, contributes to the increased likelihood of publishing false-positive findings.

The absence of SES, in particular, also offers important insights about the politics of this literature. Causal models in biomedical research often depend on the problematic binary of "nature vs. nurture" when providing explanations for health disparities; like a door on a hinge, researchers can easily swing from an environmental to a biological explanation when theorizing why racial health disparities developed in the first place (Darling et al. 2016). For example, Orwoll et al. (2010) suggest that socio-ecological factors like "diet, environment, chemicals, climate, physical activity, smoking, and social status" (E157) all have the potential to contribute to population variation in hormones. Yet, by the end of discussion section, the authors already fall back into conflating race with genetic influences that they argue could help explain the existence of hormonal differences underlying disparities in bone mineral density and fracture risk.

While both sides of the bio/social continuum are enacted to speculate on what factors could contribute to hormonal differences, the fact that only 10% of the literature includes measures of SES suggests that biomedical researchers have strong presuppositions about which factors they believe have explanatory power and which do not. Although hormonal differences could be attributable to social and/or environmental phenomena,

the absence of socio-economic measures reveals that these factors are either not enough of a priority to be included in the final analyses or that they do not actually matter enough to allocate research funds to collect that information in the first place. Biomedical researchers must strategically forget about economic and racial oppressions that contribute to environmental differences of the populations they compare. The continued pursuit of biological differences further entrenches the project of molecularizing race, steering resources away from intervening on the structural factors that many minority groups already know contribute to their health in deleterious ways (see Shim 2014).

Conclusion

This chapter explored the ways that population differences in testosterone are (re)constructed by biomedical researchers. In the first section, I outlined the most durable preoccupations with racial difference: researchers widely claim those of African descent have the highest levels of testosterone, those of Asian descent have the lowest levels of testosterone, and those of European descent have testosterone levels somewhere in between. My findings show that this "racial testosterone theory" is not supported by the literature as a whole, as the majority of the literature contains null or contradictory findings.

Building on these findings, I argue that absence shapes patterns of citation bias in the testosterone literature, perpetuating the myth that there are racial differences in testosterone. Notably, my findings conflict with Richard et al. (2014), a meta-analysis that suggests black men have higher levels of total testosterone than white men. However, my sample of 53 studies comparing black and white men, rather than just 14 in that meta-analysis, provides a more robust basis to reject the racial testosterone theory while also

showing how the omission of null or contradictory results perpetuates this claim. Of course, to suggest that establishing biological differences between racial groups is an empirical matter ignores the ways that race is both historically and contextually contingent (Morning 2011; Omi and Winant 2014; D. Roberts 2011).

To the contrary, this chapter demonstrates that population differences in testosterone hinge on a set of mechanisms that researchers use to perpetuate the myth of racial differences in testosterone. The ambiguity of population classifications occurs across the literature, within scientific articles, and through citation slippage. Data recycling provides both a mechanism to enact testosterone as a racial construct and to proliferate the testing of racial differences for various health outcomes. Finally, the absence of covariates, routinely used in the broader testosterone literature, but consistently omitted in population comparisons, increases the likelihood of false positive findings, further biasing an already conceptually flawed literature.

Together, these findings provide evidence that the racial testosterone theory is a myth. Re-writing this fiction is important for two reasons. First, the racial testosterone theory was an integral component of some of the most thinly veiled scientific racism of the latter half of the 20th century. Rushton's (1995) neo-eugenic theory explicitly argued that racial differences in testosterone explain racial differences in crime, intelligence, and social deviance. Although no studies in my sample explicitly cite Rushton's work, the racial T hypothesis still circulates through psychology, sociology and criminology journals today (Ellis 2017; Mazur 2016; Mazur and Booth 1998).

In terms of improving health disparities research, re-conceptualizing the racial testosterone theory allows biomedical researchers to focus more squarely on policy inter-

ventions that matter. While I do not intend to foreclose the possibility that scientists can learn valuable lessons from researching testosterone, this chapter demonstrates that economic, sociological, and environmental causes are largely absent from how researchers conceptualize population health disparities. Without understanding the fundamental causes of health (Link and Phelan 1995), using biomarkers to explain racial disparities inevitably re-inscribes race as essential property of human difference.

The durable preoccupations outlined in this chapter do not just have the potential to re-inscribe testosterone as a molecular marker of masculinity, but perpetuate specific constructions of essentialism. Testosterone has a history of being the molecular marker of masculinity, but embedded within these discourses testosterone is also highly racialized. Because of the conceptual powers that testosterone has in and outside of scientific literature (R. Jordan-Young and Karkazis forthcoming; Karkazis and Jordan-Young 2018), scholars must attend to the ways that testosterone is used to enact specific forms of racialized masculinities relating to health, athletics, crime, mental health and familial relationships, among others. These stereotypes direct resources away from appropriately combating various forms of inequalities, especially in the domain of health disparities. By t/racing the assumptions that underpin this project, we can move towards a more critically attuned model of studying health outcomes; one that does not depend on the fiction of race as a biological entity.

Chapter 3: Reassessing Racial Disparities in Prostate Cancer

The claim that African American men die from prostate cancer (PCA) at two to three times the rate as white Americans is widely cited throughout the biomedical and public health literature (DeSantis et al. 2016; Siegel, Miller, and Jemal 2017). Yet, a review of leading "gold-standard" studies on this topic suggests that disparities in PCA mortality between white and black men are consistently explained by group socioeconomic differences (Bach et al. 2002; Simon Evans et al. 2008; Peters and Armstrong 2005; Sridhar et al. 2010). Despite this being the case, many PCA researchers still believe that racial differences in testosterone, as well as a host of other biological markers that supposedly differ between these two groups, drive PCA disparities and, as a result, require racially-specific biomedical interventions. For example, PCA researchers use the racial health disparities discourse to justify racially-specific clinical screening procedures (Morgan et al. 1996; J. Moul 2000) and, more recently, the development of novel pharmaceuticals for specific racial groups (S. Freedland 2018).

In this chapter, I use insights from critical race theory and science & technology studies to examine how the scientists construct PCA disparities between black and white men. To start, I give a brief overview of how past work has examined the construction of racial health disparities research and the development of racialized biomedical interventions like BiDil. Next, I draw from existing work in at the intersection of critical race theory and agnotology, pointing how that the biomedical researchers' proclivity to focus on molecular explanations to describe health disparities comes at the expense of addressing broader structural concerns. I then move into my analysis, outlining how these insights apply to the context of PCA research. My findings are threefold.

First, I provide an overview of the racial PCA disparities literature, focusing specifically on the tensions that exist between studies that emphasize socioeconomic differences compared biological risk factors. Akin to Chapter 2, I find that, while researchers do discuss some types of social factors like equal access to healthcare, they largely omit socioeconomic status from their analytic models. Following from this, I detail how researchers employ the discourse of "aggressive biologies" to characterize black's men PCA risk in molecular terms. More specifically, black men are thought to have higher levels of testosterone, prostate-specific antigen and shorter androgen receptors, which are thought to lead to earlier onset of disease, more aggressive tumors and worse overall survival. Lastly, I outline how the molecularization of these disparities influences clinical practice, including through the use of group-differentiated prostate-specific antigen testing, racially-specific treatment guidelines, and variability in the distribution of pharmaceutical and surgical interventions.

To end this chapter, I follow Wendy Chun (2013) by arguing that race is a technology that PCA researchers use to mobilize racialized biomedical interventions. By allowing race to be simultaneously biological *and* socially constructed, scientists bypass ontological debates about what race is and instead solely focus on what it allows them to do, including develop racially-specific biomedical interventions on shaky scientific evidence. Furthermore, drawing from work in the field of agnotology (Bowleg 2017; Mills 2007; Sullivan and Tuana 2007), I contend that the racialization in PCA research hinges on a structure of (white) ignorance that omits the contemporary conditions and historical legacy of structural oppression in communities of color in the United States and abroad. By suggesting that black men have inherently more aggressive biologies, PCA research-

ers prioritize molecular explanations over structural factors without necessarily excluding either possibility. In doing so, they perpetuate the misallocation of funding towards group-differentiated interventions that low income that shift funding away from policy interventions that are most likely to address the structural inequalities that black men disproportionately face in the US today.

Racial Health Disparities in the Sociogenomic Era

Over the course of the last two decades, epidemiological research has played a major role in the rise of what Catherine Bliss calls the sociogenomic paradigm (Bliss 2012, 2018). Within this paradigm, many aspects of the world, including the etiology of racial health disparities, are seen as the result of an interdependent mix of social *and* biological, especially genetic, factors. Many believed that the search for biological differences between "races" would end after the Human Genome Project found that no biomarkers could reliably distinguish between such groups, but the sociogenomic era has given way to a proliferation of population-based scientific research that incorporates biological variables (Bliss 2012, 2018; Panofsky and Bliss 2017). While corporations, governmental agencies, and academic institutions have all played an important role in the re-emergence of race-based scientific inquiry (Bliss 2012, 2018; Epstein 2007; D. Roberts 2011; TallBear 2013; Wailoo, Nelson, and Lee 2012), this chapter focuses on the how race is conceptualized in health disparities research on PCA.

In epidemiological research, racial health disparities are typically studied through the use of multi-factorial models where both biological and socioeconomic processes play apart in shaping health outcomes. In these models, scientists work to untangle a

complex 'web of causation' where multiple determinants are used to create and assess risk profiles for individuals and groups based on the routinized use of race, gender and socioeconomic status (Shim 2002). As Shim and Thomson (2010) argue, the flexibility of such models allows researchers to carry out their research without making overt or specific claims about the causal priority of disease etiology, which allows researchers to work collaboratively with scholars in adjacent fields that prioritize different techniques and agendas. In fact, as the 20th-century progressed, epidemiologists have shifted their focus away from researching the causes of disease progression toward focusing on medical interventions and other proximate outcomes (Krieger 1994). In the process, a rigorous attempt to operationalize the *social* side of these multi-factorial models has largely fallen to the wayside before efforts to redress these shortcomings in recent years (Krieger 1994; Shim 2002).

Likewise, the meaning of race in health disparities research is ambiguous (Panofsky and Bliss 2017), used to advance highly heterogeneous ends based on the broader initiatives of research programs and the fields they are embedded within (Shim et al. 2014). As Shim (2002) has shown, epidemiologists often employ race and other population classifiers in highly routinized ways. For example, scientists may include race in a multifactorial model to control for its effects, but without clarifying what race means (i.e. whether it is a biological difference or a proxy for societal conditions that differ between racial groups). In doing so, researchers also fail to think about why race is theoretically relevant (Fullwiley 2007; Lee 2009; Montoya 2011; Panofsky and Bliss 2017), which means that when racial differences do arise they can just as easily be interpreted as innate differences or the result of structural factors. Accordingly, race is not only a *multiplicity*

in that can be enacted in diverse ways across a variety of contexts (M'charek 2013), but is also a *technology* that is used to 'do things' without researchers needing to take on strong ontological stances on whether race is biological or social (Chun 2013). As I argue below, race is a technology that PCA researchers employ to advance racialized biomedical interventions.

By far, the most well-known example is the pharmaceutical BiDil - a medication marketed for the treatment of congestive heart failure in and only in African Americans (Kahn 2008, 2013; Pollock 2012). As Kahn (2008) notes, BiDil was never a drug that was designed to work better for African Americans compared to other racial groups. Instead, BiDil was repurposed and tested exclusively in African Americans after the drug originally failed to demonstrate efficacy in a general population, which allowed BiDil to lock down monopoly patent control until the year 2020. Here, race is used as a technology to produce a (profitable) difference. Because racial difference is already legitimated as an ideology of difference, drug developers do not need to justify the biological mechanisms that are presumed to vary between white and black consumers; race works to fill that gap. Furthermore, as Kahn (2003) argues, the discourse that African Americans suffer from heart disease at twice the rate of whites in the US helped propel BiDil's marketing campaign. As his analysis uncovers, however, the claim that black men suffer from cardiovascular disease at higher rates is wrong (i.e. there is no evidence to support that claim) and, in turn, misleading: "Without this statistic, the impetus to find a biological explanation for differences between blacks and whites is lost" (Kahn 2003, 481) and so too is the logic for developing and marketing a racialized pharmaceutical to treat a disparity that does not exist.

The fact that scientists were willing to overlook conflicting or contradictory evidence to advance racialized interventions hinges on structures of ignorance and absence (Mills 2007; Sullivan and Tuana 2007). As McGoey (2012) argues, ignorance can serve as a productive asset that allows individuals and institutions to consolidate resources, deny liability, and establish expertise in ongoing scientific controversies. Others have shown that corporate interests shape knowledge production in debates about public health by predicating research on probabilistic models and individualized risk factors rather than concerns over the consumption of known carcinogens, exposure to environmental toxins, or the broader conditions of capitalism that reproduce racial and classed inequalities in health (Krieger 1994; Michaels and Monforton 2005; Proctor 1996; Sanabria 2016).

As Bowleg et al. (2017) contends, the widespread and uncritical use of the term "health disparities" in U.S. public health research also reflects an epistemology of ignorance that functions to bolster white privilege (see also Mills 2007; Sullivan and Tuana 2007). When researchers uncover racial health disparities, an array of potential explanations can be advanced to frame why those disparities exist and what can be done to minimize their affects (Shim and Thomson 2010; Weatherford Darling et al. 2016). For example, Shim (2002, 2005) notes that public health scholars tend to explain racial disparities in heart disease through biological or cultural lenses. When employing biological explanations, the onus of poor health is displaced back onto (poor) people of color because of their genetics. While these discourses clearly molecularize race, shifting the responsibility for managing risk back onto individuals rather the public health officials that may have a more direct impact on the structures that shape health outcomes (see Rose 2007),

researchers also have to ignore the broader landscape of racial/classed subordination that drive racial health disparities when making these claims in the first place.

Beyond the context of PCA, researchers have identified a number of mechanisms, both interpersonal and structural, that drive racial health disparities. Perhaps, the most elegant synopsis is Mindy Fullilove's model of serial forced displacement (Fullilove and Wallace 2011). Fullilove argues that a series of policies implemented over the course of the past three centuries, including segregation, redlining, deindustrialization, mass criminalization, and gentrification, have disproportionately impacted people of color throughout the United States. These factors have historically deprived black Americans, for example, of the same capacities as their white counterparts to accrue social and economic capital that translates to better jobs, income and the accumulation of wealth, which, in turn, negatively affects health outcomes from birth (Williams and Sternthal 2010; Williams et al. 2010). This includes, but is not limited to, differing access to resources such as education, healthy foods and proper healthcare play a cumulative role in exacerbating racial disparities over the life course (Mulia et al. 2008; Shuey and Willson 2008). In addition to these structural factors, people of color also experience various forms of racial discrimination that influence mental and physical health outcomes, typically even after controlling for group differences in socioeconomic status (Williams and Mohammed 2009; Williams, Neighbors, and Jackson 2003).

Even when social or structural proxies are included in health disparities literature, researchers often fail to fully define and/or theorize what socioeconomic status is and how it impacts health outcomes. For example, Shim (2002) finds that socioeconomic status is typically included in multi-factorial models as an individualized variable that is op-

erationalized using proxies like income, occupation or education. When these measures are included into studies that assess racial disparities (usually as a control variable), it is often assumed that these variables capture all of the social or structural effects, despite little discussion of what may *not* be measured, including variation in socioeconomic status during childhood or additional economic hardships that black families are more likely to experience even when they have similar incomes (Roberts 2011). Instead, the residual of these models is interpreted as the result of some biological or genetic influence - a common methodological error referred to as residual confounding (Kaufman, Cooper, and McGee 1997). In a sense, these errors show that scientists are often more focused on making sense of differences rather than robustly understanding what the actual factors – whether biological or structural – that drive health outcomes are in the first place.

While social scientists are familiar with the structural models outlined above, Bowleg's (2017) point is that biomedical scientists often fail to take these factors into account when developing models that examine racial health disparities. When scientists advance claims about racial health disparities, they often ignore (either intentionally or by just not allocating the space to discuss these possibilities) how historical and structural factors disproportionately impact the health of racial and ethnic minorities. Worse, when scientists advance claims that biological differences between racial groups help to explain differences in health outcomes, they advance policy recommendations that direct funds away from the structural interventions that are most likely to have an impact on these groups' long-term health. In the burgeoning era of precision medicine, pharmaceuticals and other individualized treatments are more frequently proposed as a means to manage individual or, more accurately, *group-differentiated* risk. Unfortunately, these personal-

ized treatments typically fail to translate to groups who cannot afford health care, leaving (white) individuals who can afford them as the primary beneficiaries. Here, ignorance (i.e. a *lack* of information) perpetuates a structure of racialized (dis)advantage.

To some extent, others have already documented how racialized epistemologies function in the context of cancer research. For example, Wailoo (2011) details that scientists in the first half of the 20th-century conceptualized cancer as a disease of civilization that mostly afflicted well-to-do white women. As Wailoo notes, scientists during this time believed those from "primitive" societies were protected from cancer because of an "innate immunity" that protected them from the harsh modern environments they now inhabited. By the 1960's, however, cancer disproportionately affected black people and, in turn, scientific theories changed accordingly, suggesting that modifications in diets and exercise in their post-plantation lifestyles were the root causes of black people's elevated cancer risk (Wailoo 2011: 151-161). Wailoo also documents that cancer rates can shift as the result of racialized clinical practices, like when PCA incidence rose after the introduction of prostate-specific antigen testing, which actually exacerbated racial disparities in PCA during the 1990's (Etzioni et al. 2002; Telesca, Etzioni, and Gulati 2008).

My analysis in this chapter speaks to work at the intersection of critical race theory, science & technology studies, and public health. More specifically, my work seeks to contribute to theories of racialized ignorance and the exploitation of the health disparities discourse in agnotology (Bowleg 2017; Mills 2007; Shim 2002) as well as the medicalization and pharmaceuticalization of race literatures (Kahn 2008; Pollock 2012; Wailoo 2011). While Wailoo (2011) has set the foundation for this analysis, my work looks to further elaborate how biologies are racialized in the context of PCA research and the im-

pact that this has biomedical interventions beyond the cases of prostate-specific antigen testing and BiDil. Drawing from these perspectives, I analyze publications that assess PCA disparities between black and white men, largely seeking to answer the following research questions: (1) Which discourses do scientists use to construct racial disparities in prostate cancer?; (2) In what ways, if any, are molecular understanding of racial health disparities racialized?; and (3) How are biomedical interventions to treat prostate cancer deployed in different ways for black and white men?

Data and Methodology

In this chapter, I draw from three types of data: (1) "gold-standard" scientific papers (i.e. meta-analyses and systematic reviews) that examine racial disparities in PCA-specific outcomes and care; (2) original publications examining racial differences in PCA-specific outcomes that were extracted from the meta-analyses and systematic reviews in the first type of data; (3) clinical guidelines containing racially-specific claims about PCA treatments. To generate a sample for this study, I developed search terms in the ISI Web of Science database aimed to identify contemporary "gold-standard" papers that outline the best available evidence on racial disparities in PCA. To conduct my search, I looked for English-only articles from 1980-2017 in the ISI Core Collection using the following terms: TS=((race OR racial OR ethnic OR white OR Caucasian OR black OR African OR Asian) AND "prostate cancer" AND (meta-analysis OR systematic review OR clinical guideline)). This search initially yielded 544 results.

After I read each of the abstracts of these articles, I decided to narrow the focus of my study to only focus on racial disparities in PCA between white and black men and, in

turn, eliminated publications that (1) either did not measure racial disparities in PCA-specific outcomes between these two groups or (2) were not one of the three types of "gold-standard" papers mentioned above. More specifically, I limited my focus to outcomes that included only PCA incidence, mortality, specific PCA treatments (radiotherapy, radical prostatectomy, androgen deprivation therapies), and prostate-specific antigen screening.

Through this sampling process, I identified four meta-analyses (Bach et al. 2002; Simon Evans et al. 2008; Sridhar et al. 2010; Romero et al. 2012), two systematic reviews (Peters and Armstrong 2005; Romero et al. 2012), and 26 publications with original empirical comparisons of PCA-specific outcomes between black and white men. Looking to generate a more comprehensive sample, I used the "gold-standard" papers as "seeds" to snowball a larger sample of publications with original comparisons of PCA-specific outcomes, which involved collecting all of the articles that were included in the meta-analyses and systematic reviews. As a result of this process, my total sample of "gold-standard" and original publications totaled 120 articles that span from 1973-2017 (see Appendix III). These studies examine a diversity of racial disparities, including PCA incidence, PCA-specific mortality, receipt of prostatectomy, radiation therapy and androgen deprivation therapies (see Table 3). In the first section of my results, I discuss racial disparities in PCA incidence and PCA-specific mortality while I outline racial differences in the three treatment outcomes in the third section. Although I cannot claim that this is a comprehensive collection of *all* existing studies on this topic, it is still a more extensive sample than all of the existing "gold-standard" publications combined.

While my original search yielded only two clinical guidelines from the American Cancer Society and the National Comprehensive Cancer Society (Brooks et al. 2010; Carroll et al. 2016), I wanted to conduct a more systematic comparison of clinical guidelines that advance racialized recommendations for PCA *over time* - some of which were likely excluded because they do not use racially-specific claims in their titles, abstracts or keywords. To develop this sample, I read through my larger sample of original publications to find all mentions of leading oncology, urology, and endocrinology societies that have penned PCA-related guidelines in the United States, Europe, and the United Kingdom. I then used Google Scholar and Web of Science to acquire all PCA-related guidelines from the ten most prominent organizations: American Cancer Society, the American Society of Clinical Oncology, the National Comprehensive Cancer Society, the U.S. Preventive Task Force, the American Urological Society, European Association of Urology, the U.S. Endocrine Society, the International Society of Andrology, the International Society of Aging Men, and the British Society of Sexual Medicine (see Appendix IV). In sum, my sample included 70 total clinical guidelines that span from 1993-2018.

Table 3. Data Summary of Racial Disparities in Prostate Cancer Literature (n = 120)

Studies measuring racial disparities in:	n	Percent
PCA incidence	13	10.8%
PCA-specific mortality	69	58.0%
Prostatectomy	34	28.6%
Radiation therapy	38	32.0%
Androgen deprivation therapy	27	22.7%

My first goal in analyzing these data was to evaluate claims that black men suffer from PCA at higher rates than white men. In a sense, my objective is similar to Kahn's (2003) analysis of racial disparities in cardiovascular disease, which looks to assess how

disparities are constructed to understand how they impact the development and distribution of racialized biomedical interventions. To do this, I started by reading the meta-analyses and systematic reviews in my sample, which revealed that socioeconomic disparities tend to explain racial disparities in PCA incidence and mortality. (These findings are explained in more detail in the results section below.) Building on this finding, I conducted a content analysis of my original empirical studies to quantify how often socioeconomic status was included as a control variable across these studies in addition to the effect this variable has on PCA-specific outcomes.

Second, following the insights of science & technology studies scholars (Shim 2002; Shim and Thomson 2010), I conducted a critical discourse analysis of explanations that researchers used to conceptualize racial disparities in PCA. I opted to focus on these discourses as a way of understanding what causal factors researchers ascribe to these disparities. Using an inductive approach to coding my data, I identified four main discourses given by researchers to explain disparities (lifestyle factors, social factors, biological factors, and clinical factors – defined below). Once I identified these four themes, I reanalyzed the sample of meta-analyses, systematic reviews, and original articles to establish how frequently these discourses surfaced across the literature. I used a spreadsheet to track these themes in each article and included notable examples in analytical memos that I developed as progressing through my analyses. In my results section, the majority of my findings derive from the sample of 120 publications, including meta-analyses and systematic reviews. The 70 clinical guidelines, on the other hand, were only used to inform my claims of how various biomedical interventions are distributed to black and white

men in different ways and to help ground how this distribution changed over time (as detailed in the third section below).

I detail my results in the following three sections. In the first section, I describe how the literature constructs racial disparities in PCA between white and black men in and outside of the US context. In the second section, I briefly outline the four main explanations given to explain racial disparities in PCA. Here, I focus on how health disparities are simultaneously molecularized and racialized through the discourse of black men's "aggressive biologies." While the biological markers that are used to explain PCA disparities change from testosterone to prostate-specific to androgen receptors, researchers consistently articulate that black men have biological risk profiles that are inherently more prone to developing cancer. Finally, in the third section, I explain how the racialization of these various biomarkers carries over to clinical practices by summarizing the various ways that biomedical treatments are administered to black and white men in different ways over time. Together, this work helps to show how the broader assemblage of PCA research has become racialized and how this, in turn, shapes the distribution of resources in differing ways for black and white men.

Constructing Racial Disparities in Prostate Cancer

Throughout the literature, researchers widely claim that black men suffer from PCA at higher rates than white men. As Evans and his colleagues (2008) put it, "[i]t is relatively uncontroversial to state that Black men have a greater incidence of prostate cancer than white men" (430). Most times, similar rates in disparities are given to describe the gap between black and white men in prostate-specific mortality as well (DeSantis et al. 2016).

Perhaps the most authoritative sources for this claim are comes from a series of papers produced by the American Cancer Society, the National Cancer Institute, and the Centers for Disease Control (DeSantis et al. 2016; Siegel, Miller, and Jemal 2017). These publications, which are republished every couple of years and cited thousands of times, show that PCA plays a major role in the rise and fall of overall cancer incidence over the past 50 years in the United States, advancing a number of claims about how these trends vary between racial groups.

While cancer rates have steadily declined since 1975, PCA more than doubled between 1980 and 1993 (Siegel, Miller, and Jemal 2017). These authors explain that the growth in incidence is largely explained by the introduction of prostate-specific antigen (PSA) testing, which not only led to over-diagnosis and over-treatment of PCA for men of all racial groups (Bangma, Roemeling, and Schröder 2007; Loeb et al. 2014), but also appears to have exacerbated the disparities in PCA incidence between black and white men, which is explained in more detail below (Etzioni et al. 2002; Telesca, Etzioni, and Gulati 2008). Since the mid-1990's, PCA incidence and mortality rates have both dropped dramatically (Siegel, Miller, and Jemal 2017), though this has had almost no effect on the rates of racial disparities in PCA-specific mortality since the year 2000 (DeSantis et al. 2016). Still today, studies published by U.S. health agencies uniformly claim that PCA incidence and mortality is *two to three times* as high in African American men as it is in white men in the United States (DeSantis et al. 2016; Siegel, Miller, and Jemal 2017; U.S. Cancer Statistics Working Group 2018).

When looking at the effect of socioeconomic status (SES) on racial disparities in prostate cancer, there are differentiating effects on incidence and mortality. First, only

10.8% of my overall sample (or 13/120 publications) assess the incidence of racial disparities in PCA. Nearly all of these studies find that black men have a higher incidence of PCA, but very few actually assess how SES impacts these disparities (3/13 or 10.8%). While it is likely that racially-specific PSA testing plays a factor in these trends (as discussed below), it is hard to dispute the notion that black men do have higher incidence rates. Of course, the limited quantity of evidence makes it difficult to know how racial differences in SES might confound these outcomes.

Once SES is factored into PCA-specific mortality outcomes, the story becomes a bit more complex, which is best evidenced by debates in the various gold-standard studies on this topic (Bach et al. 2002; Simon Evans et al. 2008; Peters and Armstrong 2005; Sridhar et al. 2010). Bach et al. (2002), for example, find that black men do have a decreased risk of survival compared to whites for PCA-specific outcomes. With that said, they ultimately claim that this disparity is largely due to "[d]ifferences in stage at diagnosis, socioeconomic status, and health insurance coverage" (2106). Similarly, Peters and Armstrong's (2005) systematic review finds black men have worse 5-year survival rates than white men, focusing on the role that healthcare plays in shaping this disparity.

Evans and his colleagues' (2008) meta-analysis, on the other hand, finds that black men do have a higher rates of PCA-specific mortality after adjusting for age, clinical, and SES factors. These authors argue that this is, in part, due to clinicians engaging in more conservative treatment regimens for black patients, but that the disparity could not be fully accounted for by the various control variables. In the end, Evans and his colleagues suggest that some "underlying biological factors" (434) must play a role in perpetuating these disparities. When conducting a similar meta-analysis, however, Sridhar et

al. (2010) conclude that there are no differences in PCA-specific survival between black and white men either before or after controlling for age, clinical, and other demographic factors. This study differs from Evans et al. (2008) study in that it excluded publications that used overlapping datasets from the same time periods. Sridhar and colleagues point out Evans et al. (2008) did conduct sensitivity analyses that excluded recycled data, but omitted these from their final publication for reasons that remain unclear.

Like in my second chapter, the practices of *data recycling* and *omission* have important ramifications on the enactment of racial differences. Here, the politics of in/exclusion are not just about which participants get incorporated in biomedical research, but also about the weight that particular data have on the construction of disparities and the underlying policy interventions that are proposed in response to those differences. While Evans and his colleagues argue that inherent biological differences drive racial disparities in PCA-specific mortality, the other "gold-standard" studies fall on the side of explaining *differences* using socioeconomic status and access to healthcare without ever detailing what divisions in SES actually mean. In other words, researchers fail to clearly articulate what SES is beyond basic explanations of income (either as individual measure of yearly wage or as a proxy based on median income in census tract or zip code). In doing so, SES is treated as a routine control variable, abstracted from the structure of material risks that men are exposed to as a result of their occupation, education or (lack of) income.

When looking more specifically at papers that examine racial disparities in PCA outside of the US, I found similar tensions in how researchers attribute the causal role of biological and social factors in PCA disparities. In studies conducted in the United King-

dom, researchers seem to expect that nationalized healthcare should eliminate racial disparities in PCA (Ben-Shlomo et al. 2008; S. Evans et al. 2010; Lloyd et al. 2015). However, when these studies find that black men still suffer from PCA at 2-3 times than white men in the UK, these scholars argue that some underlying biological or genetic mechanism must ultimately drive these disparities. What is implicit in this discourse is that researchers reduce differences in socioeconomic status to "access to healthcare." As a result, researchers based in the UK rarely control for factors like education, income levels, or other common SES proxies in PCA disparities papers. The absence of these variables and researchers' insistence that "access to healthcare" is the only socioeconomic factor that contributes to health disparities shows that the "social" is largely un-theorized and radically misunderstood. Not only are differences in racialized and/or discriminatory healthcare treatment completely ignored, but the UK is also depicted as a geographical region devoid of racial segregation and other forms of racialized/classed subordination, especially when contrasted to the US context.

It is also common for epidemiological research to juxtapose PCA incidences between black and white men across other geographical regions. The most frequent comparisons between patients in the US and those in the UK, South Africa, Nigeria, Brazil, and various countries in the Caribbean (Ben-Shlomo et al. 2008; Ebughe et al. 2016; Hennis et al. 2011; Heyns et al. 2011; Romero et al. 2012). For those familiar with the history of slavery, you should notice a similitude in the countries being discussed. Indeed, in Odedina et al.'s (2009) "critical review" of the literature, they compare racial disparities in PCA across several countries connected to the Transatlantic Slave Trade, even offering a short synopsis of this tumultuous history. Yet, before the end of their article, the

authors argue that: "[t]he consistent[ly] higher incidence of prostate cancer relative to other groups, observed in populations of West African descent may be attributed to the fact that these populations share ancestral genetic factors" (Odedina et al. 2009, 7). The logic here is that because black men have a higher PCA incidence than white men, regardless of the geographic region, some underlying biological mechanism must explain why these disparities persist. With that said, researchers almost always include some vague mention of environmental or social factors, which apart from healthcare are rarely elaborated on within their introduction or discussion sections (see below).

These debates about the relationship between race, SES and PCA disparities reflect those of my broader dataset on PCA disparities research. While available studies robustly support the notion that black men have a higher incidence of PCA compared to white men, only about a quarter of those publications measure SES (3/13, 23.1%). Less than half of the data comparing black and white men on PCA-specific mortality (29/69, 42%) find support for racial disparities on this outcome, but only about a quarter of those studies control for SES (8/29, 27.6%). Thus, overall, the literature does suggest that black men have a higher incidence in PCA than white men, but that PCA-specific mortality is largely explained by SES differences between these two groups. However, the fact that so few studies of incidence even measure SES should raise some level of concern about whether these statistics do hold up when these effects are factored in.

Overall, the general trend that emerges is that PCA-specific mortality between white and black men is largely explained by differences in SES. When reducing racial disparities to a definition of "race" that is removed from the structural subordination that black men are disproportionately embedded within, racial disparities may seem "uncon-

troversial" (Simon Evans et al. 2008, 430). However, when one considers that differences in SES are not just about equal access to healthcare, but also about equal access to jobs, wealth, income, healthy foods, and diminishing exposure to environmental toxins, among many other factors, we have a more robust understanding of why disparities tend to disappear once even the most crude measures of SES are included into multivariate models. Following Bowleg (2017), the point here is that a systematic (white) ignorance of both historical and/or contemporary inequalities in social structures, and how they affect racial groups in different ways, allows racial disparities in PCA to *seem* robust and, in turn, actionable. As I show below, the molecularization of health disparities is a discourse that is frequently mobilized to advance racialized biomedical interventions.

Aggressive Biologies

First, when articulating why racial disparities in PCA exist, researchers tend to deploy four kinds of explanations: lifestyle factors, environmental or social factors, biological factors, and clinical factors (S. J. Freedland and Isaacs 2005). In doing so, scientists acknowledge the multi-factorial and complex nature of disease progression (Krieger 1994; Shim 2002). While they frequently mention both social *and* biological factors in tandem, swinging from one side of this duality to the other when they frame their policy interventions, PCA researchers also tend to racialize their molecular explanations by arguing that black men have more "aggressive" or "virulent" biologies than white men. More specifically, PCA researchers suggest that racial differences in testosterone, prostate-specific antigen and androgen receptors drive are the vectors that drive the disparities in disease. While this section briefly discusses each of the four main factors that re-

searchers employ to explain PCA disparities, my focal point is on the implications of the aggressive biologies discourse.

Diet, especially consumption of dietary fat, is the most common lifestyle factor given to explain PCA risk (Kolonel, Nomura, and Cooney 1999). Mirroring a trend that Wailoo (2011) noted, PCA researchers in the 1970's-1980's made claims that black Africans and Caribbeans who shift to Western diets after migration have a greater risk of developing PCA, supposedly because it triggers spikes in steroid metabolism (i.e. testosterone production) (Peter Hill et al. 1980; P. Hill et al. 1979). More recently, studies suggest that Black men and boys tend to consume more fatty foods (Gans et al. 2003; Neumark-Sztainer et al. 2002), which leads them to develop obesity at higher rates as well (DeSantis et al. 2016). Following this logic, some argue that the increased likelihood of developing obesity is then linked to PCA risk (e.g. Amling et al. 2004), though this relationship remains controversial (Allott, Masko, and Freedland 2013). While the policy interventions that follow from this discourse are not among the most common offered, some researchers do suggest that black men should work to combat PCA by engaging in exercise more frequently and by replacing saturated fat and red meat with fish and plant-based food (Chan, Gann, and Giovannucci 2005). These suggestions, of course, presuppose that those most burdened with this disease also have the viable means to access these healthy (and expensive) foods to change their current lifestyles.

Second, scientists almost always argue that racial disparities in PCA are due, at least in part, to SES factors that differ between black and white men in the US. In fact, 63.9% (76/120) of my sample mentions that SES factors may contribute to PCA disparities in their literature review or discussion sections. In contrast, only 31.1% (37/120) of

the sample includes some concrete measure of SES in their analyses. Following an argument introduced in my second chapter, the omission of SES-related variables in analytic models reveals important insights about the politics of health disparities literature. SES is discussed regularly, but only about a third of publications (24/120, 20.17%) offered strong claims that SES contributed to racial PCA disparities or that combating socioeconomic inequalities should be prioritized in future policy interventions.

While biological measures are not incorporated into the models that evaluate disparities in PCA incidence and/or mortality, mechanistic studies that do incorporate biological variables are pervasive throughout the literature more broadly. Authors do commonly argue that biological differences explain racial PCA disparities, despite very few studies that actually assess *both* racial differences in biology and their affect on PCA disparities in the same study. In fact, one of the most prevalent claims in the literature is that black men have "more aggressive biologies" than white men, which drive disparities in overall PCA incidence, more advanced tumor grade and stage, earlier onset, greater frequency for biochemical recurrence, and higher overall mortality compared to white men (S. J. Freedland and Isaacs 2005; Underwood et al. 2004). This discourse arose in over a third of my sample (41/120, 34.5%). Though the molecular vectors through which black men's aggressive biologies are diverse and ever-changing, the most common biomarkers included in my sample are prostate-specific antigen (J. W. Moul 2000), testosterone (R. Ross et al. 1986), and androgen receptors (Heinlein and Chang 2004) with genetic markers like chromosome 8q24 and allele variations in the CYP3A family being mentioned in some of my data as well (Bhardwaj et al. 2017; Farrell et al. 2013; Freedman et al. 2006). There are at least two points of interest embedded in this finding.

First, the notion that black men's biologies are more aggressive is interesting when comparing this finding to the feminist science studies literature on testosterone. As Jordan-Young and Karkazis (forthcoming) argue, testosterone is often infused with cultural stereotypes about men and masculinities. For example, biosocial researchers have traditionally conceptualized testosterone as a molecule that supposedly masculine behaviors, among which aggression and associated behaviors (e.g., crime or other forms of social deviance) figure prominently (Archer 2006). In subsets of biosocial research, including one of the most widely cited publications in all of the testosterone literature (Mazur and Booth 1998), scientists claim that testosterone undergirds patterns of aggressive behavior in "honor cultures," which is both implicitly and explicitly racialized throughout the literature on the topic (R. Jordan-Young and Karkazis forthcoming). Mirroring these findings, prostate cancer researchers also claim that black men's biologies, fueled by testosterone and/or androgen receptors, are also "more aggressive". In other words, the trope that black men are inherently more aggressive is not just used to describe their engagement in deviant behaviors, but is also used to describe the inherent tendencies of cancer tumors in black bodies. In both cases, racial differences in testosterone are thought to provide as an explanation for inherent tendencies that put black men are at greater risk of either engaging in aggression or crime and/or developing more potent forms of cancer.

Drawing from Evelyn Fox Keller (1995, 8), the "aggressive biologies" discourse represents a form of syndedochic error (i.e. where a part misleadingly stands in for the whole) (see also Richardson 2012). In Fox Keller's example, she notes that bodies are often divided into two kinds (male/female), which leads additional properties of bodies to become imbued with cultural (i.e. gendered) meanings (like active/passive, independ-

ent/dependent, etc.). In turn, these discourses are then used to represent the whole (e.g. all male/female bodies are stereotyped as active/passive). In a similar sense, the discourse that black men's biologies are more aggressive than white men's is predicated on the notion of distinct racial groupings (black/white) exhibit different tendencies (e.g. behavioral differences in crime and deviance that are encoded as more aggressive). These discourses are then transposed onto to the biologies of black and white men, which in turn reinforce clinicians' assumptions that black and white men have distinct biological mechanisms that justify treating these two groups differently in clinical settings (see below). While testosterone is not a necessary condition for these discourses to manifest, it is certainly remarkable that testosterone (as a hormone that is both highly gendered and highly racialized) is the biological vector that is thought to propel both aggressive behaviors and aggressive biologies across these two contexts.

My second point is that discourses about racialized biologies are not constant, but instead are continually shifting from biomarker to biomarker over time. In the first two analytic chapters of this dissertation, I discussed the ways that testosterone is linked to PCA. In short, PCA researchers predominantly claim that higher levels of testosterone contribute to carcinogenesis and that black men's higher PCA risk is likely due to their higher testosterone levels. While testosterone remained a prominent biomarker in biomedical research through the 2000's when hormone research and the testosterone replacement market were both burgeoning (Handelsman 2013), the rise of Human Genome Project ushered in a new era of biomedical science centered around genetics and genomics research (Bliss 2012, 2018). In turn, PCA researchers' focus shifted to testosterone's genetic corollary the androgen receptor, suggesting that shorter CAG and GGN

repeats are linked to an elevated risk of PCA. Here, shorter receptors are associated with higher functional potency, which would, in theory, explain group differences in testosterone production (Qin et al. 2017; J.-H. Sun and Lee 2013; Weng et al. 2017).

Racialized Interventions

These historical shifts in racialized biologies shape the ways that clinical interventions are deployed differently for white and black men. In PCA research, scientists link biomarkers together into a complex chain that has broader repercussions on biomedical practice. For example, Moul (2000: 250) suggests that racial differences in testosterone and androgen receptor activity may help to explain why some studies find racial differences in prostate-specific antigen. Indeed, PCA scientists widely advance the theory that black men have higher levels of prostate-specific antigen compared to white men.[2] Furthermore, some experts suggest that black men should have *higher* age-specific PSA cutoff thresholds to account for their higher prostate-specific antigen levels (Morgan et al. 1996). Conversely, Moul (2000) recommends that clinicians engage in "targeted screening" of black patients by *lowering* prostate-specific antigen cutoff thresholds to a value that account for their more aggressive biologies. This contradiction is interesting in that it shows how racial differences can be interpreted differently by leading "experts" in the field while simultaneously being used to mobilize differentiated uses of clinical interven-

[2] While addressing the question of racial differences in prostate-specific antigen would require an additional research project akin to my first chapter, my brief engagement with the prostate-specific antigen literature shows that evidence is quite mixed on these outcomes (see Moul 2000). My argument is that, much like the case of racial differences in testosterone, false positive results may drive the racialization in prostate-specific antigen while the majority of analyses would likely proffer null or mixed results. This, to my knowledge, is still an empirical question that remains to be addressed with a meta-analysis or systematic review.

tions (Chun 2013; M'charek 2013). Unfortunately, it is also indicative of how the ideology of racial difference carries over to affect clinical practices in concerning ways.

Contextualizing these claims against my clinical guidelines data, it is first imperative to point out that leading experts associated with oncology and urology societies in the US and Europe now recommend *against* the use of prostate-specific antigen testing to diagnose PCA (Bibbins-Domingo, Grossman, and Curry 2017; Heidenreich et al. 2014a). These guidelines describe that prostate-specific antigen testing contributed to a dramatic increase in over-diagnosis and over-treatment of PCA symptoms via widespread false positive results; in short, the risks that this test offers considerably outweigh the benefits it provides (Bangma, Roemeling, and Schröder 2007; Loeb et al. 2014). While African American men are less likely to receive PCA treatments (as I explain more below), black men are *more* likely than white men to have receive PSA tests before the age of 50 (Fowke et al. 2005; L. E. Ross, Berkowitz, and Ekwueme 2008), which seems to have played some role in the over-diagnosis and over-treatment of PCA in this group over the past 20 years (Etzioni et al. 2002; Telesca, Etzioni, and Gulati 2008; Verges et al. 2017).

For example, when the U.S Preventive Task Force made updates to their guidelines to adjust for trends in overtreatment in 2012, they offered no mention of race (Moyer 2012), which seems to have briefly eliminated racial differences in PSA testing (Barocas et al. 2015; Jemal et al. 2015). But now some PCA researchers are calling for racially-specific guidelines to once again promote early PSA testing in African American men, especially during mid-life (Shenoy et al. 2016). Following these calls to "selectively apply" PSA testing to "high risk" groups (Sandhu and Andriole 2012, 148), most of the major health agencies based in the US, like the American Cancer Society and American

Urological Society, now include language in their guidelines that advise targeted prostate-specific antigen testing for black men (Carter et al. 2013; Wolf et al. 2010). In other words, black men in the US are more likely to be diagnosed with PCA, in part because they are administered prostate-specific antigen tests more regularly on the basis of racially-specific guidelines. In contrast to the US guidelines, all but one of the guidelines issued by the European Association of Urology offer racially-specific recommendations and generally advise against prostate-specific antigen testing for general diagnoses (Cornford et al. 2017; Heidenreich et al. 2014a, 2014b; although see Mottet et al. 2017) (see Appendix IV). The various differences I just mentioned provide some evidence that the racialization of clinical interventions are agency-specific, suggesting that this process can be undone by intervening upon those associated with these organizations.

That said, a prominent discourse that runs through my data is the notion that clinicians should engage in more screening and "active surveillance" of black men through the use of early prostate-specific antigen testing. Technically speaking, active surveillance refers to a protocol where clinicians attempt to maintain quality of life and avoid complications associated with over-treatment and to monitor PCA progression while still intervening when necessary (Leinwand et al. 2018). The move towards active surveillance was implemented in response to the prostate-specific antigen over-treatment controversy, but this paradigm shift has not played out to impact black and white men in the same way.

According to recent studies, African American men are still less likely to receive active surveillance, even after controlling for SES (Abern et al. 2013; Silberstein et al. 2014). Although it is still not entirely clear why this is the case, Silberstein and col-

leagues (2014) suggest that black men tend to have more advanced tumors upon being diagnosed, which dissuades clinicians from engaging in active surveillance or other treatment regimens since it is often considered to be "too late" to have an impact on tumor progression. While the general notion that black men's aggressive biologies need to be increasingly surveilled is somewhat jolting, the fact is that these calls have not translated to quality care. Black men still receive poorer PCA care than white men no matter how you measure it.

For example, my data suggest that black men are less likely to receive the same access that white men do to surgical interventions like radical prostatectomies (Desch et al. 1996; Jones et al. 1995; Robbins, Yin, and Parikh-Patel 2007). While only a subset of the overall literature examined these outcomes, most studies find that black men have a lower likelihood of receiving prostatectomies (29/32, 90.6%). While the disparity is not as marked, black men are also less likely to receive radiation therapy compared to white men (a pattern found in 15/36 or 41.6% studies with 12/36 showing no differences between the two groups). Some even suggest that black men's aggressive biologies play a role in medical professionals' treatment decisions. As Underwood et al. (2004) puts it, "[m]ore aggressive tumor biology among African American men could be presumed to have greater propensity for extraprostatic spread and may result in lower surgical efficacy than prostate cancer among White men" (20). As this claim suggests, the notion that black men's inherently more aggressive tumors may play into clinician's decisions to *not* treat black men since it is unlikely to thwart tumor progression anyway.

Racial disparities in care can be found when looking at access to pharmaceuticals. For one, studies looking at racial differences in receipt of androgen deprivation ther-

apies (ADTs) suggest that white men are more likely to be given these drugs than their black counterparts with two caveats (Moses et al. 2010; C. Nguyen et al. 2019; Schapira, McAuliffe, and Nattinger 1995). First, Shahinian et al. (2010) suggests that while black men are less likely to receive ADTs for *appropriate* PCA treatment regimens, their analyses show that black men and men residing in low income areas are more likely to be prescribed ADTs for *inappropriate* uses (i.e. when the drug was unlikely to provide much therapeutic benefit). Second, studies that use data from the late-1980's to mid-1990's, when ADTs were just beginning to be widely used on the market, show that black men were about the same or, in some cases, more likely to receive ADTs during that time (Harlan et al. 2001; Potosky et al. 2002). This means that black men were much more likely to receive ADTs during this early period of relative uncertainty, which means that either clinicians were either particularly enthusiastic about what ADTs could do for black patient's PCA or that clinicians were more willing to test these experimental drugs on black patients before distributing them to white patients. Together, these trends suggest that while black men are less likely to receive quality care for PCA, they tend to receive poorer care when they do utilize health services.

Extant evidence also suggests that the discourse of racial disparities in PCA shape the guidelines and distribution of testosterone replacement therapies (TRTs). The most obvious place this manifests is in the Endocrine Society's clinical guidelines on TRTs (Bhasin et al. 2010, 2018), which explicitly advises clinicians not to prescribe African American men testosterone because of their higher risk of PCA. This recommendation does seem to have an effect on distribution practices, as black men are less likely to receive TRTs in equal access healthcare settings (Jasuja et al. 2017). As I argue in Chapter

4, this argument is predicated on the assumptions that (1) black men have higher levels of endogenous testosterone than white men and (2) that these higher levels of testosterone contribute to black men's PCA risk. My second chapter shows that, despite widespread claims that racial differences do exist in testosterone, the existing evidence does not support this. Furthermore, my fourth chapter, as well as a large meta-analysis on this subject (Endogenous Hormones and Prostate Cancer Collaborative Group et al. 2008), outlines that higher levels of testosterone do not reliably predict PCA risk. In other words, the Endocrine Society's recommendation that African American men should not be prescribed TRTs is predicated on two historical myths, suggesting that these guidelines mislead experts to withhold TRTs from black consumers.

While the various disparities outlined above rarely employ the discourse of precision medicine, recent work has now begun to call more explicitly for the development of racialized pharmaceuticals and diagnostic tools predicated on this paradigm (Burke, Trinidad, and Press 2014; S. Freedland 2018; Weitzel et al. 2011). The dream of precision medicine is to develop individualized therapies based on one's unique genetic risk profile. In practice, these products are more regularly *group-differentiated* treatments. Recently, Freedland (2018) proposes that a personalized cancer drug could be developed on the results of a large genetic study of Chinese men. Similar genome-wide association studies have already attempted to establish genetic risk factors that vary between white and black men, including androgen receptors, chromosome 8q24 loci, among a host of other biomarkers (Bhardwaj et al. 2017; Farrell et al. 2013; Freedman et al. 2006; Powell and Bollig-Fischer 2013; Singh, Lillard Jr, and Singh 2017). Given that oncology is one of the fields that have been most active in advocating for the development of personalized

treatments (Collins and Varmus 2015), these calls should prompt scholars critical of BiDil to prepare for a racialized pharmaceutical to emerge within this context of PCA in the coming years.

In sum, this section outlined the various ways that biomedical interventions have been racialized in PCA research. While scientists do discuss how racial disparities in PCA may be affected by differences in SES, racialized biologies are still frequently leveraged to advance clinical interventions in different ways for black and white PCA patients. While scholars often propose racially-specific treatments and clinical guidelines to alleviate racial disparities in PCA, African American men are still less likely to receive ADTs, TRTs, surgeries like radical prostatectomies or radical radiotherapies, or be actively surveilled to minimize their risk of biochemical recurrence. Furthermore, biomedical researchers continue to work toward the dream of precision medicine; the logic here is that racialized pharmaceuticals will ameliorate racial disparities in PCA. Although prostate-specific antigen, testosterone, and androgen receptors have traditionally been the biomarkers through which disparities are molecularized, the emerging sociogenomic era has ushered in a multitude of new biomarkers for scientists to use. It is through these emerging biologies that we should expect to see PCA re-racialized in the coming years.

Conclusion

Using insights from critical race theory and science & technology studies, this chapter examines how racial disparities in PCA are constructed and mobilized in biomedical literature. While researchers widely endorse the discourse that black men's PCA mortality rates are about two to three times higher than that of white men in the United States, me-

ta-analyses show disparities in mortality are explained by socioeconomic differences between these two groups (Simon Evans et al. 2008; Sridhar et al. 2010). Despite this being the case, PCA researchers tend to exploit the notion that racial disparities in PCA do exist by emphasizing molecular explanations at the expense of formally analyzing the effects of SES on PCA-specific outcomes. Although PCA researchers usually give lip service to the importance of SES in their introduction and discussion sections, they typically reduce socioeconomic disparities to concerns about access to healthcare and rarely include measures of class inequality into their analytic models. In doing so, scientists reveal their own "molecular imperative" (Weatherford-Darling et al. 2016) to advance biologically-oriented research, clinical interventions and personalized pharmaceuticals without explicitly analyzing how socioeconomic factors may contribute to racial disparities in PCA.

Following from this analysis, I argue that PCA researchers use race as a technology to mobilize biomedical interventions in disparate ways for black and white men (Chun 2013). PCA researchers very rarely take strong ontological stances about what race "is" in their work. At times, race is used in a routine fashion, allowing it to become a risk factor in-and-of-itself (Shim 2002). Other times, PCA researchers suggest that race is a "social construct" (DeSantis et al. 2016). These variations suggest that race is a multiplicity that can be enacted and conceptualized in a variety of ways across publications (M'Charek 2013), but to become preoccupied with whether race is biological or social is to miss the point (Chun 2013). My interest is more about how the concept of race, racial difference, and the discourse of racial health disparities are enacted to shape the distribution of resources in PCA treatments.

Akin to Bowleg (2017), my argument is that PCA researchers employ the discourse of health disparities in ways that function to bolster white privilege (see also Mills 2007; Sullivan and Tuana 2007). The first way that this happens is by researchers ignoring studies that show SES differences explain racial disparities in PCA between black and white men (Evans et al. 2008; Sridhar et al. 2009). When SES is mentioned, scientists often reduce their discussions of this variable to debates about access to healthcare. This is most evident in the way that British scientists conceptualized their findings, suggesting that racial disparities in PCA must be driven by some biomarker that underlies racial difference since black men in the UK have the same access to healthcare than white men do in the national healthcare system. With this said, many US-based studies that drew from equal access healthcare samples (like Veteran Affairs data) mirrored these discourses closely (see Graham-Steed et al. 2013). Despite most studies mentioning SES in some capacity, studies rarely made strong claims that SES was a major contributing factor in driving health disparities. Instead, it was more likely that researchers employed the discourses of racial difference or racial health disparities to call for more research or (racialized) biomedical interventions.

> "What needs to be done," as Charles Mills suggests, "is to extrapolate some of this literature to a social context - one informed by the realities of race. Because of its marginalization of social oppression, the existing [epidemiological] literature tends to ignore or downplay such factors. In contrast … (class) domination and exploitation were the foundation of the social order, and as such they produced not merely material differentials of wealth in the economic sphere but deleterious cognitive consequences in the ideational sphere" (Mills 2007: 34).

As Mills (2007) suggests, these racialized discourses reflect an ideational sphere of (white) ignorance that circulates through the PCA research. Researchers that ignore SES or treat differences in SES as a routinized control variable fail to fully conceptualize

or operationalize the realities of how black and white men's lives tend to differ in the United States and abroad. Mindy Fullilove's model of serial forced displacement shows this very clearly (Fullilove and Wallace 2011). Racial disparities are not driven solely by equal access to healthcare, though addressing this would be an important start, but instead to equal access in jobs, wealth, income, healthy foods, and diminishing exposure to environmental toxins, among many other factors (Fullilove and Wallace 2011; Williams, Neighbors, and Jackson 2003; Williams and Mohammed 2009). Thus, the absence of these factors in my data speaks volumes. Even in work that makes explicit note of this history by mentioning the Transatlantic Slave Trade (Odedina et al. 2009), there is no real conversation about the mechanisms that reproduce racial inequalities today. When these authors conclude by suggesting biological factors contribute to PCA disparities, it suggests that racial subordination can be ameliorated with more research and biomedical advances rather than addressing the underlying structure of racial/classed inequalities.

The endorsement of these liberal tendencies reflects the politics of contemporary biomedical and epidemiological research more broadly. While research do discuss socioeconomic factors that shape the poorer health of black men in the United States, little is done in terms of actually proposing changes to the availability of resources to those communities. Instead, the interventions that emerge are predicated on individualized and/or group-differentiated interventions that often exacerbate existing problems or create new controversies like the dilemma with prostate-specific antigen testing discussed above. This is one of the inherent problems with the "health disparities" motif and why the pursuit of "health equities" is an increasing emphasis of many social scientists (Bowleg et al. 2017; Wailoo 2017). What "equity" in PCA treatment comes to mean will

inevitably require researchers to take a more active role in incorporating the enduring socioeconomic factors that drive the development of PCA in their work (Klein and von dem Knesebeck 2015). More to the point of my main argument, it will require researchers to re-evaluate their own role in perpetuating the racialization of PCA and the broader structure of white ignorance in epidemiological research.

Lastly, my work shows the ways that biologies are racialized through biomedical discourse. The notion that black men have more aggressive biologies is not only concerning in that it reflects racial stereotypes about how black men engage in forms of deviant behaviors in contemporary society, but also in that it displaces black men's risk of developing PCA, as well as their responsibility for treating it, back onto a biological vector that they are supposed to control through self-care and technological interventions. This aligns with Nikolas Rose's (2007) point that the molecularization of health disparities reveals an 'ethopolitical' regime that shifts the management of risk from the population level back onto individuals. However, by advancing discourses about racialized biologies, this provides a rationale to explain why PCA researchers still have yet to minimize racial PCA disparities despite their supposed best efforts. Rather than addressing the structural inequalities that social scientists have long argued are the fundamental causes behind health disparities (Link and Phelan 1995), researchers have instead have chosen to advance personalized pharmaceuticals that re-inscribe race as biological essence (S. Freedland 2018). In doing so, they do little to undermine the core social and structural mechanisms that propel PCA in *both* white and black men.

Chapter 4: Tracing Change in Scientific Consensus: Testosterone, Race, and Controversy in Prostate Cancer Research

In the era of evidence-based medicine, scientific "gold-standards" like meta-analyses, systematic reviews, and clinical guidelines are often regarded as the best available evidence for treating medical conditions. Typically, these documents are the outcome of scientific experts collaborating to establish a statement of scientific consensus that guides clinicians or experts in other adjacent fields (Knaapen et al. 2010; Moreira 2005). Yet, the reality is that these gold-standard studies do not always reflect the best available evidence. For example, the Endocrine Society's clinical guidelines on testosterone replacement therapies (TRTs) advise clinicians *not* to prescribe TRTs to those diagnosed with prostate cancer (PCA) or to African American men because of their increased risk of this disease (Bhasin et al. 2010, 2018). This recommendation is predicated on the notion that (1) racial disparities in PCA exist between white and black men, (2) PCA disparities are driven by racial differences in testosterone, and (3) that testosterone directly contributes to a higher risk of PCA. While scholars are currently deliberating whether it may be safe to prescribe TRTs to PCA patients (Morgentaler 2013), there are no existing debates, to my knowledge, about revising the racially-specific recommendations advanced in the Endocrine Society's current guidelines.

In the second chapter of this dissertation, I demonstrated that the racial testosterone theory (i.e. the thesis that testosterone differs between racial groups) is unsubstantiated by existing scientific evidence and therefore undermines the notion that racial differences in testosterone can contribute to racial disparities in PCA. Furthermore, my third chapter examines that several gold-standard studies that show that racial disparities in PCA-specific mortality are best explained by group differences in socioeconomic status,

rather than differences in testosterone. In this chapter, I interrogate the link between testosterone and PCA, analyzing how researchers' theories of this relationship have changed over time and the impact that these theories may have on the Endocrine Society's clinical guidelines.

To do this, I explore the association between testosterone and PCA using social network analysis and science & technology studies. Employing Shwed and Bearman's (2010) quantitative method to graph patterns of scientific consensus in citation network data, I show that the PCA literature goes through three cyclical waves of consensus and contestation from 1980-2017. By mapping these trends, I show that the association between testosterone and PCA has undergone a radical paradigm shift over the past 25 years. While *high* levels of testosterone were once considered an uncontroversial marker of PCA risk for more than seven decades, emerging evidence now suggests that clinically *low* levels of testosterone may be a more robust indicator of PCA risk.

The implications of this paradigm shift stand in sharp contrast with recommendations advanced by the Endocrine Society's guidelines; if high levels of testosterone do not contribute to (black men's) PCA risk, there is no scientific reason to withhold TRTs from African American men. Importantly, my analysis provides a structural measure of scientific consensus in PCA research, which complements several gold-standard studies on the relationship between testosterone and PCA (e.g. Endogenous Hormones and Prostate Cancer Collaborative Group et al. 2008), that suggest the Endocrine Society's guidelines are in urgent need of revision to ameliorate the effects that these recommendations have on withholding TRTs from African American men in clinics around the United States (see Jasuja et al. 2017).

Before embarking upon these analyses, I explain how this chapter fits into the broader field of science & technology studies. To do this, I start by summarizing past work on pharmaceuticalization and the history of hormone-based therapeutics. Next, I talk about how science studies scholars have started to draw from insights in social network analysis to measure patterns of scientific consensus. After contextualizing my work within these two literatures, I analyze how the relationship between testosterone and prostate cancer has changed since 1980. More specifically, I document how three waves of consensus and contestation have emerged around the development of various hormone-based pharmaceuticals over time. While these results speak more broadly to how patterns of scientific consensus in biomedical research can be driven by the introduction of new pharmaceuticals, the main contribution of this chapter is showing how the paradigm shift from high to low testosterone as a marker of PCA risk undermines the Endocrine Society's clinical guidelines. In the conclusion, I argue that the racially-specific recommendations endorsed by the world's leading endocrinology society must be reassessed to account for the best available scientific evidence.

Pharmaceuticalization and the History of Hormonal Therapies

In recent years, social scientists have developed increasing interest in pharmaceuticalization or the "process by which social, behavioural, or bodily conditions are treated or deemed to be in need of treatment, with medical drugs by doctors or patients" (Abraham 2009, 100). One of the most famous examples of pharmaceuticalization has been the growth of TRT market. Building on the wild success of other "lifestyle drugs" like Viagra (Fox and Ward 2008), scholars argue that marketing efforts helped spark the medicali-

zation of male aging through the discourse of "low T" and andropause (Conrad 2007; Marshall 2007, 2009b, 2009a; Watkins 2007, 2008, 2012). In turn, this industry has ballooned from roughly $300 million to nearly a $2 billion global market since the early-2000s (Handelsman 2013).

The proliferation of the TRT industry is interesting because its expansion is not predicated on the discovery of a novel therapeutic entity, but instead on the repurposing of a molecule that was synthesized nearly a century ago (Oudshoorn 1994). Unlike hormone replacement therapies and hormonal contraceptives, which were widely consumed by women from the 1930s to 1990's (Krieger et al. 2005; Krieger, Chen, and Waterman 2010), the TRT market swelled only briefly during the 1930s before sales fizzled out in the 1950s (Conrad 2007; Marshall 2009a; Oudshoorn 1994; Watkins 2007, 2008). The reasons for the TRT industry languishing, however, have not been well-articulated in the sociological or historical literature, though various technical, industrial, and political factors are likely responsible for floundering sales before its re-emergence the early-2000s (Gaudillière 2004, 2005; Handelsman 2013; Laveaga 2005; Oudshoorn 1994).

One explanation that is almost entirely unexplored in the socio-historical literature, but quite obvious after consulting biomedical studies, is that TRTs have long been thought to put men at an increased risk of developing PCA. Since Huggins and Hodges' (1941) Nobel-prize winning research, which found that chemical and surgical castration reduces PCA from proliferating and testosterone administration increases the size of prostate carcinomas, clinicians have recommended against the use of TRTs in PCA patients (Bhasin et al. 2010, 2018). Interestingly, Oudshoorn (1994) mentions that the Dutch pharmaceutical Organon originally tested TRTs for the treatment of PCA, but does

not mention whether Huggins and Hodges' landmark findings contributed to the evaporation of this market. Of course, the theory that higher levels of testosterone contribute to PCA is not only influential in how it thwarted the TRT industry from growing until the early-2000s, but also in how it contributed to the research and development of androgen deprivation therapies (ADTs).

Since the 1940's, ADTs have been one of the most widely used technologies for treating PCA. While specific treatments have changed over time, its various instantiations (including estrogens, anti-androgens, luteinizing hormone-releasing hormone agonists, gonadotropin-releasing hormone antagonists, and combined androgen blockade) have all been utilized to alleviate PCA symptoms for men across the world (Denmeade and Isaacs 2002; Crawford 2004). Very little of the sociological literature has focused on the use and dissemination of ADTs apart from the masculinities literature, which centers mostly on how men's gendered ideals, identities and healthcare practices change after the onset of ADT use (Chapple and Ziebland 2002; Maliski et al. 2008; L. Navon and Morag 2003; Oliffe 2005, 2009). Although these drugs have come under considerable scrutiny over the past decade for their deleterious side-effects (Isbarn et al. 2009; P. L. Nguyen et al. 2015), the ADT market is still projected at upward of $10.1 billion as of 2017 (Zion Market Research 2018).

While seemingly unconnected, the growth of the ADT and TRT industries represent two intersecting cases of pharmaceuticalization that converge through the hormone testosterone. As Jordan-Young and Karkazis (forthcoming) argue, testosterone is a "multiplici-T" that is enacted differently across various contexts. As a result, testosterone becomes entangled in biomedical controversies in complex ways depending on how scien-

tific communities use the hormone it in their research programs. In this chapter, I examine how research conducted on ADTs and TRTs converged over time, which ultimately comes to shape the structure of scientific consensus and, in turn, alters how researchers conceptualize the risks associated with consuming these pharmaceuticals for different racial groups. In order to contextualize these findings, I briefly turn to a literature that uses social network analysis to examine patterns of scientific consensus.

Social Network Analysis and Scientific Consensus

Science studies scholars have incorporated elements of network analysis for nearly 50 years. Since Price (1965) demonstrated the importance of network measures in identifying influential work, social scientists have deployed network metrics to guide coauthor and citation analysis (Garfield 1972; Moody 2004). The meaning of citations, more specifically, have been heavily debated among science studies scholars with some suggesting that citing is an act of debt payment to acknowledge the contributions of their colleagues (Merton 1957) while others follow a more constructivist interpretation, arguing that citing is a rhetorical strategy to enact claims within a given research community (Latour 1987). Alternatively, Leydesdorff (1998) suggests that citation networks are comprised of two layers: authors employ citations in their work in addition to the actual citation's text structuring how knowledge spreads throughout networks of scientific communication. Because both authors and citations have their own autonomy, the function of citations can become somewhat ambiguous and uncertain, suggesting that a definitive theory of citations is difficult to ascertain because they can function in distinct ways depending on the specific research domain.

In recent years, science & technology studies scholars have used network analysis to examine the history of several biomedical contexts (Cambrosio, Keating, and Mogoutov 2004; Cambrosio et al. 2006, 2014). In combination with insights gained from their ethnographic work, Cambrosio et al. (2004) mapped the emergence of a collaborative network that mobilized around a socio-technical platform that used antibodies in oncology research. Likewise, Cambrosio et al. (2006) use citation analysis to graph historical shifts in translational cancer research, showing that basic bench science and clinical research have slowly coalesced over the past 40 years. Actor-network theorists, like Venturini (2010b, 2010a), also employ network analysis to map the complex relationships between human and non-human actors in several ongoing political controversies (Bounegru et al. 2017; Latour et al. 2012). Together, these publications show the various capacities that network analysis offers to the field of STS.

Scholars have also implemented network analysis to trace patterns of scientific consensus in citation data. Most notably, Shwed and Bearman (2010) have developed a quantitative method to examine patterns of scientific consensus. Using an algorithm to detect structural patterns in communities across longitudinal citation networks, these authors demonstrate that science studies scholars can map trends in consensus formation without prior expertise in those research fields. Shwed and Bearman (2010) show the utility of this approach across eight different examples in their work while others have documented the success of this method in other biomedical and political controversies as well (Adams and Light 2014, 2015; D. Navon and Shwed 2012; Shwed 2015). In this chapter, I use this quantitative methodology alongside qualitative strategies to examine the structure of scientific consensus in the context of PCA research. This allows me to

examine how researchers conceptualize the link between testosterone and prostate cancer in order to contextualize the structure of scientific consensus against the Endocrine Society's clinical guidelines on testosterone replacement therapies.

Beyond the broader theoretical questions around pharmaceuticalization and scientific consensus that this chapter addresses, I also examine how scientists conceptualize the relationship between race, testosterone and PCA. Biomedical experts and regulatory agencies generally argue that black men suffer from PCA at rates two to three times higher than white men in the US (Simon Evans et al. 2008; Siegel, Miller, and Jemal 2017) and, as my first two analytic chapters outline, testosterone has played an important role how researchers attempt to explain those disparities (R. Ross et al. 1986). In this chapter, I will also explain how racial difference testing (i.e. group comparisons in testosterone levels like those examined in Chapter 2) fits into the broader patterns of scientific consensus in the PCA literature. By situating this research program within a specific socio-historical context, I hope to better understand how racial difference testing intersects with changes in scientific consensus. Speaking to the broader questions of this dissertation, this analysis will help illuminate how racialization intersects the process of pharmaceuticalization and knowledge production in the context of PCA research.

While past literature on the pharmaceuticalization of race has focused primarily on drugs developed for specific racial groups, with BiDil being the infamous example (Kahn 2013; Pollock 2012), this chapter focuses on how the racialization of clinical guidelines shapes the distribution of hormone-based pharmaceuticals. As I detailed above, the Endocrine Society's current clinical guidelines explicitly advise against prescribing African American men TRTs (Bhasin et al. 2010, 2018), which past studies have

shown negatively affects the likelihood that black men will be prescribed TRTs in comparison with white men (Jasuja et al. 2017). Because of the growing importance of clinical guidelines in the era of evidence-based medicine (Moreira 2005; Knaapen et al. 2010), I am interested in how the association between testosterone and PCA may impact black and white men in different ways through the racialization of these evidentiary gold-standard studies. Thus, in the process of mapping changes in scientific consensus, I also trace the ways that race and ethnicity are deployed by scientists in PCA research.

Data and Methodology

The data used in this chapter derive from published articles in the ISI Web of Science database. To generate this sample, I used Web of Science to search for English language articles that include testosterone and PCA-related terms in their titles, abstracts, and keywords from 1980-2017. These search terms were: TS=(testosterone AND "prostat*" AND (cancer OR "carcinog*" OR "hyperplas*" OR "hypertroph*" OR "tumor*" OR "neoplasm*" OR "carcinom" OR "metasta*")). This search yielded a total of 8,002 total articles. After retrieving the full bibliographic information (including complete cited references lists) and abstract for each text, I converted this data into a citation network using the Sci2 software package (Sci2 Team 2009). Citation networks are composed of scientific articles that connect to other papers. In these networks, papers are represented as *nodes* while acts of citing between papers are represented as *ties*. Citations networks are directed, meaning that each tie has an arrow that indicates that one paper is cited by another. In these networks, the arrow is directed from the cited reference to the citing reference in order to demonstrate the flow of influence.

Once this network was constructed, I used Shwed and Bearman's (2010) 'temporal moving-window' strategy to create a series of citation networks which serve as the basis to examine historical patterns in the community structure of the overall network from 1980-2017. This process involved three steps. First, the overall citation network was partitioned into individual years. Second, the median citation year was established for each year from 1980-2017. Third, any articles that predated the median citation year were then removed from the year-by-year networks. What remains are 37 year-by-year networks composed of articles published within the moving-window in addition to their cited references (see Appendix V for a summary of the dynamic window properties). The purpose of this methodology is to establish a strategy for analyzing the structure of citation networks without the networks being overly-determined by papers published at very early points in the literature's history. The strategy has been successfully implemented by a number of influential papers at the intersection of network analysis and science studies (Adams and Light 2014, 2015; D. Navon and Shwed 2012; Shwed 2015; Shwed and Bearman 2010).

Next, I used the igraph package in R open-source statistical package to analyze the modularity of each year-by-year network (Csardi and Nepusz 2006). Modularity is a measure that analyzes the community structure of an overall network (Fortunato 2010). Within a citation network, communities cluster together based on how often a collection of papers cite each other. As Shwed and Bearman (2010, 2012) argue, the more often a community clusters together based on citation patterns, the more consensus forms in the overall network. In cases of high consensus, a network will have a higher modularity score to reflect fewer community subdivisions. In contrast, networks with a lower modu-

larity score are more likely to break into distinct citation communities, reflecting more scientific dissensus.

Following this logic, I used the leading eigenvector method to calculate modularity for each year-by-year network (Newman 2006), though my results did not differ appreciably when using either the standard modularity or edge-betweenness techniques in igraph (Clauset, Newman, and Moore 2004; Newman and Girvan 2004). Because modularity scores can be shaped by the size of the overall network, I followed past literature by scaling the modularity score to the overall size of the network (Adams and Light 2014, 2015; D. Navon and Shwed 2012; Shwed and Bearman 2010), which helps to control for the increase in overall scientific production from 1980-2017 (see Appendix V) (see also Kramer 2018). What emerges from this analysis is a quantitative metric to establish specific temporal junctures where large-scale structural changes occurred in PCA research.

While Shwed and Bearman's (2010) goal was to create a parameter for measuring scientific consensus without expertise within a given field, my interest in this chapter is to further explore the research topics that shape these structural changes and how those changes impact our understanding of racial health disparities in PCA. Thus, in the final step of my analysis, I employed strategies commonly used in science & technology studies to read influential publications across each historical period in order to first generate explanations about which factors drove patterns of consensus and contestation over time. Furthermore, I examined how PCA researchers conceptualize racial disparities in PCA in their work and how this feeds into patterns of consensus in the field.

Using the open-source software Gephi, I mapped each year-by-year network using the Louvain methodology to partition citation communities (Bastian, Heymann, and

Jacomy 2009). Next, I conducted descriptive network measures of the in-degree centrality for each network (i.e. how many times each paper was cited by other articles in the network). Using this statistic as a basis to guide strategic reading of the most highly cited articles in each community and in the overall network, I read highly cited articles across the largest communities at critical points in the network's evolution. Using this technique, I was able to establish how changes in scientists' understandings of testosterone in prostate cancer research affected the construction of racial disparities in health outcomes.

Results

Figure 3 plots the scaled modularity of each year-by-year network cut from 1980-2017, representing patterns of consensus and contestation among researchers that measure or administer testosterone in PCA research. Overall, the networks' scaled modularity score declines over time, which indicates a clear trend toward scientific consensus. This pattern mirrors past applications of this temporal moving-window method (Adams and Light 2014, 2015; D. Navon and Shwed 2012; Shwed 2015; Shwed and Bearman 2010) in addition to making intuitive sense that researchers in a given field tend to agree more over time. Beyond the general trend towards consensus, the graph also exhibits ebbs and flows within the networks' scaled modularity. Figure 3 shows three clear spikes in contestation (around 1990, 1996, and 2006). After conducting qualitative analysis of the literature though my strategic reading method, I opted to describe the formation of scientific consensus in this domain through three distinct historical periods that center around the growth of ADT research (from 1980-1995), the growth of TRT research (from 1996-2008), and the recent convergence of the ADT and TRT literatures (from 2009-2017).

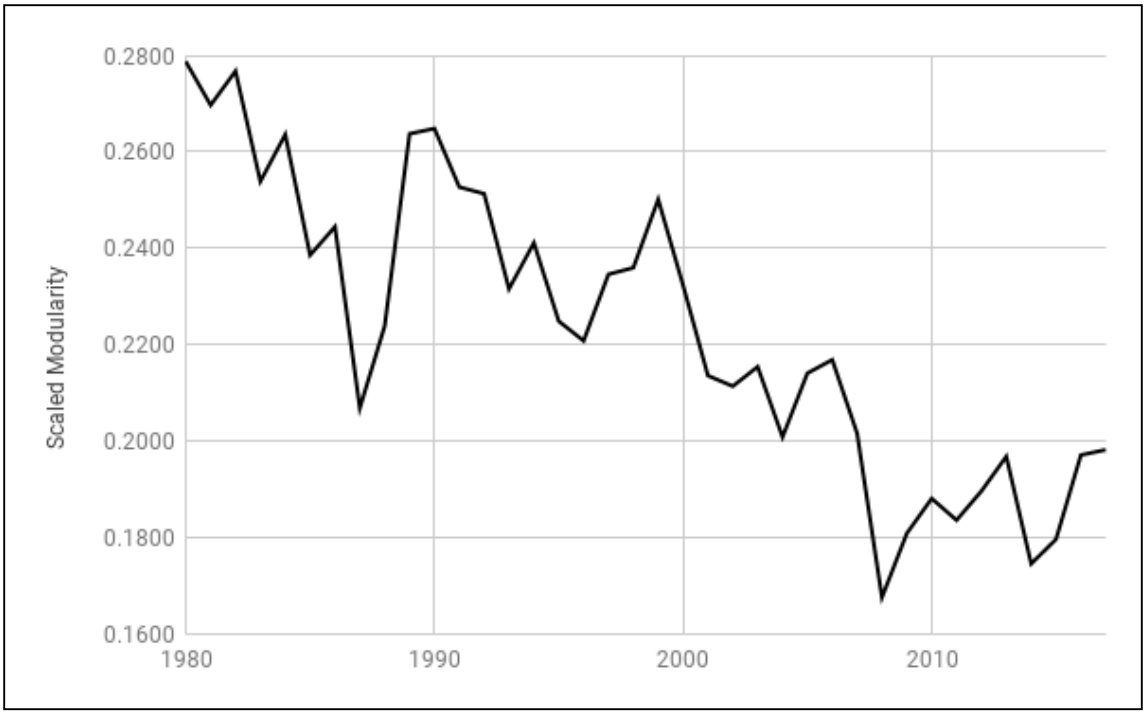


Figure 3. Scaled modularity analysis of prostate cancer and testosterone. The dark line corresponds to the modularity scores scaled for logged network size. Higher levels of this measure align with periods of higher contestation while lower levels of scaled modularity correspond to higher levels of consensus.

Consensus and Contestation in Early ADT Research (1980-1995)

For the majority of the 20th-century, the relationship between testosterone and PCA have been relatively uncontested. High levels of testosterone have been associated with the development of PCA since the early 1940's when Nobel laureate Charles Huggins and his colleagues (1941; 1940) found that surgical castration led to the regression of PCA and testosterone administration contributed to the progression of prostate carcinomas. Ever since, medical students have been taught that that adding testosterone to the prostate is like adding "fuel to a fire" or like providing "food for a hungry tumor" (Morgentaler 2006). Huggins' groundbreaking studies revolutionized the management of PCA at the

time, establishing surgical or pharmaceutical castration (via the use of estrogens and anti-androgens) as the most widely used treatment to manage PCA through the late 1970's (Denmeade and Isaacs 2002). However, by the early-1980's, the search for new forms of therapeutic interventions were building momentum in PCA research, shaping the citation networks' patterns of consensus and contestation along the way.

As Figure 3 demonstrates, PCA research in the early-1980s is marked by a low degree of consensus. At the time, prominent communities in the network were testing how experimental treatments affected carcinogenesis in animals like rats and dogs. Human research, on the other hand, focused more on the association between testosterone and benign prostate hypertrophy in addition to how androgens were linked to various molecular mechanisms. While research on androgen ablation therapies was largely peripheral in the early-1980s, the network would eventually mobilize around the development of (1) luteinizing hormone-releasing hormone (LHRH) agonists, (2) gonadotropin-releasing hormone (GnRH) agonists, and (3) anti-androgens as treatments for chemically castrating PCA patients.

The development of these biomedical interventions was largely spearheaded by Fernand Labrie who is the most prolific and highly cited author in the overall citation network (90 published papers, cited 5,714 times), buoyed by his publication record during the 1980s (Labrie et al. 1980, 1983, 1985; Tolis et al. 1982). Though Labrie and his colleagues' work is scant before 1981, a community composed of Labrie and his lab mates climbs to the most highly cited by 1982 and remains among the most prominent for roughly fifteen years. By the mid-1980s, these drugs had gained the support of leading scientists and regulatory agencies with the Federal Drug Administration (FDA) approving

the LHRH agonist leuprolide in 1985 and the anti-androgen flutamide in 1989 (Denmeade and Isaacs 2002; Leuprolide Study Group 1984). In Figure 3, this joint consensus is evidenced by a low point in the scaled modularity score around 1987.

By 1989, the network had once again fragmented to nearly the same level as the beginning of the decade. Researchers working on benign prostate hypertrophy again surface to one of the top research communities. However, the most prominent communities in the network were still conducting research on androgen ablation therapies with their main focus now centered on the long-term and combinatorial effects of these various drugs. In 1989, flutamide had just hit the open market. Alongside a large randomized control trial that found combined use of leuprolide and flutamide could prolong life expectancy (Crawford et al. 1989), researchers began conducting new clinical trials and bench studies to examine how flutamide affected and interacted with 5α-Reductase receptors (Andersson et al. 1991; Andersson and Russell 1990; Gormley et al. 1992; Russell and Wilson 1994; Thigpen et al. 1993). Mirroring the pattern of consensus formation that occurred with LHRH research in the 1980s, the network eventually coalesced around flutamide research as well, moving toward consensus by 1995.

During both swells in scaled modularity from 1980-1982 and 1988-1990, the development of novel pharmaceutical interventions drove scientific fragmentation. Although we do not see dramatic changes in how researchers think about testosterone and PCA (i.e. high levels of testosterone were still thought to promote carcinogenesis in the prostate gland), the hormone was always an essential component of the models that researchers used to develop emerging pharmaceuticals. In other words, the theory that *high*

levels of testosterone contributed to PCA was not only widely accepted, but also contributed to the economic underpinning of the network's structure.

During this time, researchers also used the "fuel for the fire" model to examine whether higher levels of testosterone in black men could also explain why black men had a higher risk of developing PCA than white men. While studies analyzing racial differences in testosterone were conducted in the 1970s (Jackson et al. 1975, 1977), 1980s (R. Ross et al. 1986; Henderson et al. 1988), and early 1990s (Nomura and Kolonel 1991; R. K. Ross et al. 1992, 1995), this work played a fairly marginal role in the PCA citation networks during the first historical juncture. Despite these studies not being heavily cited relative to ADT research, the models used to explain racial differences do change notably over time. Early work suggested that racial differences in testosterone were attributable to diet and lifestyle factors (Ahluwalia et al. 1981; P. Hill et al. 1979, 1982). As time progressed, PCA researchers began to test whether high *in utero* exposure to testosterone could explain why black men had a higher risk of PCA (Henderson et al. 1988). Eventually, researchers would suggest that hormonal differences between white, black and Asian men were the result of genetic mechanisms that "controlled" each racial group's overall risk of developing PCA (R. K. Ross et al. 1995). Regardless of these changes, researchers remained consistent in their belief that higher testosterone levels in black men helped explain a higher risk of PCA for that group. Implicit in this model is the notion that more testosterone contributes to prostate carcinogenesis. As we see in the following decade, this "fuel for fire" model would eventually come under great scrutiny.

Consensus and Contestation in TRT Research (1996-2008)

Although the network had been moving toward consensus before 1995, a number of counterintuitive findings arose in the mid-1990's which once again triggered community fragmentation. At peak consensus in 1996, four of the eight largest communities had mobilized around pharmaceutical development. While some focused on 5α-Reductase inhibitors like finasteride, others were still researching anti-androgens like flutamide, including the drugs' unanticipated and deleterious effects on long-term health outcomes. During this time, one group of researchers began to argue that flutamide *withdrawal* may improve clinical outcomes (Kelly and Scher 1993; Scher and Kelly 1993). This finding was counter-intuitive given that flutamide had been shown to improve PCA survival rates during the 1980s. Shortly after the first flutamide withdrawal studies, several meta-analyses began to emerge, finding that anti-androgens, in general, provided no added benefit when used as a supplement to medical and surgical castration (Caubet et al. 1997; Laufer et al. 2000; Dalesio et al. 2000). As a result, anti-androgen research became marginalized in the citation network with finasteride and other 5α-Reductase inhibitor studies taking over as the most highly cited cluster over the course of the decade.

By 1996, two large research communities had centered their work on biological risk factors associated with racial PCA disparities. As PCA disparities between black and white men became more pronounced over the 1980s and early-1990s (Krieger et al. 2012), several hormonal and genetic biomarkers were proposed to explain these trends. More specifically, a research group based out of the University of Southern California argued that racial differences in testosterone could help explain why African American men had higher rates of PCA compared to white and Asian men (R. Ross et al. 1986). Here, the "racial testosterone theory" aligns with the "fuel for fire" model in that both frame-

works assume the logic that more testosterone corresponds to a higher PCA risk. These models were seemingly substantiated by a highly cited study published in the mid-1990s that found higher levels of testosterone were associated with a higher incidence of PCA at the population level (Gann et al. 1996).

While testosterone was the initial vector through which these racial disparities were molecularized, PSA, 5-alpha reductase receptors, and androgen receptors each became a central focus in the literature as the decade progressed (S. J. Freedland and Isaacs 2005; Hsing 1996; J. Moul 2000; R. K. Ross et al. 1992, 1998; Hsing 2001). Although Ross and his colleagues (2005) suggest that the selection of candidate biomarkers to explain racial PCA disparities research is "somewhat arbitrary" (191), I would argue it is more accurate to suggest that these biomarkers *became* racialized after they had already been integrated into a more general (i.e. profitable) program of biomedical innovation. In other words, biomarkers are not as likely to take on racialized character until they have already been shown to have some utility within clinical settings.

Although this point deserves further elaboration elsewhere, there is at least some evidence that biomarkers become racialized as a result of biomedicalization and pharmaceuticalization in the context of PCA research. First, PSA testing was awarded FDA approval in 1986 and became more widely used during the early-1990s. As detailed in Chapter 3, researchers did not begin to test for racial differences in PSA until the mid-1990s when they began to advance racially-specific PSA testing regimens (Morgan et al. 1996; J. W. Moul 2000). Second, the ADT flutamide is designed to inhibit 5-alpha reductase production. As detailed in the last section, the use of flutamide grew throughout the 1980s, but researchers only began to conceptualize 5-alpha reductase as a racialized bi-

omarker after these drugs had already achieved considerable market success (e.g. R. K. Ross et al. 1992). Lastly, while researchers looked for how racial differences in testosterone since the 1960s, studies that conducted racial difference testing proliferated dramatically after the testosterone replacement therapy industry began to grow in the early-2000s (see Appendix V). In each of these examples, these biomarkers do not start as molecular markers that distinguish between biological "races," but instead *become* racialized only after researchers have already mobilized around these biomarkers as a means to develop pharmaceuticals and other biomedical interventions.

Returning more directly to the patterns of consensus and contestation observed in Figure 1, the mid-1990s also marked the reinvigoration of testosterone replacement therapy (TRT) research, which had laid relatively dormant since the 1950s (Watkins 2007, 2012). While early TRTs were marketed for the treatment of prostate hypertrophy (Oudshoorn 1994, 99–105), Huggins and Hodges (1941) finding that testosterone triggered prostate growth likely played a major role in thwarting the progression of the TRT market in the US and Europe. Despite these concerns, research in the 1990s, echoing the work done in the latter half of the 20th century, began suggesting that testosterone levels tended to drop later in life, contributing to reduced libido, erectile dysfunction, and depression after men reach the age of 40 (Tenover 1992; Wang et al. 1996).

From this work, two major research programs emerged with one centering on health outcomes that correlate with hypogonadism (i.e. clinically low levels of testosterone) and another focusing on the benefits of TRTs for treating those symptoms. In the process, a series of surprising studies followed, which suggested that men with clinically *low* testosterone could also develop PCA (Michael A. Hoffman, DeWolf, and

Morgentaler 2000; Morgentaler, Bruning, and DeWolf 1996; Schatzl et al. 2001). This point is important because it undermines the notion that more testosterone contributes directly to a higher risk of PCA, which, at the time, had been the unquestioned tenet that ADTs had been developed on for over 50 years.

As the 1990s progressed, the joint controversies of (1) testosterone's association to PCA and (2) the potential dangers of using anti-androgens drove dissensus in the citation networks. This dissensus peaks in 1999, which is followed by a sharp decline and a plateau in 2006. During this period, several large studies, reviews, and meta-analyses were still publishing contradictory findings: some found that higher testosterone levels were linked to an increased risk of developing PCA (Shaneyfelt et al. 2000), some found that testosterone and PCA shared no association (Eaton et al. 1999; Kaaks, Lukanova, and Sommersberg 2000; Stattin et al. 2004), and others argued that *lower* testosterone levels may be a risk factor for PCA (Hoffman, DeWolf, and Morgentaler 2000; Morgentaler, Bruning, and DeWolf 1996; Schatzl et al. 2001).

The major turning point in this debate seems to have occurred in 2006 when Harvard University's Abraham Morgentaler proposed the "saturation model" as an alternative to the "fuel for fire" model. In his saturation model, Morgentaler (2006, 2012) proposes that testosterone only contributes to PCA growth at castrate levels. In other words, beyond a particular saturation point (i.e. beyond castrate levels), cell proliferation in the prostate gland no longer responds to additional hormonal stimulation. More precisely, as Morgentaler (2012) summarizes:

> "prostate tissue requires androgens for optimal growth. However, it can only use a relatively small amount, beyond which additional androgen is merely excess. The saturation point is well below physiologic concentrations, which explains why manipulation of serum T into or out of the castrate range produces large changes

> in prostate biology, whereas normal prostate and [PCA] appear completely indifferent to variations in serum T from the near-physiologic to supraphysiologic range" (Morgentaler 2012).

Morgentaler suggests that this model explains away the literature's contradictory claims by showing more precisely how testosterone binds to the prostate at the cellular level. While it does not seem that *all* clinicians adhere to the saturation model today, the citation network does take a notable shift towards consensus shortly after the manuscript is published.

The implications of this paradigm shift also seem to have had a dramatic effect on the growth of the TRT industry. First, Morgentaler's saturation model reframes the relationship between testosterone and PCA, removing the concern that TRTs exacerbate PCA risk for those that consume them (Morgentaler 2006, 2013). Furthermore, the saturation model opens up the possibility that TRTs could actually be *beneficial* for the treatment of PCA. This is because, according to Morgentaler and his colleagues (Khera et al. 2014; Morgentaler 2013), adding testosterone to hypogonadal men's bodies would bring them above the saturation point and help minimize the symptoms of "testosterone deficiency" by bringing hormone levels back within a normal range. While TRTs are now more widely accepted among leading scientific experts, the Endocrine Society's clinical guidelines still recommend *against* giving men with PCA diagnoses testosterone in fear that it will exacerbate their risk of mortality (Bhasin et al. 2010, 2018). As a recent study of TRT prescription patterns suggests, clinicians still heed to this advice and regularly withhold testosterone from PCA patients (Jasuja et al. 2017).

As shown in Table 4, these clinical guidelines also explicitly advise clinicians not to give TRTs to African American men based on their elevated PCA risk (Bhasin et al.

2010, 2018). However, this recommendation makes little sense unless (1) there are racial differences in testosterone and/or (2) higher levels of testosterone contribute to PCA risk. As the data from my second chapter demonstrate, the majority of studies that evaluate testosterone differences between white and black men offer null results. Furthermore, if Morgentaler and his colleagues are correct, the Endocrine Society's clinical guidelines are not only predicated on the historical myth of the "fuel for fire" model. Extending that logic even further, I argue it is also predicated on the historical myth of the "racial testosterone theory." Importantly, these contradictions are not a by-product of outdated expertise, as the clinical guidelines were last updated in May of 2018 with no change to the underlying logic that blocks equal access to TRTs for black men (Bhasin et al. 2018). Perhaps more surprisingly, I have still yet to encounter *any* publication that debates whether these racialized claims about TRT prescribing should be reassessed, despite the 2010 version of these clinical guidelines being among the most highly cited publications in all of the testosterone literature (as of March 1st they were cited 1,092 and 1,647 times by Web of Science and Google Scholar respectively). Given the prominence that these historical myths still have in the literature today, my analysis is suggestive of the power that early false positive findings (like Huggins and Hodges' research on testosterone and PCA) have on biomedical research, including the supposed "gold-standards" of evidence-based medicine.

Despite these contradictions, the latter half of the 2000s was marked by a fairly dramatic shift towards scientific consensus. While Morgentaler's work triggered a spike in dissensus in the mid-1990s, it seems that his saturation model also played a large role in adjudicating the controversy that existed about the relationship that testosterone and

PCA share. By 2008, the network reached its highest consensus point across my overall temporal window, evidenced by the low scaled modularity score in Figure 3. In congruence with this peak consensus point, the Endogenous Hormones and Prostate Cancer Collaborative Group 4/4/2019 2:28:00 PM published a meta-analysis of 18 prospective studies of nearly 4,000 men that found no association between the risk of PCA and serum testosterone concentrations. Today, this paper has been at the fulcrum of many ongoing debates about the role that hormones play in the development of PCA.

TABLE 4. Conditions in which testosterone administration is associated with a high risk of adverse outcome and for which we recommend against using testosterone

Very high risk of serious adverse outcomes
Metastatic prostate cancer
Breast cancer
Moderate to high risk of adverse outcomes
Unevaluated prostate nodule or induration
PSA >4 ng/ml (>3 ng/ml in individuals at high risk for prostate cancer, such as African-Americans or men with first-degree relatives who have prostate cancer)
Hematocrit >50%
Severe lower urinary tract symptoms associated with benign prostatic hypertrophy as indicated by AUA/IPSS >19
Uncontrolled or poorly controlled congestive heart failure

Table 4 shows the Endocrine Society's clinical guidelines for testosterone replacement therapies, which explicitly advise clinicians not to give African Americans these drugs. Note that PSA levels of >4 ng/mL are typically the cutoff for receiving testosterone, but African American men are thought to have lower cutoffs, as noted in Chapter 3, because of their supposedly higher risk of developing that disease.

Consensus and Contestation in Contemporary PCA Research (2009-2017)

Since its peak consensus point in 2008, the research network has exhibited only minor patterns of dissensus. Like in past periods, this discord has been driven, at least in part, by pharmaceutical research, but scientists in this decade are primarily debating the deleteri-

ous effects of ADTs. By 2008, ADTs had been widely distributed for over 15 years. As a result, large-scale longitudinal datasets began to emerge documenting that ADT consumption was linked to an increased risk of heart disease, diabetes, metabolic syndrome, sarcopenia, osteoporosis and fracture risk (Basaria 2008; Isbarn et al. 2009; Levine et al. 2010; L. G. Taylor, Canfield, and Du 2009). In 2008, this topic was fairly nominal in the citation data, but, by 2013, this debate was circulating through one of the most central research communities in the network. In addition to this group comprising a larger percentage of the network, the scaled modularity score also rose slightly. Today, the deleterious effects of ADTs on men's health are still being debated with new strategies being offered to help mitigate the damage these drugs induce (P. L. Nguyen et al. 2015).

After 2008, the debate about the association between testosterone and PCA has also resurfaced in some circles. After the Endogenous Hormones and Prostate Cancer Collaborative Group (2008) published the meta-analysis demonstrating that testosterone and PCA shared no association, a number of researchers have been working to adjudicate the literature's contradictory findings. For example, Salonia et al. (2012) argues that the association between testosterone and PCA may follow a nonlinear-U pattern where those with relatively high and relatively low levels of the hormone exhibit an increased risk to those with normal testosterone levels. Klap et al.'s (2014) review suggests that while low testosterone does consistently predict PCA risk, the association between high testosterone and PCA is much more tenuous. In line with the insights I offered in Chapter 2, these authors also point out that existing contradictions are likely due to very poor methodological standards. For example, PCA researchers often fail to consider diurnal patterns, age variations, and/or collect repeat measurements of testosterone in their studies.

Of course, the most obvious and profound implications of these debates relate to the growth or decline of the TRT market. Multiple research groups have called for new clinical trials to assess how TRTs impact PCA patients, reminding clinicians to obtain informed consent to ensure their patients know that the long-term repercussions are still unknown (Khera et al. 2014; Klap, Schmid, and Loughlin 2015). While there is still limited evidence available, the best available evidence suggests that TRTs do not exacerbate PCA risk among men who have hypogonadism (Boyle et al. 2016; Cui et al. 2014; Shabsigh et al. 2009). Of course, it is necessary to reiterate that the evidence on this topic is still quite limited.

When examining how these controversies play out structurally, the citation networks from 2014-2017 generally bifurcated into two large components with smaller research groups embedded within. In one component, the ADT and TRT controversies have clustered together, suggesting that the evidence used to inform both debates has coalesced over time. The second large component focuses mostly on the molecular mechanisms underpinning castration-resistant PCA and other novel forms of ADTs that being developed to treat this disease.

Although the concept of castration-resistant PCA was introduced around the turn of the century (Chen et al. 2004; Feldman and Feldman 2001), this variant of the disease only became a prominent focus of the citation data around the year 2008. This is likely due to the development of several new anti-androgen therapies designed to treat castration-resistant PCA in the early half of this period (de Bono et al. 2011; Locke et al. 2008; Tran et al. 2009; Scher et al. 2012). As we saw in each of the previous two epochs, the scientific structure of these citation networks is often entwined with the development and

testing of various hormone-based pharmaceuticals. While the specific products change in each period, there is a robust effect on the patterns of consensus and contestation in each of the three historical junctures.

Discussion

Using a quantitative method to examine patterns of scientific consensus in PCA research from 1980-2017, this chapter demonstrates that the relationship between testosterone and PCA has undergone a dramatic paradigm shift over the past 25 years. While *high* levels of testosterone were considered a marker of risk in PCA research for more than seven decades, prominent biomedical scholars now argue that clinically *low* levels of testosterone may be a better indicator of PCA risk. More generally, this chapter shows that pharmaceutical development can play an important role in structuring patterns of scientific consensus. From 1980-2017, the data exhibit four waves of consensus and contestation that coincide closely with research programs on hormone-based pharmaceuticals. While two generations of ADTs shaped these ebbs and flows from 1980-1996, TRT research provoked a period of structural disjunction around the year 2000 that eventually tapers off in the latter half of the decade. Over the past ten years, the network has reached a relative level of consensus, which has been slightly interrupted by ongoing debates around the risks associated with TRT and ADT consumption.

This research lends to two fields of inquiry in science & technology studies. First, according to Abbott (2001), there are three patterns in the formation of scientific consensus: (1) spiral - where controversial research questions are answered and reassessed in greater detail at a later point in time; (2) cyclical - where research questions are assessed

with consistent movement towards consensus; and (3) flat - where no consensus formulates in the field. My data show clear support for the cyclical tenet of Abbott's (2001) theory, which also aligns with the Shwed and Bearman's (2010) work on the structure of scientific consensus in research examining the effects of smoking on cancer. Figure 3's general trend downward over time indicates a general pattern toward scientific consensus while the swells in the scaled modularity score between 1987-1990, 1996-1999, and 2004-2006 suggest that there is also a cyclical pattern of scientific fragmentation that reoccurs over time as well. These findings add to the growing literature that employs this methodology to examine patterns of scientific consensus (Adams and Light 2014, 2015; D. Navon and Shwed 2012; Shwed 2015; Shwed and Bearman 2010).

Second, this work speaks to the ways that various biomarkers within a given research field can become racialized over time. While my analysis mainly speaks to how the racialization of testosterone has been embedded into prostate cancer research, the second section also finds that prostate-specific antigen and androgen receptors have also been racialized in this domain. Despite my historical analysis suggesting that scientists have moved towards relative consensus, the paradigm shift from high to low levels of testosterone being a marker of PCA risk has not played out in the same way for all racial groups. What most researchers do not realize is that the "fuel for fire" theory has long been racialized. When Ross and his colleagues (1986) began advancing the racial testosterone theory, higher levels of testosterone were uncontroversially linked to PCA. And while the "fuel for fire" thesis has shifted over time, the racial testosterone theory, or at least its implicit endorsement within the clinical guidelines, has gone unchallenged. Throughout my research for this project, I am still yet to find any explicit debates that

speak directly about how the paradigm shift away from the "fuel for fire" theory affects different racial groups or and that the Endocrine Society's clinical guidelines racially-specific claims should be reassessed (Bhasin et al. 2010, 2018).

With that said, this chapter, as well as this dissertation project more generally, challenges the Endocrine Society's recommendations that clinicians should withhold TRTs from African American men based on their higher risk of PCA. These claims are predicated on the notion that (1) black and white men have differing levels of testosterone and (2) that higher levels of testosterone contribute to PCA risk. In my second chapter, I show that extant evidence examining population differences in testosterone do not support the claim that this hormone varies between black and white men. In this chapter, I show that the literature's shift from high to low levels of testosterone as a marker of risk also undermines the second assumption of the Endocrine Society's recommendation. If TRTs do not contribute to one's risk of PCA, why do these life-altering drugs continue to withheld from black consumers?

And while these guidelines have played out to significantly decrease the likelihood that black men will receive TRTs in clinics around the United States (Jasuja et al. 2017), TRTs are certainly not immune from their own controversies. Despite the reported benefits of using TRTs for sexual function, weight loss, metabolic syndrome, and diabetes (Corona, Monami, et al. 2011; Corona et al. 2014, 2016; Cai et al. 2014), the Federal Drug Administration (FDA) has now mandated that all TRTs include a warning label that they may induce an increased risk of cardiovascular disease (CVD) (FDA 2015). Some experts, including Abraham Morgentaler, have contested the FDA's decision, arguing that select studies reporting negative CVD outcomes were sensationalized by the media

and that extant evidence does not support the claim that TRTs are dangerous (Morgentaler et al. 2016). However, Xu et al.'s (2013) recent meta-analysis suggests that the literature is predictably divided; existing studies that are funded by pharmaceutical companies report no increased risk of CVD while studies without this source of funding reveal a two-fold increase of CVD risk (see also Huo et al. 2016).

Perhaps, it is not surprising then that Morgentaler is well connected to the pharmaceutical industry's various funding mechanisms. In the 49 manuscripts that he contributed to in my citation data, Morgentaler reports conflicts of interest with at least 12 different pharmaceutical companies, including AbbVie, Bayer, Lilly, Pfizer, Schering, and Solvay. Morgentaler has also started his own pharmaceutical company - Men's Health Boston - a clinic that prescribes TRTs using the paradigm of personalized medicine. How this instantiation of personalized medicine will play out to impact future patterns of racialization in TRT distribution still remains to be seen. Unfortunately, history does not bode well for the possibility of equal access to pharmaceuticals for black and white men. For the time being, it is difficult to ascertain how the paradigm shift that Morgentaler is responsible for spearheading is shaped by pharmaceutical funding and whether the limited evidence available will turn out to support his proclamations that TRTs may help treat select PCA patients (Khera et al. 2014; Morgentaler 2013). Despite evidence suggesting that the literature has moved *toward* scientific consensus over the past four decades, this constellation of controversies seems far from being settled.

Chapter 5: Conclusion

In this dissertation, I show how testosterone has been racialized in scientific research and examine the impact that this has on the construction of racial disparities in prostate cancer. Following the conceptual model outlined in my introductory chapter (see Figure 1), this project critically examined the associations between: (1) testosterone and race, (2) race and prostate cancer, and (3) testosterone and prostate cancer. In Chapter 2, I showed how testosterone is racialized in scientific research by evaluating studies that measure population differences in testosterone. To do this, I conducted a content analysis of 147 studies on this topic, finding that the literature provides scant evidence to support racial differences in testosterone. Despite this being the case, the racial testosterone theory – which holds that those of African descent have the highest levels of testosterone, those of Asian descent have the lowest level of testosterone, and those of European descent have testosterone somewhere in between – continues to circulate through biomedical research today through the use of various mechanisms used to enact racial difference. I argued that ambiguity, absence and the recycling of datasets all play a role in shaping how biomedical researchers enact population differences, which preserves the cultural myth of testosterone as a molecular marker of racialized masculinity.

In Chapter 3, I evaluated the linkage between race and prostate cancer. Biomedical and epidemiological researchers widely claim that black men suffer from and ultimately die from prostate cancer at two to three times the rate of white men. While scientists often acknowledge the possibility that social inequalities shape racial disparities, they rarely study these social and structural factors or advance recommendations to promote policy interventions to address structural issues. Researchers do, however, frequent-

ly advance the theory that black men have more aggressive biologies, which they argue helps to explain why disparities in prostate cancer incidence and mortality persist between the two groups. Here, the racialization of these biological claims also shapes differences in how each group is administered prostate cancer treatments, including group-differentiated prostate-specific antigen testing, racially-specific treatment guidelines, and various pharmaceutical and surgical interventions. Furthermore, in the process of molecularizing racial health disparities, researchers also fail to prioritize the structurally-oriented interventions that social scientists argue address the fundamental causes underlying racial health disparities in the first place.

In Chapter 4, I conducted a historical analysis of how the relationship between testosterone and prostate cancer has changed over time. Using Shwed and Bearman's (2010) quantitative method to examine patterns of scientific consensus in citation networks from 1980-2017, I documented a paradigm shift in the association between testosterone and prostate cancer over the past 25 years. While *high* levels of testosterone were considered a robust predictor of prostate cancer risk for more than seven decades, a group of biomedical experts now argue that clinically *low* levels of testosterone may, in fact, be a more accurate risk factor for diagnosing this disease. As a result of this change, I argued that the Endocrine Society's clinical guidelines on testosterone replacement therapies, which suggest that neither prostate cancer patients nor African American men should receive these drugs, need to be updated to reflect today's best available evidence. In essence, these guidelines are predicated not only on the historical myth that more testosterone triggers prostate carcinogenesis, but also on the myth that racial differences in testosterone help explain prostate cancer disparities.

As a whole, this dissertation speaks to the sociological literatures on medicalization and pharmaceuticalization. First, past work has focused on the expansion of the testosterone replacement therapy industry, suggesting that this market's expansion is primarily driven by marketing tactics that attempt to frame this drug as a viable way to rejuvenate men's masculinity and bolster sexual function (Conrad 2007; Marshall 2007, 2009a; Watkins 2007, 2008, 2012). My work adds to these discussions by describing the ways that testosterone therapies are talked about in other biomedical contexts like prostate cancer research. By looking at testosterone outside of the context of sexual medicine, we can see why testosterone's market dwindled in the first place; testosterone was long thought to contribute to PCA, which led to medical professionals contraindicating TRTs for decades. More importantly, my work shows how knowledge production in the context of prostate cancer (i.e. that higher levels of testosterone contribute to prostate carcinogenesis) shape the Endocrine Society's clinical guidelines for prescribing testosterone therapies and, in turn, leads these drugs to being administered in different ways for white and black men.

Thus, my larger contribution to this sociological literature is showing that the testosterone replacement therapy market is also racialized in addition to being highly gendered. This insight is important, in one sense, because it suggests that sociologists should attend to the ways that "gold-standard" studies are constructed in distinct ways for different racial or ethnic groups. Given their growing prominence in evidence-based medicine today, these studies play an important role in brokering knowledge to diverse fields. They also offer an important sight for critically-oriented social scientists to intervene on how biomedical scholars construct race and gender in essentialist ways. Further, by demon-

strating how testosterone has become racialized and, more importantly, how this process affects access to testosterone replacement therapies for black men, my work provides a complementary example to the case of BiDil (Kahn 2008, 2013; Pollock 2012). As others have shown (Hansen and Netherland 2016; Netherland and Hansen 2017), pharmaceuticals do not necessarily need to be marketed and/or developed under the paradigm of personalized medicine to become racialized. My dissertation shows some of the mechanisms through which this process manifests. For example, the insights outlined in Chapter 2, including those pertaining to ambiguity, absence and data recycling, are all examples of research practices that perpetuate racial difference testing, which in turn sustains the persistent medicalization and pharmaceuticalization of race in biomedical research. The mechanisms may help to reveal how racial difference is enacted in the biomedical contexts.

My work also shows one way that the molecularization of race becomes embedded in the larger project of health disparities research. As Bliss (2018) suggests, epidemiological researchers regularly advance both social *and* biological arguments when explaining racial health disparities. In the sociogenomic paradigm, researchers rarely take an ontological stand on what race "is," but instead more often focus on what they can do with race to advance their overall research programs (Chun 2013; Shim et al. 2014). While my findings, especially in Chapter 3, align closely with these insights, I also documented *how* race has been molecularized in prostate cancer research and how this shapes the distribution of various biomedical interventions. Today, black men are more likely to be diagnosed with prostate cancer, in part because they are administered prostate-specific antigen tests more regularly on the basis of racially-specific guidelines. However, black men are also less likely to receive quality care (i.e. androgen deprivation

treatments and active surveillance) for this disease. While these trends may not be surprising to many social scientists, biomedical researchers continue to advance molecular claims, oftentimes at the expense of socioeconomic interventions. As a result, these disparities are reproduced without any clear steps to address the underlying structural causes, despite the scientific evidence being quite clear. In the future, prostate cancer researchers would be wise to shift their focuses towards analyzing how social and environmental factors, including racial segregation and exposure to environmental toxins, have on the progression of this disease and proposing solutions that public health and governmental agencies can do to minimize these deleterious effects.

I believe that Figure 1, which provides a model to critically interrogate the relations between biomarkers, race and health outcomes more broadly, could be employed by other researchers working at the intersection of sociology, anthropology, and science & technology studies. This triangular model helps to provide a conceptual framework for organizing work in relation to existing literature and emphasizing new types of relationships yet unexplored. To date, most work focuses on examining how the molecularization of race emerges within particular fields like genomics (Bliss 2012, 2018), oncology (Wailoo 2011), or cardiovascular research (Pollock 2012; Shim 2014). My third chapter fits into this mold by showing how racial disparities in prostate cancer are molecularized and the impact that this has on differing patterns of biomedical interventions. However, by interrogating the other two relations in this triangle, I was also able to examine racialization from an alternative lens.

For example, by looking at a specific biomarker (testosterone), my analysis helps to reveal the broader implications of molecularization, across and beyond specific bio-

medical subfields. The racialization of testosterone not only affects scientific understandings of prostate cancer, but can also shape research being conducted on cardiovascular disease, polycystic ovary syndrome (Carlin and Kramer 2018), athletics (Karkazis and Jordan-Young 2018) and various other kinds of biosocial research (R. Jordan-Young and Karkazis forthcoming). Given that the structure of biomedical research has become increasingly interdisciplinary, focusing on objects may help to garner important insights about how racializing mechanisms travel across scientific fields in complex ways.

While testosterone is the main biomarker that this project was developed around, Chapters 3 and 4 both demonstrate that testosterone is just one of many biomarkers that researchers employ to molecularize prostate cancer disparities between black and white men. Focusing on testosterone research, as I did in Chapter 2, helped me to identify some of the mechanisms that researchers use to enact racial differences. However, conducting the same analysis on each of the dozens of other biomarkers that scientists have racialized would be quite time consuming, and unlikely to catch up to biomedicine's ever-shifting gaze. For example, as Chapters 3 and 4 demonstrate, researchers shift from testosterone to prostate-specific antigen to androgen receptors as the biological entities that are used to molecularize racial disparities. Future research should examine the similarities and differences in how this practice of "biomarker cycling" unfolds over time.

Lastly, framing my fourth chapter around the relationship between testosterone and prostate cancer allowed me to examine the racialization of prostate cancer and testosterone from another perspective. I was able to examine how research groups within the citation networks became more prominent or marginalized over time, providing a better basis to know when and by whom the racialization of testosterone was advanced in this

domain. While race was not the primary focal point of this analysis, this chapter arguably informs the most important policy implications that flow from this dissertation. In examining how the association between testosterone and prostate cancer changed over time, I argue that the *structure* of scientific consensus in prostate cancer research is opposed to the recommendations that the Endocrine Society advances. While this is not a meta-analysis or systematic review, it does provide another form of evidence that goes beyond particular scientists or research groups taking opposing stands in an ongoing controversy. More importantly, by situating the findings of this chapter alongside the other two links in the triangle, my argument that the Endocrine Society needs to change their policies carries more legitimacy.

With this said, my work speaks to biomedical researchers who incorporate testosterone in scientific models across various biomedical fields. First, this dissertation demonstrates that racial differences in testosterone are unsupported by existing scientific evidence, despite the claims being widespread through the literature. I believe these insights can help to address why this myth continues to proliferate, and potentially help to stem its flow. Most plainly, my work shows that the Endocrine Society's clinical guidelines on TRTs need to remove the recommendation to withhold these pharmaceuticals from black men. These suggestions are predicated not only on the racial testosterone theory, a myth unsupported by extant literature, but also on an antiquated model that suggests testosterone exacerbates one's risk of developing prostate cancer. My work contributes to a growing consensus of researchers working at the intersection of endocrinology, oncology and urology (see Chapter 3) suggesting that these guidelines are outdated and need to be revised to reflect existing best evidence.

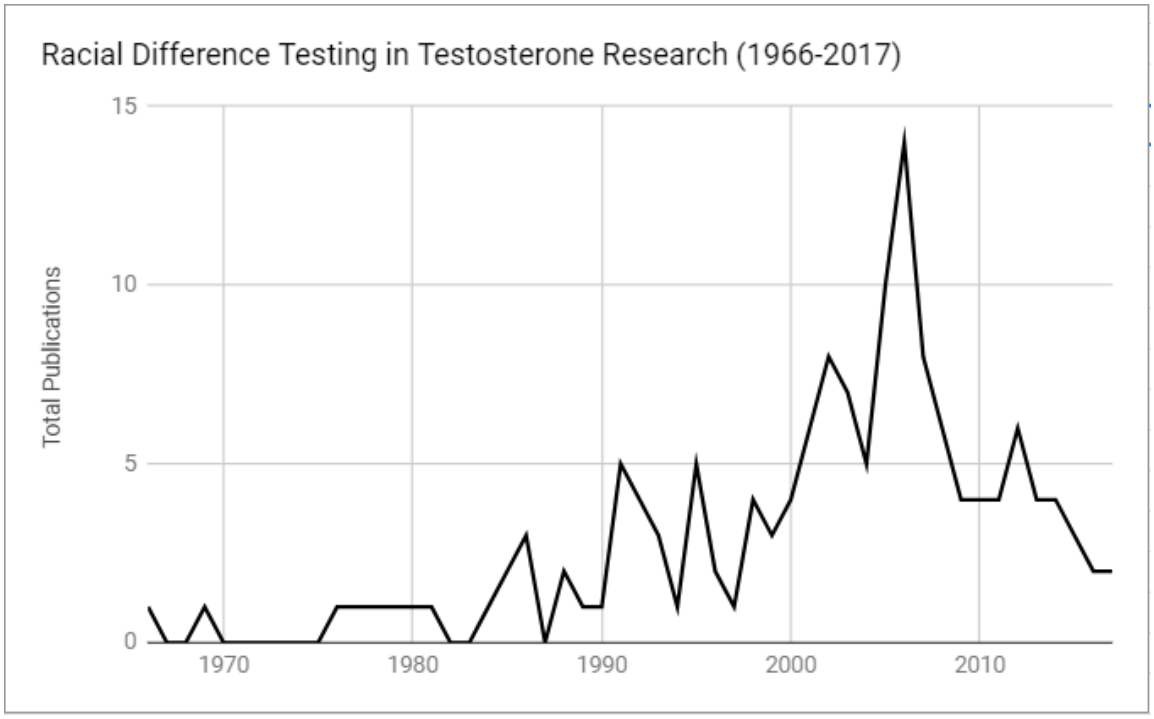
While my intention in advancing these recommendations is to advocate for equal access to pharmaceuticals between black and white men, it is also important to acknowledge the risks – both known and unknown – of using testosterone replacement therapies. As I outlined at various points in this manuscript, testosterone-based pharmaceuticals have been found to contribute to cardiovascular risk, especially when the papers reporting these results are not shaped by pharmaceutical funding (Lin Xu et al. 2013; Huo et al. 2016). Furthermore, as I warned in Chapter 3, the paradigm shift from high to low testosterone as a marker of prostate cancer risk was largely spearheaded by researchers, like Abraham Morgentaler, who are funded by major pharmaceutical corporations. Therefore, it is still not entirely clear how funding mechanisms manufacture doubt and uncertainty in this context (see Michaels and Monforton 2005) or how the evidence my recommendations are predicated upon may be biased by these factors.

Lastly, a major theme of this dissertation is the notion of absence and omission. Throughout this manuscript, I have pointed to various controversies that have surfaced in biomedical research – from the dangers associated with hormone replacement therapies to the recent realization that androgen deprivation therapies may induce various acute health risks. While cardiovascular risks are the main point of contention in contemporary literature on testosterone replacement therapies, the long-term use of these drugs is still largely unknown. As these pharmaceuticals continue to be used more widely, it will be important for advocates of these products to keep a watchful eye on the ways that these drugs may induce unanticipated effects. Thinking about this through the findings of this dissertation, I have to recognize the possibility that, much like the case of hormonal replacement therapies over two decades ago (Krieger et al. 2005), black men's reduced ac-

cess to testosterone-based drugs may, in fact, serve as a protective mechanism against mortality. In other words, there is a distinct possibility that the negative risks of testosterone replacement therapies may end up disproportionately affecting economically privileged white men who have used these drugs more regularly to date.

Despite this potential, there is still no clear rationale to why clinicians should follow different clinical guidelines or take part in different biomedical practices for treating black and white men for prostate cancer. This dissertation demonstrates the ways that the assumptions underlying these policies play out in contemporary biomedical research, and I hope that this work will have some bearing on how these policies are reconstructed moving forward. This will likely require social scientists to take part in collaborations with biomedical researchers to reveal and combat the ways that racism is perpetuated in scientific research. It is to this project I now turn…

Appendices



Appendix I demonstrates the growth of published articles that conduct racial difference testing of testosterone from 1966-2017.

Appendix II. Summary Table of Data Used in Chapter 2							
Study	**Sample**	**Recycled Data**	**Time Diff.**	**Age**	**Weight / BMI**	**Chronic Illness**	**SES**
Abdelrahaman 2005	Children		No	Yes	No	Yes	No
Agurs-Collins 2012	Children	HUB	Yes	Yes	No	Yes	No
Ahluwalia 1981	Men	DC, Nigeria	No	Yes	Yes	Yes	No
Al Fozan 2005	Women		No	Yes	No	No	No
Araujo 2007	Men	BACH	No	Yes	Yes	Yes	No
Araujo 2008	Men	BACH	No	Yes	Yes	No	No
Asbell 2000	Men		Yes	No	Yes	No	No
Berman 2001	Women		No	No	Yes	Yes	No
Bribiescas 1996	Men	Congo, Nepal, Paraguay	No	Yes	No	No	No
Campbell 2003	Men	Kenya	No	Yes	No	No	No
Campbell 2006	Men	Kenya	No	Yes	Yes	No	No
Cappola 2007	Women		No	Yes	No	Yes	No
Carmina 1992	Women		No	No	No	No	No
Carmina 2003	Women		No	No	Yes	No	No
Cauley 1994	Women		No	Yes	No	Yes	No
Chang 2016	Women		No	Yes	No	No	No
Chen 2004	Men		No	Yes	No	Yes	No
Cheng 2005	Men		No	Yes	Yes	Yes	No
Choi 2013	Men		Yes	Yes	Yes	No	No
Colangelo 2007	Men	CARDIA	No	Yes	No	No	Yes
Coward 2010	Men		No	Yes	No	Yes	No
Crawford	Men		No	No	No	Yes	No

2007							
Cunningham 2014	Men		Yes	Yes	No	No	No
DeJong 1991	Men		No	Yes	No	No	No
Dowling 1993	Women		Yes	Yes	No	Yes	No
Dunaif 1993	Women		No	Yes	No	Yes	No
Eastham 1998	Men		No	Yes	Yes	No	No
Ellis 1992	Men	VES	No	Yes	No	No	No
Ellison 1989	Men	Kenya	No	No	Yes	No	No
Ellison 1996	Men	Nepal	No	No	Yes	No	No
Ellison 2002	Men	Congo, Nepal, Paraguay	No	Yes	Yes	No	No
Engmann 2017	Women		Yes	Yes	No	Yes	Yes
Ettinger 1997	Mixed	CARDIA	Yes	Yes	Yes	Yes	No
Ewing 1978	Women		No	No	Yes	No	No
Falk 2002	Women		No	Yes	No	Yes	No
Freedman 1991	Men	VES	No	Yes	No	No	Yes
Gapstur 2002	Men	CARDIA	No	Yes	No	No	No
Giton 2011	Men		No	Yes	No	Yes	No
Glintborg 2010	Women		Yes	Yes	No	Yes	No
Golden 2007	Women	MESA	No	Yes	No	Yes	No
Goldin 1986	Women		No	No	Yes	Yes	No
GuerraGarcia 1969	Men		Yes	No	Yes	No	No
Guo 2012	Women		Yes	Yes	No	Yes	No
Hall 2008	Men	BACH	No	Yes	No	Yes	Yes
Hannon 2012	Children		Yes	Yes	No	Yes	No
Heald 2003	Men		Yes	Yes	No	No	No

Heald 2007	Men		No	Yes	No	No	No
Henderson 1988	Women		Yes	Yes	Yes	Yes	No
Hill 1976	Mixed		No	No	Yes	No	No
Hill 1979	Men		Yes	No	Yes	Yes	No
Hill 1980	Men		No	No	No	Yes	No
Hill 1984	Children		No	Yes	Yes	No	No
Hillman 2014	Women		Yes	Yes	No	No	No
Hoffman 2005	Mixed		No	No	Yes	Yes	No
Hu 2015	Men	NHANES	Yes	Yes	No	No	No
Hui 2003	Children		Yes	Yes	Yes	No	No
Jackson 1977	Men	DC, Nigeria	No	Yes	Yes	Yes	No
Jakobsson 2006	Men		No	No	Yes	No	No
Jin 1999	Men	China, Australia	Yes	Yes	Yes	No	No
Jin 2000	Men	China, Australia	Yes	Yes	Yes	No	No
Kauffman 2002	Women		No	Yes	Yes	Yes	No
Kauffman 2006	Women		No	Yes	No	Yes	No
Kehinde 2006A	Men	Kuwait, Oman, Germany	No	Yes	Yes	Yes	No
Kehinde 2006B	Men	Kuwait, Oman, Germany	No	Yes	Yes	Yes	No
Key 1990	Women		Yes	No	Yes	No	No
Kim 2012	Women	DPP	Yes	No	Yes	Yes	No
Kim 2013A	Women	SWAN	No	Yes	No	No	No
Kim 2013B	Women	DPP	Yes	Yes	No	No	No
Kirchengast 2017	Men		No	No	Yes	No	No

Kitabchi 1999	Women		No	Yes	No	No	No
Knochenhauer 1998	Women		No	No	Yes	No	No
Kobayashi 1966	Mixed		Yes	Yes	Yes	No	No
Kubricht 1999	Men		No	Yes	Yes	No	No
Kumar 2005	Women		No	Yes	Yes	Yes	No
Kupelian 2008	Men	BACH	No	Yes	No	Yes	No
Ladson 2011	Women		No	Yes	No	Yes	No
Lagiou 2011	Women		Yes	No	Yes	No	No
LamonFava 2005	Women		No	Yes	Yes	Yes	No
Lasley 2002	Women	SWAN	No	Yes	Yes	Yes	No
Lee 2010	Children	NHANES	No	Yes	No	No	Yes
Legro 2006	Women		No	No	Yes	Yes	No
Lewis 2005	Men		No	Yes	Yes	Yes	No
Litman 2006	Men	BACH	No	Yes	No	Yes	Yes
Lookingbill 1991	Mixed		Yes	No	Yes	No	No
Lopez 2013	Men	NHANES	Yes	Yes	Yes	No	No
Manson 2001	Women		Yes	Yes	No	Yes	No
Marks 2006	Men		No	Yes	No	No	No
Mazur 1995	Men	VES	No	Yes	No	Yes	Yes
Mazur 2009	Men	NHANES	No	Yes	No	No	No
Mazur 2016	Men	NHANES	Yes	Yes	No	No	Yes
McTiernan 2008	Women		No	No	Yes	Yes	No
Miller 1985	Men		Yes	No	Yes	No	No
Mohler 2004	Men		Yes	Yes	No	No	No
Mongraw-Chaffin 2015	Mixed	MESA	No	Yes	No	No	Yes
Morrison 2000	Children	SHLMAS	Yes	No	Yes	No	No

Morrison 2002	Children	SHLMAS	Yes	Yes	Yes	No	No
Muller 2009	Men		No	No	Yes	Yes	No
Mydlo 2001	Men		No	Yes	No	No	No
Nyante 2012	Men	NHANES	No	Yes	No	No	No
Orwoll 2006	Men	MrOS	No	Yes	No	Yes	No
Orwoll 2010	Men	MrOS	No	Yes	No	No	No
Osegbe 1988	Men		No	No	Yes	Yes	No
Platz 2000	Men		Yes	No	Yes	Yes	No
Pollard 2006	Women		No	Yes	No	No	No
Potischman 2005	Women		Yes	Yes	Yes	Yes	Yes
Rampp 2008	Women		Yes	No	Yes	No	No
Randolph 2003	Women	SWAN	No	Yes	No	No	No
Reed 1993	Women		No	No	Yes	No	No
Richard 2014	Men		No	Yes	No	No	No
Richards 1992	Children	Bogalusa	Yes	Yes	No	No	No
Rohrmann 2007	Men	NHANES	No	Yes	No	No	No
Rohrmann 2009	Children	HUB	Yes	Yes	No	Yes	No
Ross 1986	Men	USC, UCLA	Yes	Yes	No	No	No
Ross 1992	Men	USC, UCLA	Yes	No	Yes	No	No
Santner 1998	Men		Yes	No	Yes	Yes	No
Schmid 2004	Women		Yes	Yes	No	No	No
Setiawan 2006	Women		Yes	Yes	No	Yes	No
Simmons 1995	Women		Yes	No	Yes	Yes	No
Sowers 2003	Women	SWAN	No	Yes	Yes	No	No
Sowers 2006	Women	SWAN	No	Yes	No	No	No

Spencer 2007	Women		No	Yes	No	Yes	No
Srinivasan 1985	Children	Bogalusa	Yes	No	Yes	No	No
Srinivasan 1986	Children	Bogalusa	Yes	Yes	Yes	No	No
Sutton-Tyrrell 2005	Women	SWAN	No	Yes	Yes	No	No
Travison 2011	Men	BACH	No	No	Yes	Yes	No
Troisi 2003	Women		Yes	No	Yes	Yes	No
Troisi 2008	Women		Yes	Yes	No	Yes	No
Trumble 2012	Men		No	Yes	No	No	No
Tsai 2006	Men		Yes	Yes	Yes	Yes	No
Ukkola 2001	Mixed		No	No	Yes	No	No
Vesper 2015	Mixed	NHANES	Yes	No	Yes	No	No
Vijayakumar 1995	Men		No	No	Yes	No	No
Wang 1991	Women		Yes	No	Yes	No	No
Wang 2004	Men		No	Yes	No	Yes	No
Weiss 2009	Women	SWAN	No	No	Yes	Yes	No
Welt 2006	Women		No	No	No	No	No
Wijeyaratne 2002	Women		Yes	No	No	No	No
Wijeyaratne 2004	Women		No	No	Yes	No	No
Williamson 2001	Women		No	No	Yes	No	No
Winkler 1991	Men		No	No	Yes	No	No
Winters 2001	Men		No	No	Yes	Yes	No
Wright 1995	Men		No	Yes	No	No	No
Wright 2002	Children		Yes	Yes	No	No	No
Wu 1995	Men		No	Yes	No	No	No
Xu 2014	Men	NHANES	No	Yes	No	No	No
Zagars 1998	Men		Yes	No	Yes	No	No

Zhang 2005	Women		Yes	Yes	No	Yes	Yes

Notes. BACH: Boston Area Community Health; DPP: Diabetes Prevention Plan; HUB: Hormones in Umbilical Cord Blood Study; MESA: Multi-Ethnic Study of Atherosclerosis; MrOS: Osteoporotic Fractures in Men Study; NHANES: National Health and Nutritional Examination Survey; SHLMAS: Sex Hormones and Lipoproteins in Adolescent Males Study; SWAN: Study of Women's Health Across the Nation; VES: Veterans' Experience Study

Appendix III: Summary Table of Data Used in Chapter 3					
Study	Incidence Disparities	Mortality Disparities	Surgery	Radiation	ADTs
Albano et al. (2007)	No	Yes	No	No	No
Amling et al. (2004)	No	No	No	No	No
Austin and Convery (1993)	No	Yes	No	No	No
Aziz et al. (1998)	No	Yes	No	No	No
Bach et al. (2002)	No	Yes	No	No	No
Ben-Schlomo et al. (2008)	Yes	Yes	No	No	No
Bernard et al. (2017)	No	Yes	No	No	No
Berry et al (1979)	No	Yes	No	No	No
Brawn et al (1993)	No	Yes	No	No	No
Cheng et al. (2009)	Yes	Yes	No	No	No
Chinegwundoh et al. (2006)	Yes	No	No	No	No
Chu et al. (2012)	No	Yes	No	No	No
Connell (2001)	No	Yes	No	Yes	Yes
Crawford et al. (1990)	No	Yes	No	No	No
Cross (2002)	No	Yes	No	No	No
Cullen et al. (2011)	No	Yes	Yes	Yes	Yes
Daskivich et al. (2015)	No	Yes	Yes	Yes	Yes
Dayal and Chiu (1982)	No	No	Yes	No	No
Dayal et al. (1985)	No	No	Yes	No	No
DeSantis et al. (2016)	Yes	Yes	Yes	No	No
Desch et al. (1996)	No	No	No	Yes	Yes
Du et al. (2006)	No	No	Yes	Yes	Yes
Du et al. (2011)	No	No	Yes	Yes	Yes
Du et al. (2012)	No	No	Yes	No	No
Eastham & Kattan (2000)	No	No	No	No	No
Evans et al. (2008)	No	No	Yes	No	No

Evans et al. (2010)	No	No	No	Yes	Yes
Faisal et al. (2014)	No	Yes	No	No	No
Fowler and Terrell (1996)	No	Yes	No	No	No
Fowler et al. (2000)	No	No	Yes	No	No
Freedland et al. (2002)	No	No	Yes	No	No
Freeman et al. (2003)	No	Yes	No	No	No
Freeman et al. (2004)	No	Yes	No	No	No
Godley et al. (2003)	No	Yes	Yes	Yes	No
Graham-Steed et al. (2013)	No	Yes	No	No	No
Grossfeld et al. (2002)	No	No	No	No	No
Halabi et al. (2006)	No	Yes	No	No	No
Hamilton et al. (2016)	No	No	Yes	Yes	Yes
Harlan et al. (1995)	No	No	Yes	Yes	No
Hart et al. (1999)	No	No	Yes	Yes	No
Hennis et al. (2011)	Yes	Yes	No	No	No
Heyns et al. (2011)	No	No	Yes	Yes	Yes
Hoffman et al. (2001)	No	No	Yes	Yes	No
Holmes et al. (2009)	No	Yes	Yes	Yes	Yes
Hussain et al. (1992)	No	Yes	No	No	No
Iselin et al. (1998)	No	Yes	No	No	No
Jack et al. (2009)	Yes	Yes	Yes	Yes	Yes
Jani (2005)	No	No	No	No	No
Johnstone et al. (2002)	No	No	No	No	No
Jones et al. (1995)	No	Yes	Yes	Yes	Yes
Kamari et al. (2007)	No	No	No	No	No
Khuntia (2004)	No	Yes	No	No	Yes
Kim et al. (1995)	No	Yes	No	Yes	No
Klanude et al. (1998)	No	No	Yes	Yes	No
Koscuiszka et al. (2012)	No	Yes	No	No	No

Kovi and Heshmat (1973)	Yes	No	No	No	No
Krieger et al. (2013)	No	Yes	No	No	No
Krupski et al. (2005)	No	No	Yes	Yes	No
Krongrad et al. (1996)	No	Yes	No	No	No
Kupelian et al. (2000)	No	No	No	No	No
Kupelian et al. (2002)	No	No	No	No	No
Lawton et al. (1994)	No	Yes	No	No	No
Lee et al. (2002)	No	No	No	Yes	No
Levine & Wilchinsky (1979)	No	Yes	No	No	No
Lloyd et al. (2015)	Yes	Yes	No	No	No
Mahal et al. (2014) CGC	No	Yes	Yes	Yes	No
Mahal et al. (2014) UO	No	Yes	Yes	Yes	Yes
Mahal et al. (2015)	No	No	Yes	Yes	Yes
McLeod et al. (1999)	No	Yes	No	No	No
Merrill and Lyon (2000)	No	Yes	No	No	No
Metcalfe et al. (2008)	No	No	No	No	No
Mettlin et al. (1997)	No	No	Yes	Yes	Yes
Moses et al. (2010)	No	No	Yes	Yes	Yes
Moses et al. (2017)	No	No	Yes	Yes	No
Moul et al. (1996)	No	No	No	No	No
Muralidhar et al. (2016)	No	No	No	No	Yes
Natarajan et al. (1989)	No	Yes	No	No	No
Nielson (2006)	No	No	No	No	No
Oakley-Girvan et al. (2003)	No	Yes	No	No	No
Oliver et al. (2006)	Yes	No	No	No	No
Optenberg et al. (1995)	No	Yes	Yes	Yes	Yes
Page and Kuntz (1980)	No	Yes	No	No	No
Peters and Armstrong (2005)	No	Yes	Yes	Yes	Yes

Polednak et al. (2003)	No	Yes	No	No	No
Powell et al. (1995)	No	Yes	No	No	No
Powell et al. (2004)	No	Yes	No	No	No
Pressley et al. (2013)	No	Yes	Yes	Yes	No
Quek et al. (2013)	No	No	No	No	Yes
Roach et al. (1992)	No	Yes	No	No	No
Roach et al. (2003)	No	Yes	No	No	No
Robbins et al. (1998)	No	Yes	No	No	No
Robbins et al. (2000)	No	Yes	No	Yes	Yes
Robbins et al. (2007)	No	Yes	Yes	Yes	Yes
Romero et al. (2012)	Yes	No	No	No	No
Saltzman et al. (2015)	No	No	No	No	No
Schmid et al. (2016)	No	Yes	Yes	Yes	No
Schreiber et al. (2013)	No	No	No	No	No
Schreiber et al. (2015)	No	No	No	No	No
Scwartz et al. (1996)	Yes	No	No	No	No
Scwartz et al. (2009)	No	Yes	No	No	No
Srindhar et al. (2009)	No	Yes	No	No	No
Stokes et al. (2013)	No	No	No	No	No
Strom et al. (2006)	No	No	No	No	No
Taksler et al. (2012)	Yes	Yes	Yes	Yes	Yes
Tarman et al. (2000)	No	No	No	No	No
Tewari et al. (2004)	No	Yes	No	No	No
Tewari et al. (2005)	No	Yes	No	No	No
Tewari et al. (2009)	No	Yes	Yes	Yes	No
Thatai (2004)	No	Yes	No	Yes	Yes
Thompson et al. (2001)	No	Yes	No	No	No
Underwood et al. (2005)	No	No	No	No	No
Verges et al. (2016)	No	No	No	No	No

Ward et al. (2004)	Yes	Yes	No	No	No
Williams et al. (2000)	No	Yes	No	No	No
Wu et al. (2014)	No	Yes	No	No	No
Wyatt et al. (2004)	No	Yes	No	No	No
Young et al. (2000)	No	No	No	Yes	Yes
Zagars et al. (1998)	No	Yes	No	No	No
Zeliadt et al. (2004)	No	No	Yes	Yes	Yes
Ziehr et al. (2015)	No	No	Yes	Yes	No

Appendix IV. Table of Clinical Guidelines Analyzed for Racially-Specific Claims		
Societies (n=10)	**Studies (n=70)**	**Racially-specific claims**
American Cancer Society		
	Mettlin et al. (1993)	Yes
	Von Eschernback et al. (1997)	Yes
	Smith et al. (2001)	Yes
	Smith et al. (2002)	Yes
	Smith et al. (2003)	Yes
	Smith et al. (2004)	Yes
	Smith et al. (2005)	Yes
	Smith et al. (2006)	Yes
	Smith et al. (2007)	Yes
	Smith et al. (2008)	Yes
	Smith et al. (2009)	Yes
	Wolf et al. (2010)	Yes
	Brooks et al. (2010)	Yes
	Smith et al. (2010)	Yes
	Smith et al. (2011)	Yes
	Smith et al. (2012)	Yes
	Smith et al. (2013)	Yes
	Skolarus et al. (2014)	Yes
	Smith et al. (2014)	Yes
	Smith et al. (2015)	Yes
	Smith et al. (2016)	Yes
	Smith et al. (2017)	Yes
American Society of Clinical Oncology		
	Loblaw et al. (2004)	No
	Loblaw et al. (2007)	No
	Kramer et al. (2009)	No

	Basch et al. (2014)	No
	Chen et al. (2014)	Yes
American Urological Association		
	Cookson et al. (2007)	No
	Carter et al. (2013)	Yes
British Society for Sexual Medicine		
	Hackett et al. (2008)	No
	Hackett et al. (2017)	Yes
Endocrine Society		
	Bhasin et al. (2006)	No
	Bhasin et al. (2010)	Yes
	Bhasin et al. (2018)	Yes
European Association of Urology		
	Aus et al. (2001)	No
	Aus et al. (2005)	No
	Heidenreich et al. (2008)	No
	Heidenreich et al. (2011a)	No
	Heidenreich et al. (2011b)	No
	Heidenreich et al. (2011c)	No
	Heidenreich et al. (2011d)	No
	Dohle et al. (2012)	No
	Heidenreich et al. (2014a)	No
	Heidenreich et al. (2014b)	No
	Mottet et al. (2014)	No
	Dohle et al. (2016)	No
	Mirone et al. (2017)	No
	Mottet et al. (2017)	Yes
	Cornford et al. (2017)	No
Joint Statements from European Association of Urology, International Society of		

Andrology, International Society for the Study of the Aging Male		
	Lunenfeld et al. (2005)	No
	Nieschlag et al. (2006)	No
	Wang et al. (2008)	Yes
	Wang et al. (2009)	Yes
	Lunenfeld et al. (2015)	Yes
National Comprehensive Cancer Society		
	Scherr et al. (2003)	No
	Mohler et al. (2007)	No
	Mohler et al. (2010)	No
	Kawachi et al. (2010)	Yes
	Mohler et al. (2012)	No
	Carroll et al. (2014)	Yes
	Mohler et al. (2014)	No
	Carroll et al. (2016)	Yes
	Mohler et al. (2016)	No
	Carroll et al. (2018)	Yes
U.S Preventive Task Force		
	O'Berg et al. (2002)	Yes
	Calonge et al. (2008)	Yes
	Chou et al. (2011)	No
	Moyer et al. (2012)	No
	Bibbins-Domingo et al. (2017)	Yes
	Grossman et al. (2018)	Yes

Appendix V. Properties of Dynamic Windows for Citation Network Data					
Calendar Year	Published in Calendar Year	Median Citation Width	Focal Period	Total N	Scaled Modularity
1980	59	4	1976-1980	701	0.279
1981	67	5	1976-1981	818	0.270
1982	68	5	1977-1982	908	0.277
1983	88	5	1978-1983	1358	0.254
1984	97	5	1979-1984	1299	0.264
1985	83	5	1980-1985	1083	0.239
1986	84	6	1980-1986	1283	0.244
1987	113	5	1982-1987	1412	0.207
1988	121	6	1982-1988	1606	0.224
1989	99	6	1983-1989	1179	0.264
1990	120	6	1984-1990	1688	0.265
1991	122	6	1985-1991	2174	0.253
1992	119	6	1986-1992	2075	0.251
1993	126	6	1987-1993	2181	0.232
1994	131	6	1988-1994	2640	0.241
1995	155	6	1989-1995	2920	0.225
1996	151	6	1990-1996	2861	0.221
1997	179	6	1991-1997	3494	0.235
1998	155	5	1993-1998	3009	0.236
1999	159	6	1993-1999	3120	0.250
2000	186	6	1994-2000	3855	0.232
2001	216	6	1995-2001	5951	0.214
2002	234	6	1996-2002	5542	0.211
2003	220	6	1997-2003	5214	0.215
2004	251	6	1998-2004	6289	0.201
2005	277	6	1999-2005	6151	0.214
2006	271	6	2000-2006	5814	0.217
2007	307	5	2002-2007	6233	0.202
2008	361	5	2003-2008	7758	0.168
2009	358	6	2003-2009	8789	0.181

2010	348	7	2003-2010	9569	0.188
2011	328	7	2004-2011	8280	0.184
2012	380	7	2005-2012	9023	0.190
2013	392	7	2006-2013	8020	0.197
2014	392	7	2007-2014	9760	0.175
2015	400	7	2008-2015	10080	0.180
2016	384	7	2009-2016	8670	0.197
2017	401	8	2009-2017	10077	0.198

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