Component Racialism and Race-Based Perceptions: The Roles of Racial and Biogeographical Ancestries

BY

DREXLER JAMES
B.Sc., Illinois Institute of Technology, 2013
M.A., University of Illinois at Chicago, 2016

THESIS

Submitted as partial fulfillment of the requirements
for the degree of Doctor of Philosophy in Psychology
in the Graduate College of the
University of Illinois at Chicago, 2018

Chicago, Illinois

Defense Committee:
Courtney Bonam, Chair and Advisor
Daniel Cervone
Sylvia Morelli
Tyrone Foreman, African American Studies
Brandon Hill, Planned Parenthood

DEDICATION

For My Mother, May I Inherit Half Her Strength

TABLE OF CONTENTS

CHAPTER PAGE

LIST OF TABLES

TABLE PAGE

I.

II.

III.

IV.

LIST OF FIGURES

FIGURE PAGE

SUMMARY

This research investigates a novel concept, *component racialism*: the belief that racial groups possess unique genetic configurations that determine within-racial group similarities and between-racial group differences in behavioral, biological, and physical characteristics and outcomes. In particular, this research uses biogeographical ancestry (i.e., the idea of that after migration populations developed geographic-specific biomarkers), through its misrepresentation as "race genes", to examine how component racialist ideologies exacerbate biased perceptions of racial groups and targets. In four studies, this research investigated the questions: (1) How does biogeographical ancestry information influence racial categorization, racial stereotyping, and perceived racial differences in illness/disease susceptibility? (2) To what extent do people misrepresent biogeographical ancestry as "race genes", and how does this misrepresentation explain perceptions of racial boundaries, racial differences, and illness/disease susceptibility? (3) To what extent is component racialism different from (or similar to) other forms of biological racial essentialism?

Results showed that people use racial ancestry information to make judgments about a target's biogeographical ancestry (Pilot Study). In addition, results show that people use biogeographical information to make race-based judgments about targets (Study 1 -2); relationships that are sometimes moderated by racial ancestry (Study 2). Furthermore, results show that people misrepresent biogeographical information as "genes genes", which they then use to make target judgments (Study 2). For example, increasing sub-Saharan Black biogeographical ancestry was related to increasing Black (decreasing White) racial categorization (Study 1 -2); increased beliefs that a target engaged in Black-associated cultural activities (Study 1 -2), increased beliefs that a target possessed Black-associated stereotypical

traits (e.g., decreased academic orientation; Study 1), and increased beliefs that a target was biologically different from White people (Study 2). Results also showed that increasing sub-Saharan Black racial ancestry was associated with increased beliefs that a target shared genetic material with Black people and that this increased belief was then associated with increased beliefs that a target (1) had a darker skin tone, (2) engaged in Black associated cultural practices, (3) was susceptible to physical illnesses, and (4) was susceptible to mental illnesses (Study 2). Last, results show that shifting people in component racialist views of race (vs. racialist and biological essentialist) more greatly influences the extent to which people believe that Black and White people are genetically different, which in turn lead them to increasingly believe that Black-White differences in illness/disease rates (e.g., STD/I), and Black-White biological, genetic, and socio-behavioral differences (e.g., education) are due to "natural" causes.

Examining these more nuanced beliefs about the genetic basis of race hold the potential to further understand theories of psychological essentialism and factors that reinforce already exaggerated views about innate racial differences that contribute to prejudice and stereotyping. Addressing such limitations are also practically and socially relevant given the increased use of and familiarity with personal genetic testing services that use patterns of racial/ethnic genetic variation to provide clues about biogeographical ancestry (e.g., 23&Me) and issues surrounding the use of biogeographical ancestry in understanding disease risk, progression, treatment, and susceptibility, especially among racial groups.

1. INTRODUCTION

For centuries people have used geographical ancestral origins to categorize others, and themselves, to racial groups (Plaks, Malahy, Sedlins & Shoda, 2012). For example, in *Systema Naturae* (1758, 10th edition) Carl Linneaus used geographical ancestral origins to subdivide humans into four racial groups: (*Europæus albus* [white European], *Americanus rubescens* [red American], *Asiaticus fuscus* [brown Asian] and *Africanus Niger* [black African]). Linneaus also believed race differences emerged because racial groups each possessed unique genetic compositions, which resulted from interactions with their situated geographical environments (Graves, 2015; Morning, 2011; Richeson & Sommers, 2016).

Although population-level genetic[1] analyses do not support such claims of genetically derived racial groups (e.g., Human Genome Project; Feldman, Lewontin, & King, 2003; Ossorio & Duster, 2005), in the 21st century, people continue to falsely believe that genetic variation, resulting from environmental influences, determines race and racial differences (Condit, Parrott, & Harris, 2002; Dar-Nimrod & Heine, 2011a; Jayaratne et al., 2006). Common misconceptions about the scientifically supported concept of *biogeographical ancestry* (i.e., the idea that after migration populations developed geographic-specific biomarkers; Bamshad, Wooding, Salisbury, & Stephens, 2004; Hadler, Shriver, Thomas, Fernandez, & Frudakis, 2008; Tishkoff

[1]The U.S National Institutes of Health (2018) defines "gene" as the "basic physical and functional unit of heredity [each individual has two copies of each gene, one inherited from each parent] …which are made up of DNA [deoxyribonucleic acid], [that] act as instructions to make molecules called protein." See Gerstein et al. (2007) for a more detailed definition of a "gene": "genomic sequence (DNA [deoxyribonucleic acid] or RNA [ribonucleic acid]) directly encoding functional product molecules, either RNA or protein" (p. 676).
Population-level genetic analysis examines genetic variation within populations, and involves the examination and modelling of changes gene frequencies in populations over space and time (Excoffier, Laval, & Schneider, 2005). Robust population-level genetic analyses show small genetic variation among human populations (e.g., among racial groups), and show greater genetic similarity between individuals from different populations relative to those from the same population (Witherspoon et al., 2007).

& Kidd, 2004) help perpetuate these false beliefs in racial genetic differences (Bolnick et al., 2007; Duster, 2006). Indeed, people often misinterpret the science of biogeographical ancestry as evidence that genes determine race and racial differences (Bolnick et al., 2007; Duster, 2006; Feldman, 2010; Koenig, 2010).

Empirical work (e.g., Lawton & Foeman, 2017; Morning, 2017; Nelson, 2008) has just begun investigating how people misrepresent biogeographical ancestry as *race genes* (i.e., genes specific to racial groups; e.g., "Black genes") and some consequences of these misrepresentations, such as in-group preference and out-group stereotyping (Keller, 2005; Schmalor, Cheung, & Heine, *unpublished*; as cited in Heine et al., 2017). Despite these emerging contributions, this area has yet to systematically establish that people misconstrue biogeographical ancestries (e.g., sub-Saharan African) as race genes that determine race and racial differences. Additionally, this area has yet to examine how such *biogeographically-based racial categorization*—and other false beliefs about race-specific biogeographical ancestries—may uniquely (beyond other ideas about racial determinism; e.g., biological, genetic) predict generalized stereotypes about and attitudes toward entire racial groups, and racial stereotype application and attitudes towards individual targets.

This potential form of racial genetic determinism represents a more nuanced, concrete conception of race, compared to conceptions of race as being biologically derived in a more general sense (e.g., Williams & Eberhardt, 2008). Examining these more nuanced beliefs can further elucidate theories of psychological essentialism and promote empirical investigations of other factors that reinforce views about innate racial differences. Examining this form of racial genetic determinism is also practically and socially relevant, given the increased use of, and familiarity with, personal genetic testing services that provide clues about biogeographical

ancestry (e.g., 23&Me; National Institutes of Health [NIH], 2017; Wolinsky, 2006); along with issues surrounding the use of biogeographical ancestry in understanding disease risk, progression, treatment, and susceptibility both within and between racial groups (Fujimura & Rajagopalan, 2010; Jorde & Wodding, 2004; Rotimi, 2004; Tishkoff & Kidd, 2004).

1.1 Psychological Essentialism

Psychological essentialism is defined as the attribution of a shared essence to members of the same (social) category (Rothbart & Taylor, 1992; Yzerbyt, Judd, & Corneille, 2004), along with the belief that essences constitute and differentiate among (social) categories (Soylu Yalcinkaya, Estrada-Villalta, & Adams, 2017). Psychological essentialism occurs when people believe that categories have an underlying, non-trivial, non-observable, fundamental nature that makes them what they are (Gelman, 2003; 2004; Medin & Ortony, 1989). Consequently, psychological essentialism is a process that facilitates the formation of categories (e.g., racial and gender categories; Dar-Nimrod & Heine, 2011a).

A number of characteristics describe people's beliefs about the nature of "essences". Specifically, people perceive essences to be (1) *immutable*: underlying essences do not change, even when observable traits are transformed (Gelman & Wellman, 1991; Keil, 1989; Rips, 1989); (2) *internal*: essences lie deep within, beyond the reach of external influences (Gelman & Welman, 1991; Rips, 1989); (3) *transferable*: essences can be transferred from individual to individual, while not changing (Gelman, Frazier, Noles, Manczak, & Stilwell, 2015; Heyman & Gelman, 2000); and (4) *category-boundary demarcating*: essences represent what members of a category share in common to differentiate them from members of other categories (Gelman, 2004; Heine et al., 2017; Medin & Ortony, 1989).

For the above reasons, essentialist thinking is associated with homogenizing and endorsing category-associated stereotypes about out-groups (e.g., Bastian & Haslam, 2006; 2007; Haslam, Rothschild, & Ernst, 2000), perceiving members of out-groups as less human (Leyens et al., 2001), and prejudice towards out-group members (Haslam, Rothschild, & Ernst, 2002). Psychological essentialism is also purported to be a general human tendency (Dar-Nimrod & Heine, 2011a; Haslam & Whelan, 2008; Heine, Dar-Nimrod, & Proulx, 2017; Norenzayan & Heine, 2005). However, although people have specific ideas about the qualities of essences, more often than not, their overall mental representations of essences remain low on the *abstract-concreteness continuum*[2] (i.e., they remain abstract in that they do not encompass delineated traits; Heine et al., 2017). As a result, to aid in concretizing essences, people often use an *essence placeholder* (i.e., "something [used] to stand in for [a] criterion" [Stevens, 2003, p. 2]), which leads to drawing greater casual inferences about group behavior and characteristics (Medin & Ortony, 1989).

1.1.1 Psychological essentialism forms

Past research has identified two common, more concrete, forms of psychological essentialism: cultural essentialism and biological essentialism. Cultural essentialism is the idea that "people are ... more or less passive carriers of their culture, whereby their attitudes, beliefs

[2] The abstractness-concreteness of a stimulus or broader category is mediated by its ability to stimulate imagery (Paivio, 1969). For this reason, the continuum is often thought of in terms of *cognitive complexity*; that is, the number of traits and qualities used to represent a category/stimulus (Hendrick, 1979; Johnson & Kisielius, 1985). The abstractness-concreteness continuum is also described in terms of *subordinate* and *superordinate* categories (Johnson & Kisielius, 1985). Superordinate categories (abstract; high generalities) represent broad classes of stimuli defined by category-wide attributes; such categories do not provide specific compositions, configurations, or properties. On the other hand, superordinate categories (concrete; low generalities) represent clearly identifiable, individuating, and specific compositions, configurations, or properties (Croft & Cruse, 2004; Johnson & Kisielius, 1985; Machunsky & Meiser, 2009). For example, "German Shepard" is a more concrete representation of the category "Animal", than "Invertebrate". Indeed, past research shows that concrete (vs. abstract) stimuli facilitate stronger mental representations (Smith, 1981; Wickens & Engle, 1970) and better memory for these stimuli (Hamilton & Rajaram, 2001).

and achievements are supposed to reflect typical cultural patterns" (Verkuyten, 2003, p. 385); for example, the belief that cultural characteristics (e.g., customary ways of thinking and feeling, language) explain why groups are the "way they are" (Buhagiar, Sammut, Rochira, & Salvatore, 2018). On the other hand, biological essentialism is the belief that social categories have natural constitutions (Bastian & Haslam, 2006); for example, the belief that social groups are determined by their respective biological makeup (e.g., genes). In both forms of psychological essentialism are the beliefs that social groups are "determined by a fixed and uniform essence that resides within and defines all [group] members" (Soylu Yalcinkaya et al., 2017, p. 2). Indeed, previous research shows that the two forms of essentialism are positively correlated, are associated with out-group prejudice, and that people often use both forms to define social groups (e.g., racial groups; Morning, 2009; Soylu Yalcinkaya et al., 2017; Zeromskyte & Wagner, 2017).

1.2 Biological Essentialism

Research suggests that people use two distinct essence placeholders to explain the biological nature of social categories (i.e., distinct forms of biological essentialism): neuroessentialism and genetic essentialism. Neuroessentialism is the "belief that brains and their abnormalities define and determine identity" (Haslam, 2011, p. 820). In this type of biological essentialism, people use non-genetic, biological attributions (e.g., hormonal imbalance or brain processes), as essence placeholders (Dar-Nimrod & Heine, 2011b). On the other hand, with genetic essentialism, people use genes as an essence placeholder to explain the biological nature of social categories (Dar-Nimrod & Heine, 2011a). Although both forms of biological essentialism have negative consequences, the extent to which they prompt determinist judgments differ (Dar-Nimrod & Heine, 2011b; Haslam, 2011). For example, Shiloh, Rashuk-Rosenthal, and Benyamini (2002) showed that people judged illnesses as less controllable when the cause of

the illnesses was genetic compared to biological (other non-genetic) factors. As a result, even though the two forms of biological essentialism are correlated, their "magnitude of essentialist bias" differs. Dar-Nimrod & Heine (2011b) suggest this difference in essentialist bias is perhaps because genetic essentialism, relative to neuroessentialism, is a more concrete (less abstract) form of biological essentialism, as genes "have been perceived as puppet masters…[and] because neurological attributions occupy an intermediate level of causation..." (p. 830).

1.2.1 Genetic essentialism

Like essences, people often believe genes to be internal (Heine et al., 2017), transferable (Gil-White, 2001), stable (Chandler & Proulx, 2008), and natural (Dar-Nimrod & Heine, 2011a; Jayaratne et al., 2009). As a result, genes are one common essence placeholder, as genes and essences share similar features (Dar-Nimrod & Heine, 2011a; Heine, 2017 as cited in Heine et al., 2017). These beliefs have consequences for how people perceive, evaluate, and treat groups, and group members (Brescoll & LaFrance, 2004; Horvath & Ryan, 2003; Jayaratne et al., 2006; Teachman, Gapinski, Brownell, Rawlins, & Jeyaram, 2003). For example, Brescoll and LaFrance (2004) have shown that people who read an article claiming that genetic (vs. socio-cultural) factors lead to observed differences in plant-identifying abilities between men and women endorsed gender stereotypes to a greater extent. Similarly, other work by Horvath and Ryan (2003) shows that genetic (vs. choice) beliefs about homosexuality are associated with decreased positive attitudes toward lesbians and gay men (c.f. Jayaratne et al., 2006).

In addition, genetic essentialist beliefs also influence how people perceive illnesses and those who suffer from them. For example, people making stronger genetic (vs. environmental) attributions for schizophrenia desire more social distance from sufferers (Angermeyer & Matschinger, 2004), perceive sufferers as less active agents in the disease's onset (Phelan, Cruz-

Rojas, & Reiff, 2002), see the illness as more serious (Phelan, 2005), and are more likely to believe that a sufferer's siblings and children will develop schizophrenia (Angermeyer & Matschinger, 2004). Similarly, people who imagine that they are at increased risk for arthritis or heart disease because of their genetic predisposition (vs. an unspecified factor) are less likely to believe that the illness is preventable, are less likely to attribute the onset of the disease to diet or lifestyle choices, and are more likely to believe that genes are important to the disease's onset (Senior, Marteau, & Weinman, 2000). Last, genetic essentialist beliefs also affect individual behavior.

1.3 Racialism

Related work also shows that *racialism* (i.e., the belief that genes determine race; genetic essentialist beliefs about race, in particular) heightens perceptions of racial group differences, racial stereotyping, and prejudice (Smedley & Smedley, 2012). For example, Jayaratne and colleagues (2006) find that a representative sample of White Americans espouse genetic explanations for racial differences in math ability, drive to succeed, violent tendencies, and intelligence. These beliefs are positively associated with traditional anti-Black prejudice (e.g., negative attitudes toward a son or daughter dating a Black person) and modern racism (e.g., a belief that Blacks have only themselves to blame for not doing well), even after controlling for common predictors of prejudice such as age, education, and political orientation (see also Jayaratne et al., 2009).

Similarly, Condit, Parrott, Bates, Devan, and Achter (2004a) find that White people, who are exposed to messages about genes as the cause of heart disease among Black or White people, show increased levels of modern racism and genetically-based racism, compared to those receiving universal (i.e., non-race specified) messages or a no-messages control group. More

specifically, for example, those receiving messages that genes cause heart disease among Black people (vs. control) are more likely to demonstrate both modern and genetically-based racism[3]. Likewise, Williams & Eberhardt (2008) find that people exposed to a news article arguing for a genetic (vs. social) basis of racial categories report less concern and negative emotion after reading about racial inequities, and less willingness to seek friendship with other-race (vs. same race) targets. Moreover, Williams & Eberhardt (2008) show that, although people's race conceptions vary along a continuum from thinking about race as biologically derived and fixed (a broader conception which encompasses more specific beliefs about the genetic basis of race) to socially derived and flexible, people more often espouse a biological race conception. This pattern means that, even unprompted by researchers, most people operate within a fixed, biological mindset regarding race, similar to those people assigned to read the news article arguing for a genetic basis for race.

1.3.1 Component racialism

Additional anecdotal and historical evidence suggests that people hold even more nuanced beliefs about race and genes—beyond the basic idea that race is genetically derived. For example, Jean-Phillipe Rushton, a prominent psychologist, argues that through genetic inheritance White people possess all socially desirable personality and intellectual traits, while Black people possess all antisocial and undesirable traits (Rushton & Jensen, 2005; 2010). Likewise, in their controversial book, *The Bell Curve*, Herrnstein and Murray (1994) contend that European Americans are intellectually superior to African Americans because they inherit distinct intellect-related genes; a belief also shared by James Watson, winner of a Nobel Prize for the discovery of the double helix structure of DNA (Nugent, 2007; see also Sternberg,

[3] For example, a greater endorsement statements like, "members of one racial group are more musical than members of another racial group because of genetics", and "Racial differences in academic ability are caused by genetics."

Grigorenko, & Kidd, 2005; c.f. *The Mismeasure of Man* for a refutation of these arguments, Gould, 1996). Others (e.g., Edwards, 1972; Entine, 2000; Hoberman, 1997) have also claimed that, due to natural selection, Black people (relative to other groups) possess more genes that determine athletic prowess and physical superiority.

Recent quantitative evidence reveals that such beliefs are not isolated and are commonplace, at least in the United States. In particular, Condit and colleagues (2004b) estimate that a majority of U.S Americans believe that "genetics play a primary role in determining an individual's race". They also find that people believe racial genetic differences—especially those that they believe determine physical characteristics (e.g., skin color, facial features, blood type, body structure)—are responsible for some racial disparities. These disparities include physical abilities, and illness rates and susceptibility, but not racial differences in non-physical attributes (e.g., personality, behavior). These findings suggest that people often believe that a subset of genes (i.e., genes that determine physical characteristics) determine similarities among racial group members and differences between racial groups.

Collectively, this evidence reinforces claims that people hold genetic determinist beliefs about race (i.e., that people engage in racialism). Critically, going beyond this more basic assertion, this evidence also begins to suggest that people believe in complex genetic distinctions between racial groups—distinctions that they believe (1) have developed through natural selection and genetic inheritance patterns, and (2) are the root cause of perceived racial differences. This belief that racial groups possess unique genetic profiles (or compositions) is what I term *component racialism*. More precisely, *component racialist beliefs* assume that racial groups possess unique genetic configurations that explain the (perceived or actual) within-racial group similarities (i.e., shared behavioral and physical characteristics among members of the

same racial group) and between-racial group differences (i.e., disparate behavioral and physical characteristics between racial groups). Recent work has recently begun to investigate how ideas about racial genetic variations can influence perception of racial groups and targets.

For example, Kang and colleagues (2015) show that believing there is greater genetic difference between White and Black people (i.e., less genetic similarity; low genetic overlap) predicts stronger neural avoidance responses (i.e., greater right versus left frontal lobe activation) to Black/White biracial compared to Black and White mono-racial targets. Similarly, Plaks and colleagues (2012) find that, as endorsement of the belief that there are genetic differences between Black and White people increases, so too does people's use of physical racial cues (e.g., skin tone) to categorize Black and White targets. Additionally, people led to believe that there is greater (vs. less) genetic difference between Black and White people (i.e., those induced to hold component racialist beliefs) rate prototypically Black targets more negatively; this effect remains significant even when controlling for global belief in genetic determinism (Kang et al., 2015; Plaks et al., 2012). Thus, component racialist ideology distinctively predicts perceptions of racial out-group members. In a similar investigation, Kimel and colleagues (2016) show that Jewish people led to believe that Arabs are genetically dissimilar (vs. similar) to Jewish people are more likely to be physically aggressive toward an ostensibly Arab target (i.e., they direct higher noise levels at the target).

Together, these studies begin to suggest that, beyond more general genetic determinist beliefs, the belief that racial groups possess unique genetic compositions (i.e., component racialism) influences the extent to which people: (1) view racial groups as distinct from each other (e.g., racial categories become more homogeneous and discrete); (2) hold negative attitudes (e.g., prejudice) toward racial out-groups; (3) stereotype racial out-groups; (4) react negatively to

racial out-group members (e.g., neural avoidance); and (5) are physically aggressive toward racial out-group members. Therefore, exposing people to component racialist ideology may further concretize mental representations of racial genetic essences, in a way that more strongly predicts beliefs about both racial differences and the very basis of racial categories (Dar-Nimrod & Heine, 2011b; Heine et al., 2017; Simons & Keil, 1995).

Although these studies have begun investigating the consequences of component racialist ideologies, they do so without fully and directly examining how people mentally represent racial genetic differences, along with how these mental representations predict racial categorization and race-based perceptions: What do people believe creates the genetic variation that they believe defines racial group boundaries and characteristics? How do these beliefs, along with other beliefs about genetic variation and race, influence assumptions about racial disparities? I contend, based on other previous research, that representations of biogeographical ancestry function as one concrete genetic form of a racial essence placeholder that shapes people's beliefs about genetics as a basis for race and perceived racial group differences. I also argue that biogeographical ancestry represents a more extreme form of racial genetic essentialism (i.e., component racialism) that explains racial stereotyping and group attitudes beyond more general genetic, and even more general biological, essentialist beliefs about race (see Figure 1).

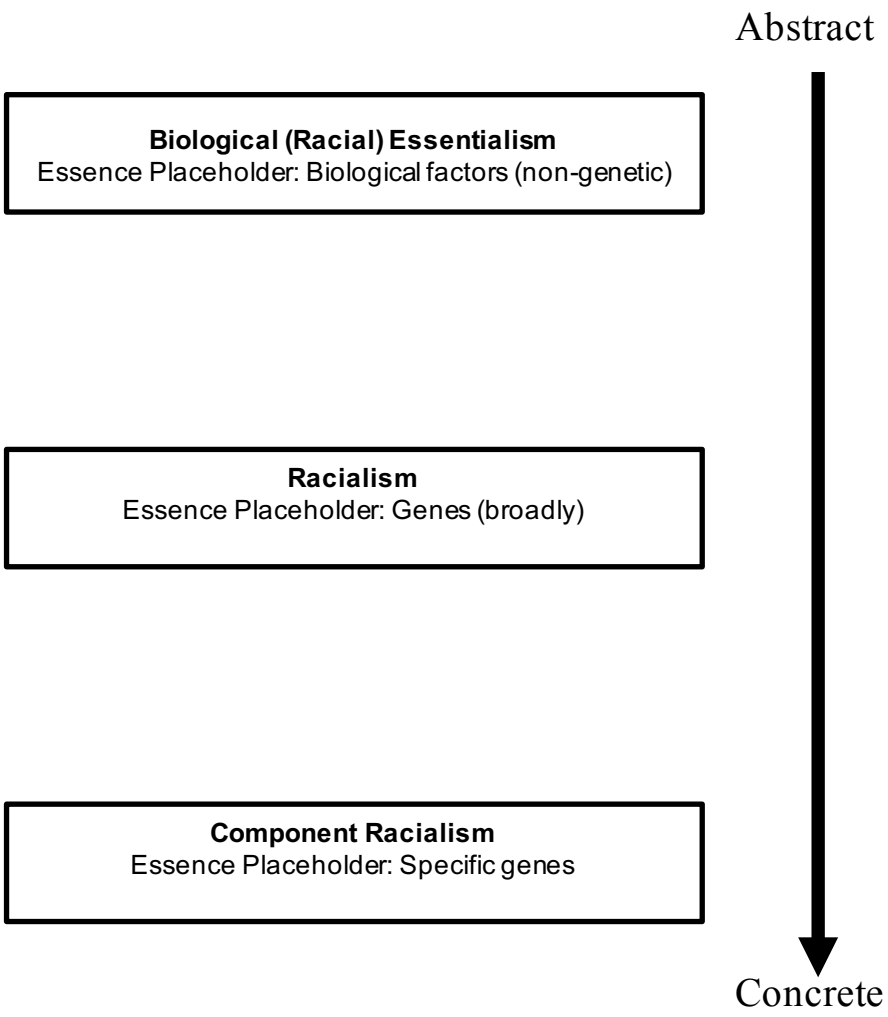


Figure 1. Representation of biological race conceptions along an abstract-concrete continuum.

1.4 Biogeographical Ancestry

In the U.S, the structures used to racially classify people have shifted considerably. For example, people were—and continuingly are—racially categorized in accordance with their racial ancestry (i.e., origin or descent, the person's parents' racial category, or their ancestors before their arrival in the United States; Hirschman, Alba, & Farley, 2000). With the advent of genetic science in the late 19th century and into the 20th century, gene-based evolutionary theories of race, specifically the "discovery" of human genetic variation, shifted racial classification toward biological domains (Smedley & Smedley, 2005; Sternberg, Grigorenko, & Kidd, 2005;

Wang & Sue, 2005). For example, the "one-drop rule" or "hypodescent" enforced the idea that one drop of African blood, or any amount of African ancestry, meant that an individual was Black (Genetic Working Group, 2005; Guo et al., 2014). Morphological features (e.g., skull volume and size, skin color, facial features, and other visible qualities) were also used to racially classify individuals (Smedley & Smedley, 2005; Wang & Sue, 2005). Presently, ideas surrounding biogeographical ancestry, propelled by genetic ancestry tests, have further reinforced these gene-based evolutionary theories of race (Genetic Working Group, 2005; Guo et al., 2014; Smedley & Smedley, 2005; Sternberg, Grigorenko, & Kidd, 2005; Wang & Sue,).

Biogeographical ancestry describes an individual's ancestral origin (line of descent) in relation to major population groups (e.g., Native American, East Asian, European, sub-Saharan African; Halder, Shriver, Thomas, Fernandez, & Frudakis, 2008). It is based on the idea that, after humans migrated out of Africa, these numerous populations developed distinct genetic markers—as a result of isolation and restricted population mating until the 15th century[4]—that vary across the geographical areas in which they settled (Bamshad et al., 2004; Hadler et al., 2008; Tishkoff & Kidd, 2004). An individual's biogeographical ancestry is estimated with genetic ancestry tests that use panels of Ancestry Informative Markers (AIMs). AIMs are a subset of genetic markers with different allele[5] frequencies across geographic populations, and they have been characterized in a number of well-defined population samples (e.g., European; Kosoy, 2009).

[4]During and post-15th century, several factors (e.g., mass movement of people across isolated land masses via ships and airplanes, colonization, and the African slave trade) decreased geographic and sociocultural barriers among previously isolated populations. This increased movement of people facilitated interethnic mating, resulting in greater population admixture (i.e., the introduction of new genetic material into a population as a result of interbreeding between two or more previously isolated populations (Bamshad et al., 2004; Gannett, 2014; Tishkoff & Kidd, 2004).

[5]Alleles are different forms of the same gene (NIH, 2018).

Using an individual's DNA, after AIMs analyses are conducted, the likelihood (or probability) that the individual has ancestors from one or more parental populations is calculated. Parental ancestry calculations take the form of a series of percentages that add to 100%, which can include a single population or a combination of populations. Parental populations with the highest likelihood are then taken to represent the best estimate of the individual's ancestral proportions (Elhaik et al., 2014; Halder et al., 2008)). Thus, using this method, biogeographical ancestry can provide information about an individual's likely geographic ancestral origin. For example, Rosenberg et al., (2002), with a sample of about 1000 participants from 52 ethnic groups distributed worldwide, allocated participants into 1 of 6 different genetic clusters using Analysis of Molecular Variance. Five of these genetic clusters corresponded to major geographic regions: sub-Saharan Africans; Europeans and Asians west of the Himalayas; East Asians; inhabitants of New Guinea and Melanesia; and Native Americans (see also Bowcock et al., 1991; Stephens et al., 2001). Others have also demonstrated correlations between geographic distribution of genes and traditional racial categories (Jorde & Wodding, 2004). For example, Bamshad, and colleagues (2003) assigned 206 individuals in 20 ethnic groups from sub-Saharan Africa, East Asia, and Europe to their corresponding major continent using genetic analysis with a mean accuracy of at least 90%. Likewise, Shriver and colleagues (2003) were able to approximate an individual's ethnic ancestry using DNA analysis and a number of well-defined genetic markers (see also Bamshad, et al., 2003; Risch, Burchard, Ziv, & Tang, 2002).

However, AIMs do not carry racial essences, and, as a result, do not provide evidence for genetically-defined, discrete, racial groups (Haga & Venter, 2003; Ossorio & Duster, 2005). Indeed, genetic research shows that two randomly selected humans share approximately 99.9% of their genetics (Bonham, Warshauer-Baker, & Collins, 2005), and that only 11%-23% of

observed genetic variations are due to genetic differences among populations (for review, see Tishkoff & Kidd, 2004)—where most variation exists *within* relative to *between* social groups (e.g., race; Sternberg, Grigorenko, & Kidd, 2005). Collectively, genetic data hold that races are not genetically distinct human populations (Bonham et al., 2005). However, in spite of this evidence, genetic ancestry testing companies purport to be able to use AIMs to reveal an individual's genetic ties to racial groups (Bolnick et al., 2007; Panofsky & Donovan, 2017)—at least one genetic ancestry testing company claims to have "race-determining genetic markers" (Ossorio & Duster, 2005). In addition, these companies tend to suggest that these ancestral populations are "distinctive" (Bolnick et al., 2007; Panofsky & Donovan, 2017; Royal et al., 2010; Scodari, 2017). A number of scholars have suggested these companies' misrepresentation of genetic ancestry science are products of their non-adherence to scientific evidence on, and standards related to, genetic testing, along with the "questionable" scientific assumptions that guide those tests (Bolnick et al., 2007; Fullwiley, 2014).

Despite these testing issues, Americans are very receptive to genetic ancestry tests that purport to provide biogeographical ancestry information. For example, in a nationally representative survey, 63% of Americans were "strongly" or "somewhat" favorable to the use of DNA to research one's ancestry, compared to 9% who were "unfavorable" (Hochschild & Sen, 2011). However, Duster (2006), argues that the rise of genetic testing, and its favorability among the general population, reinforces genetic determinist beliefs about race and racial differences (Bolnick et al., 2007). Similarly, scholars have argued that genetic ancestry tests provide a mechanism for racism and racialization by reviving ideas of racial categories as proxies for biological differences (Hirschman & Panther-Yates, 2008; Scodari, 2017). On the other hand, others (e.g., Hochschild, Weaver, & Burch, 2012) have argued that increased familiarity and use

of genetic tests lead to more fluid ideas about race that challenge racial assumptions (i.e., "race as social"). Not supporting this position, however, empirical work suggests that exposure to genetic ancestry tests and information *exacerbates* beliefs about racial genetic determinism (Phelan, Link, Zeiner, & Yang, 2014), perhaps because geographic sub-divisions are misrepresented as racial categories (Greely, 2008).

1.4.1 Biogeographical ancestry information influences (racial) group perception

Some qualitative work shows that people, even when not prompted with a definition or representation of biogeographical ancestry, refer to genes and geography to explain race and racial differences (Condit et al., 2004b; Dubriwny, Bates & Bevan, 2004). For example, Condit and colleagues (2004b) find that, in addition to believing that physical attributes (e.g., skin tone) determine race, people believe geography determines race, primarily because racial groups have historically occupied separate geographic regions (e.g., "Black people" with sub-Saharan Africa, "White people" with Europe). Dubriwny and colleagues (2004) also show that people make connections between characteristics often used to determine racial category (e.g., skin color, hair texture) and geography. Both studies demonstrate that people believe geography determines racial differences because it affects genetic outcomes. In particular, they suggest that people believe geography influences genes, which then affects physical and behavioral characteristics. In addition, these studies show that people use geographic origin to explain visible racial differences, and see geography, through its influence on genes, as part of race. Moreover, they indirectly show that people have access to and offer biogeographical ancestry as an explanation for race and racial differences and believe that "race genes" exist; a perception that is exaggerated as contemporary and historical racial taxonomies correlate strongly with geographic regions.

Only two studies—to my knowledge—have directly investigated how such beliefs influence people's view of racial/ethnic groups. In the first study, German students who read an essay about the geography of human genetic diversity showed stronger in-group bias (i.e., greater liking of Western Europeans than Eastern Europeans) compared to those who read a "neutral" essay; this relationship was not moderated by general genetic determinist beliefs (Keller, 2005). In the second study, American adults who read an essay about a geographic distribution of genes were more likely to believe that ethnic stereotypes result from group genetic differences (e.g., French have a more sophisticated palate) compared to those who read an essay describing human genetic homogeneity, or a control topic (Schmalor, Cheung, & Heine, *unpublished*; as cited in Heine et al., 2017). These two studies show that priming beliefs about biogeographical ancestry influences the extent to which people like in-group and out-group members and the extent to which they assume genes are responsible for the veracity of racial/ethnic stereotypes.

These studies also indirectly suggest that assumptions about the geographic distribution of genes may lead people to conclude that groups (i.e., racial, ethnic, national) have unique genetic compositions. However, while these studies investigate the consequences of biogeographical ancestry, they do so for human geographic diversity broadly. They do not investigate the beliefs—or content—associated with each specific locale. It is still unclear, for example, in Keller (2005) how beliefs about Western and Eastern European geographic regions may have separately influenced perceptions of Western and Eastern European people. To address this gap in the literature, the present studies examine whether people use biogeographic ancestries (e.g., sub-Saharan, European) when racially categorizing and making race-based judgments (e.g., stereotyping) about singular targets and racial groups as a whole. I also test my contention that the relationship between biogeographical information and these outcomes is

driven by people's misinterpretation of biogeographical ancestry information as "race genes" (e.g., sub-Saharan African biogeographical ancestry as "Black genes").

1.4.2 Inferring racial identity and racial category from biogeographical ancestry information

According to Hirschman & Panther-Yates (2008), biogeographical ancestry information from DNA testing results can "reshape how individuals think of themselves and their ethnic heritage…and bind us to specific groups of people." (p. 64). As a result, DNA testing can influence perceptions of racial boundaries, racial identification, and racial categorization (Hochschild & Sen, 2011). Previous work shows that people (e.g., African Americans) use the biogeographical information from ancestry tests to form ideas about their own racial identities and group affiliations (Lawton & Foeman, 2017; Morning, 2017; Nelson, 2008; see also Nelson, 2016). For example, Roth and Lyon (2018), found that 73.9% of about 600 individuals who took genetic ancestry tests reported that the results impacted their racial or ethnic identity, in that they later racially/ethnically identified differently and sought to affiliate with new and different racial/ethnic groups based on their reported biogeographical ancestry (as cited in Heine, Dar-Nimrod, & Proulx, 2017; and Morning, 2017).

Although no empirical work has examined how exposure to biogeographical information influences how people racially categorize others, some anecdotal evidence points to an existing relationship. For example, Cleon Brown, a 47-year-old police sergeant, believing he was of European and American Indian ancestry, took a genetic genealogy test, which revealed that he had "18 percent sub-Saharan African" ancestry. Upon sharing the results with his all-White co-workers, Sgt. Brown reported instances of racism such as being called "Kunté" and "Negroid" (Eligon, 2017). This example highlights how, in addition to shaping an individual's idea of their

own racial identity and group membership, biogeographical ancestry information can shape how people perceive and racially categorize others.

1.4.3 Inferring illness susceptibility from biogeographical ancestry information

In addition to providing biogeographical ancestry information, some genetic ancestry testing companies provide health information, and information about an individual's susceptibility to illness (Donovan, Pasquetto, & Pierre, 2018). For example, in 2017 and 2018, the U.S. Food and Drug Administration (FDA) granted 23&Me, a direct-to-consumer testing company, authorization to provide information on an individual's genetic predisposition to ten illnesses (e.g., Celiac Disease, Parkinson's Disease; FDA, 2017), and breast cancer (FDA, 2018). However, according to Jeffrey Shuren, M.D., director of the FDA's Center for Devices and Radiological Health, "[While] Consumers can now have direct access to certain genetic risk information…it is important that people understand that genetic risk is just one piece of the bigger puzzle, it does not mean they will or won't ultimately develop a disease." (FDA, 2017). Nevertheless, on the 23&Me website (23andme.com), along with cautionary notes about the limits of the tests for informing medical decisions[6] is a list of illnesses paired with biogeographical ancestries (e.g., Celiac Disease, "relevant for European descent"; Parkinson's Disease, "relevant for European, Ashkenazi Jewish, North African Berber descent). This pairing may lead people to associate illnesses with biogeographical ancestries.

Although racial and ethnic groups in the U.S often do show different rates in disease incidence, severity, progression, and response to treatment (Genetic Working Group, 2005), genetic research suggests that this relationship is not driven exclusively, or largely, by genetic

[6] For example, "Each genetic health risk report describes if a person has variants associated with a higher risk of developing a disease, but does not describe a person's overall risk of developing the disease. These reports are not intended to tell you anything about your current state of health, or to be used to make medical decisions…"

differences. Instead, research suggests that the differences in illness rates across racial groups are driven by social factors such as socioeconomic status, neighborhood environment, and lack of access to health care and discrimination (Cooper, Kaufman, & Ward, 2003; Genetic Working Group, 2005; Mersha & Abebe, 2015). For example, among African Americans, self-reported African-American race, but not sub-Saharan African biogeographical ancestry, was associated with increased risk for kidney disease (Peralta et al., 2006). Despite this and other mounting evidence, both medical health professionals and lay people still believe that biological differences explain racial differences in susceptibility to and manifestations of illness and disease (Institute of Medicine, 2003; Shields et al., 2005).

For example, Hoffman, Trawalter, Axt, and Oliver (2016), found that White lay people, and medical experts endorsed false beliefs that Black people are biologically different from White people (e.g., "Whites have larger brains than Blacks", "Blacks' nerve endings are less sensitive than Whites'"). They then show, among medical experts, that endorsing these false beliefs predicts (1) rating a Black (vs. White) patient's pain as lower, and (2) less accurate treatment recommendations for a Black (vs. White) patient. Although this study does not directly test biogeographical ancestry's influence on perceptions of one's susceptibility to illness/disease, it more broadly shows that endorsing beliefs about racial biological difference influences health-related judgments about targets. Hence it is conceivable that misrepresenting biogeographical ancestry as "race genes" is another—more concrete—mechanism driving perceived associations between race and illness/disease susceptibility.

Nevertheless, there is some evidence to suggest that racial genetic differences are related to health disparities and racial susceptibility to illness/disease (Genetic Working Group, 2005; Rowe & Rodgers, 2005). For example, Reiner et al. (2007) found that among self-identified

African Americans, increased sub-Saharan African biogeographical ancestry was associated with increased risk for insulin resistance. However, the authors caution that the relationship between sub-Saharan biogeographical ancestry and insulin resistance might likely "be influenced by both genetic and environmental factors…[and] it is important to recognize the correlation between cultural and biologic inheritance that exists among human populations…Therefore, formal proof that any observed trait-ancestry association is indeed "genetic" will require identification of a specific genomic locus that accounts for the association." (p. 575). Accordingly, Reiner et al. (2007) argues that statistical relationships between biogeographical ancestry and health-related outcomes are not enough to claim that biogeographical ancestry causes illness/disease.

Similarly, other scholars have argued that a hyper-focus on genetic causes of racial health disparities may lead people to overgeneralize tentative associations between illness and biogeographical ancestry (Sankar et al., 2004; Shield et al., 2005; Whitfield & McClearn, 2005). Although previous research has investigated the relationship between biogeographical ancestry and illness/disease susceptibility, no work, to my knowledge, has investigated how this information influences perceptions of illness/disease susceptibility. Studying perception of illness/disease susceptibility is important given its relationship to personal health behavior (e.g., vaccination; Brewer et al., 2007; cancer screening; Kim et al., 2008), behaviors towards others (e.g., hand shaking; Brug et al., 2009), and its potential to perpetuate racial disparities in healthcare quality (Dotson, Bonam, & Jagers, 2017; Fiscella, Franks, Gold, & Clancy, 2000; Nelson, 2002).

1.4.4 Biogeographical ancestry as a contemporary form of racial formation

Debates about biogeographical ancestry's utility in racial classification and for studying illness continues (Koenig, 2010). For example, on the one hand Bamshad and colleagues (2004)

claim that race is not a meaningful descriptor of biogeographical ancestry, while, on the other hand, Rosenberg and colleagues (2002) suggest that race provides a "suitable proxy" for biogeographical ancestry. At the same time other scholars (e.g., Fujimura & Rajagopalan, 2010; Jorde & Wodding, 2004; Rotimi, 2004; Tishkoff & Kidd, 2004) maintain that biogeographical ancestry can be useful in understanding disease risk, progression, treatment, and susceptibility, especially within and between racial groups (Epstein, 2006).

Nonetheless, Bamshad and colleagues (2004) and Feldman (2010) caution that the demonstrated relationship between race/ethnic identification and biogeographical ancestry, especially when misinterpreted, can have negative consequences for how people perceive racial groups. In particular, Feldman (2010) maintains that while "only a small fraction of genomic variation is responsible for visible morphological [race] differences such as skin color, facial features, or hair form that are commonly used to assign people to different races… [this variation] has nothing to do with supposed racial differences related to intelligence, moral character, or tendency for criminality" (p., 157). Moreover, they argue that factors such as media misrepresentation of genomic research, and limited general understanding of genetics and biogeographical ancestry, perpetuate malformed beliefs about race, genes, and racial genetic variation—specifically the belief that biogeographical ancestry represents genes that necessarily determine race and racial differences (Feldman, 2010).

Similarly, Bamshad and colleagues (2004) contend that espousing these beliefs can reinforce already exaggerated beliefs about innate differences between racial groups and the idea that racial groups each possess unique genetic compositions (see also Koenig, 2010). Thus, these misrepresentations often lead people to view "biogeographical ancestry" and "race genes" as synonymous, even though only the former concept is empirically supported (Bamshad, et al.,

2004; Feldman, 2010; Hadler et al., 2008; Koenig, 2010; Risch et al., 2002; Rosenberg et al., 2002; Tishkoff & Kidd, 2004). Thus, consistent with these past positions, I argue that biogeographical ancestry—through genetic ancestry tests—represents a contemporary form of racial formation—"the socio-historical process by which racial categories are created, inhabited, transformed, and destroyed" (Omi & Winant, 1994, p. 55-56)—and racialization—"processes by which ideas about race are constructed, come to be regarded as meaningful, and are acted upon" (Murji & Solomos, 2005, p. 1), that shifts discussions and understandings of race and genetics. In particular, I contend that not only do people misrepresent geographic sub-divisions reported in genetic ancestry tests as racial categories, they also misrepresent biogeographical ancestry as evidence for racial genetics (or race genes) that demarcate racial boundaries and define racial differences, while also presuming that racial ancestry and biogeographical ancestry are synonymous.

2. CURRENT STUDIES

This current research investigates three primary questions: (1) How does biogeographical ancestry information influence racial categorization, racial stereotyping, and perceived racial differences in illness/disease susceptibility? (2) To what extent do people misrepresent biogeographical ancestry as "race genes", and how does this misrepresentation explain perceptions of racial boundaries, racial differences, and illness/disease susceptibility? (3) To what extent is component racialism different from (or similar to) other forms of biological race conceptions (e.g., racialism)? I investigate these questions in a pilot study and three main studies.

I first examine the relationship between racial ancestry, biogeographical ancestry, and racial categorization in a Pilot Study. With the Study 1, I investigate if, and the extent to which, lay people use biogeographical ancestry to racially categorize and make race-based judgments about a target's biological/physiological and personality attributes (e.g., racial stereotyping). With Study 2, in addition to investigating the relationship between biogeographical ancestry and racial categorization and race-based judgments, I investigate the relationship between biogeographical ancestry and perceptions of a target's susceptibility to illnesses/disease (e.g., HIV/AIDS). With this study, I also examine the extent to which biogeographical ancestry is misrepresented as "race genes", along with the moderating role of racial ancestry on these relationships. Last, with Study 3, I investigate component racialism as a distinct and concrete form of biological racial conception that more strongly influences perception of racial groups compared to racialism and biological racial essentialism. With study 3, I focus on how the abstractness/concreteness of biological race conceptions influences perceptions of racial illness/disease susceptibility and other race-related differences.

For pragmatic reasons, I start to investigate these questions among White participants, with Black//White multiracial targets (i.e., a target with both White and Black racial ancestry), and with a focus on sub-Saharan African and European biogeographical ancestries. Specifically, I use White participants because they have been shown to use genetic information to make target judgments (Kang et al., 2015; Plaks et al., 2012), and because they tend to show greater levels of pro-White/anti-Black bias than non-Whites (Bar-Anan & Nosek, 2014). I focus on sub-Saharan African (i.e., "Black") and European (i.e., "White") biogeographical ancestries due to historical portrayals of "Black genes" as deficient and inferior relative to "White genes" (e.g., Condit et al., 2004a; Condit et al., 2004b; Jayaratne et al., 2006; Rushton & Jensen, 2005; 2010), and wide-held beliefs that the biological "uniqueness" of Black people influences their response to medical treatment relative to White people (e.g., Hoffman et al., 2016).

Last, I focus on perceptions of Black/White multiracial targets (as opposed to Black or White mono-racial) as I expect to most likely find evidence that people conflate biogeographical ancestry and racial category among targets with mixed ancestry—because of the ambiguous nature of how to racially categorize these individuals (Good et al., 2013; Sanchez & Bonam, 2009). In addition, although using mono-racial targets would serve as a stronger test of the component racialism hypothesis, I use multi-racial targets to first establish proof of concept because these targets would allow for greater explorations of racial gradients (i.e., extent to which biogeographical ancestry is represented as "race genes") in a way that would no be captured with mono-racial targets.

2.1 Pilot Study: Inferring biogeographical ancestry from racial ancestry

With this pilot study, I examine the extent to which people use racial ancestry to make assumptions about a Black/White multiracial target's biogeographical ancestry, and how this

then influences how the target is racially categorized. Following previous, work using a continuous racial categorization measure, I expect people to racially categorize Black/White multiracial targets as more Black as the target's Black racial ancestry increases (Good, Sanchez, & Chavez, 2013; Ho, Roberts, & Gelman, 2015; Sanchez, Good, & Chavez, 2011). I also expect people to assume that, as a target's Black racial ancestry increases, so too will their sub-Saharan biogeographical ancestry (Condit et al., 2004a; Condit et al., 2004b; Dubriwny et al., 2004). In addition, following arguments that biogeographical ancestry represents a new form of racial formation (Omi & Winant, 1994), I expect an indirect effect of target racial ancestry on racial categorization, operating through biogeographical ancestry. Specifically, I expect that as the target's racial ancestry become more Black, participants will increasingly believe that the target has sub-Saharan biogeographical ancestry, which, in turn, I will lead them to increasingly racially categorize the target as Black. Last, I explore whether these processes are evidence of component racialism by statistically controlling for biological racial essentialism beliefs, a less concrete form of racial essentialism. Providing support for component racialism, I expect all conclusions to remain the same when controlling for biological racialist beliefs.

2.1.1 Method

2.1.1.1 Participants

I set target sample size to 250 White U.S. participants in advance to detect small-to-medium effects at at least 80% power (f^2 = .02-.15; Cohen, 1988; Knofczynski & Mundfrom, 2008). I recruited 241 White U.S citizen adults (65% female; M_{age}= 37.44 years, SD_{age} = 12.57) through Amazon's Mechanical Turk (MTurk). MTurk is an online service that allows individuals (known as *requesters*) to post *human intelligence tasks* (HITs) that other individuals (known as *workers*) can complete for small sums of money. Relative to data obtained via traditional

methods (e.g., student subject pools) data obtained through MTurk are reliable (Buhrmester, Kwang, & Gosling, 2011; Crump, McDonnell, & Gureckis, 2013; Mason & Suri, 2011)

2.1.1.2 Materials, Procedure, and Measures

After providing informed consent for the web-based study, participants were asked to imagine a single target (sex or gender identity were not specified) with varying Black/White racial ancestries. Specifically, adapted from Ho et al. (2015), participants imagined a target with either 3 Black and 1 White grandparents (3B/1W), 2 Black and 2 White grandparents (2B/2W) or 1 Black and 3 White grandparents (1W/3B). Participants then made judgments about the target's racial and biogeographical ancestries, completed a measure of biological racial essentialism, and finally responded to basic demographic questions.

Racial categorization. Participants racially categorized the target as White or Black, from 1 (*completely Black*) to 7 (*completely White*). For ease of interpretation, this item was reverse coded, with higher numbers indicating greater Black racial categorization.

Biogeographical ancestry. Participants, after reading the definition of genes — "a unit of heredity that is transferred from the parent to offspring to determine some characteristic of the offspring"— reported the percentage (out of 100) of the target's genes that they believed to be attributable to their Black/sub-Saharan ancestry. For ease of presentation, results are presented in reference to Black/sub-Saharan biogeographical ancestry. Given the zero-sum nature of the biogeographical ancestry measure, recording more Black/sub-Saharan biogeographical ancestry represents less White/European biogeographical ancestry.

Biological racial essentialism. Participants completed the Race Conception Scale (RCS; see Appendix A for all 22 verbatim items; Williams & Eberhardt, 2008). The RCS captures variation in how individuals conceive of race, along a spectrum from thinking about the basis of

race and racial groups as social and flexible to biological and fixed. Examples items include: "How a person is defined racially depends on the social context" (R). "Racial groups are primarily determined by biology." Participants rated all items from 1 (*strongly disagree*) to 7 (*strongly agree*). Higher numbers indicate a more biological, fixed view of race (α = .78).

Manipulation check. Participants selected, from a list of three options, the racial ancestry of their assigned target: 2B/2W, 1B/3W, and 3B/1W.

2.1.2 Results

Preliminary analyses. Table I presents correlations among dependent variables, along with their means and standard deviations.

	1	**2**	**3**
1. Black/Sub-Saharan biogeographical ancestry	-		
2. Racial Categorization	.80***	-	
3. Biological Racial Essentialism	.00	-.08	-
M (SD)	49.32 (20.45)	3.95 (1.35)	4.19 (.86)

Note. $^{**}p < .001$

Table I. PILOT STUDY: CORRELATIONS (*r*), MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR DEPENDANT VARIABLES

No conclusions change when participants failing the manipulation check ($n = 23$)[7] are excluded, therefore the following analyses do not exclude these participants. One participant, whose combined total of Black/sub-Saharan and White/European biogeographical ancestry did not equal 100, is excluded from all analyses; doing so does not change conclusions (final $N =$

[7]An error occurred regarding the manipulation check question answer choices: the "3B/1W" option was incorrectly labeled "3B/1B". Overall, 4 participants in the 1B/3W condition and 2 participants in the 1B/3W condition did not remember the target's racial ancestry. However, in the 3B/1W condition, a majority of participants selected the 3B/1B option ($n = 56$) compared to those who did not ($n = 17$), suggesting that participants in the "3B/1W" condition might have been aware of the error.

240). Unless specified otherwise, controlling for biological racial essentialism did not change result patterns (see Appendix G).

Racial categorization. A one-way analysis of variance (ANOVA) revealed a main effect of racial ancestry on racial categorization, $F(2, 237) = 117.58$, $p < .001$, $\eta_p^2 = 0.50$. Consistent with predictions, planned contrasts revealed that participants racially categorized the 2B/2W target ($M = 4.06$, $SD = .70$) as more Black than the 1B/3W target ($M = 2.82$, $SD = 1.02$), $t(237) = 8.30$, $p < .001$, d = 1.42, but less Black than the 3B/1W target ($M = 5.16$, $SD = 1.13$), $t(237) = 7.12$, $p < .001$, d = 1.17 (see Figure 2).

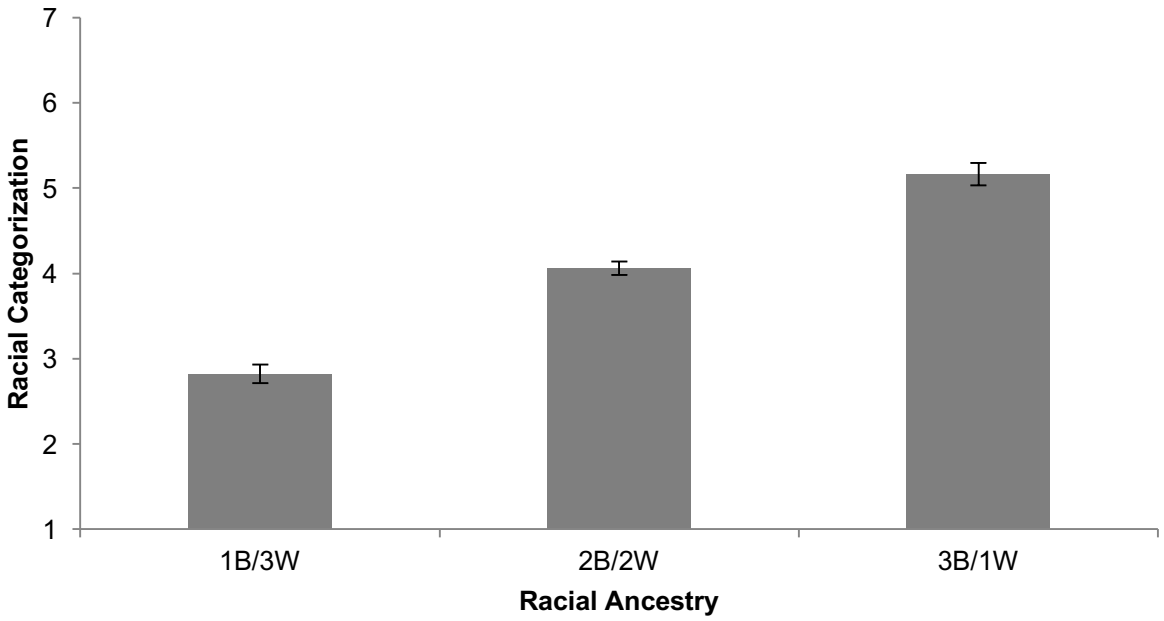


Figure 2. Effect of racial ancestry on racial categorization. Error bars represent standard errors.

Black/sub-Saharan Biogeographical ancestry. A one-way ANOVA revealed a main effect of racial ancestry on biogeographical ancestry, $F(2, 237) = 205.19$, $p < .001$, $\eta_p^2 = 0.63$. Consistent with hypotheses, planned contrasts revealed that participants rated the 2B/2W target ($M = 50.42$, $SD = 6.53$) as having more Black/sub-Saharan biogeographical ancestry than the 1B/3W target ($M = 30.39$, $SD = 14.63$), $t(237) = 10.41$, $p < .001$, d = 1.77, but as having less

Black/sub-Saharan biogeographical ancestry than the 3B/1W target (M = 70.40, SD = 14.42), $t(237) = 9.96$, $p < .001$, d = 1.79 (see Figure 3).

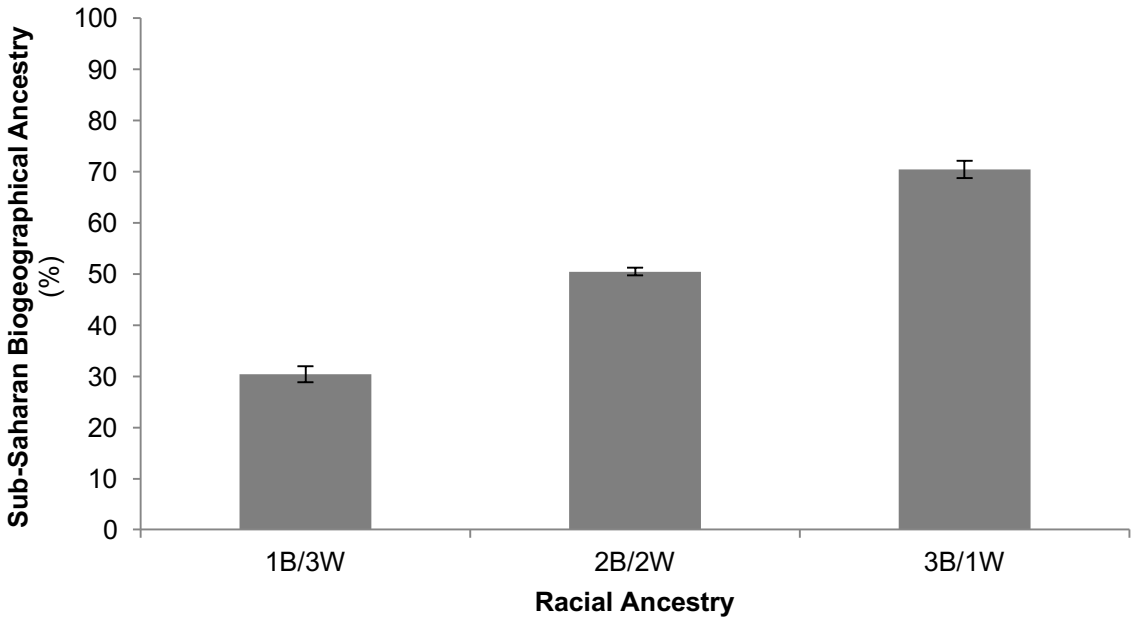


Figure 3. Effect of racial ancestry on biogeographical ancestry. Error bars represent standard errors.

Mediation analyses: Biogeographical ancestry mediates the relationship between racial ancestry and racial categorization. Next, I tested biogeographical ancestry as a mediator between racial ancestry and racial categorization using mediation procedures with Model Template 4 of the SPSS macro PROCESS version 2.16 and 10,000 bootstrap resamples, where a 95% confidence interval (CI) excluding zero indicates a significant effect at the $p < .05$ level (Hayes, 2012; 2016). I used effect coding to examine specific contrasts: D1 compared 2B/2W to 1B/3W (1B/3W = 1, 2B/2W = -1, 3B/1W = 0), and D2 compared 2B/2W to 3B/1W (1B/3W = 0, 2B/2W = -1, 3B/1W = 1).

Consistent with hypotheses, biogeographical ancestry mediated the relationship between racial ancestry and racial categorization, omnibus: b = .03, SE = .01, CI [.018, .040]. Specifically, as expected, participants ascribed more Black/sub-Saharan biogeographical

ancestry to the 2B/2W target compared to the 1B/3W target, which in turn led them to categorize the 2B/2W target as more Black, b = -.86, SE = .15, CI [-1.204, -.600] (see Figure 4 see Figure 4 for path estimates). Also consistent with expectations, participants ascribed more Black/sub-Saharan biogeographical ancestry to the 3B/1W target compared to the 2B/2W target, which in turn led them to categorize the 3B/1W target as more Black, b = .86, SE = .15, CI [.592, 1.198] (see Figure 4 for path estimates).

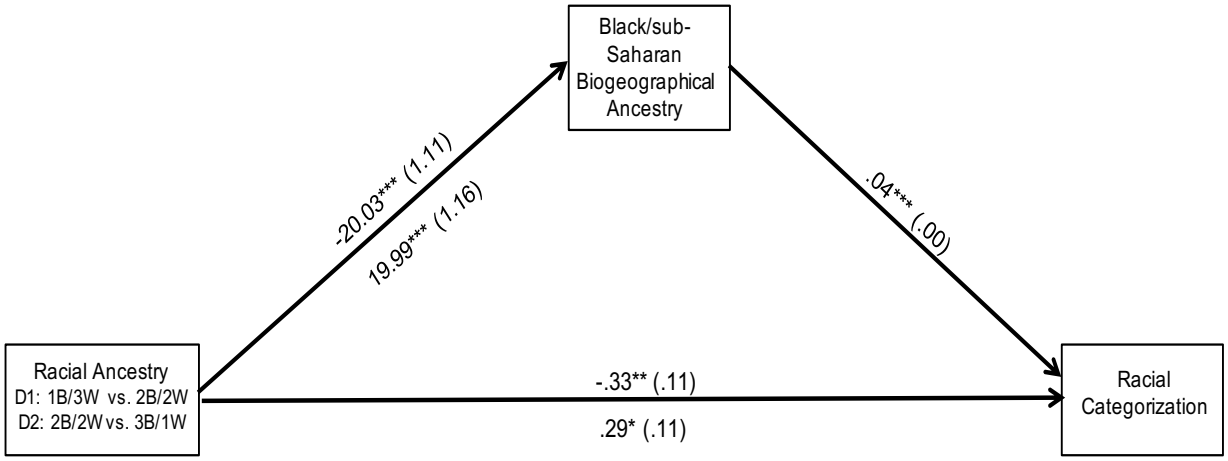


Figure 4. Statistical model depicting the indirect effect of racial ancestry on racial categorization through biogeographical ancestry. Statistics for D1 (1B/3W = 1, 2B/2W = -1, 3B/1W = 0) are presented above the line. Statistics for D2 (1B/3W = 0, 2B/2W = -1, 3B/1W = 1) are presented below the line. Reports direct effect between racial ancestry and racial categorization. All coefficients are unstandardized. Standard errors in parentheses.
*p < .05 ** p < .01 ***p < .001

2.1.3 Pilot Study Discussion

This study provides initial evidence that people engage in component racialism by drawing inferences about a target's biogeographical ancestry on the basis of their racial ancestry, and then using this to racially categorize the them. This evidence suggests that people presume that racial ancestry indicates biogeographical ancestry, which dictates racial category. To an extent, this suggests that along with misrepresenting racial ancestry and biogeographical ancestry, people might also be misrepresenting biogeographical ancestry as "race genes" that

determine racial group membership (or race). Further supporting my theorizing that component racialism is distinct from of biological race conception, controlling for biological racial essentialism did not change the nature of these relationships. Indeed, this result suggests that misrepresentation of biogeographical ancestry, as one manifestation of component racialism, is a more concrete form of biological racial essentialism, potentially with more power to explain racial stereotyping and group attitudes.

However, although this study provides some evidence showing that people use biogeographical ancestry as a genetic placeholder to determine racial categorization (which could have implications for racial stereotyping and perceptions of racial health disparities), it has one key limitation: the wording of the biogeographical ancestry measure might have prompted participants to think about biogeographical ancestry in direct relation to racial ancestry, thus driving the observed effects. To address this potential limitation, in Study 1, I present a more conservative test of component racialism by manipulating biogeographical ancestry without explicit reference to genes/genetics.

2.2 Study 1: Biogeographical ancestry influences (racialized) target perception

Study 1 manipulates biogeographical ancestry to examine its influence on person perception and whether this influence is consistent with component racialism. In particular, I investigate the extent to which biogeographical ancestry (specifically sub-Saharan [i.e., Black] and European [i.e., White]) influences how White people racially categorize and racially stereotype singular targets, along with the assumptions they make about the target's behaviors and physiology. I expect that, as sub-Saharan biogeographical ancestry increases, people will increasingly: racially categorize the target as Black, apply Black racial stereotypes to the target, and perceive the target as less biologically similar to White people.

Moreover, following the Pilot Study showing that biogeographical ancestry influences racial categorization, I expect racial categorization to mediate the relationship between biogeographical ancestry and the aforementioned outcomes (i.e., racial stereotyping and presumed biological and cultural racial difference). Last, to again examine component racialism as a distinct phenomenon, I expect these relationships to remain when controlling for biological racial essentialism.

2.2.1 Method

2.2.1.1 Participants

Following participant recruitment methods and sample size-determining procedures detailed in the Pilot Study, I recruited 374 (57.5% female; M_{age}= 38.47 years, SD_{age} = 12.59) White U.S. citizen adults from MTurk.

2.2.1.2 Procedure, Materials and Measures

After providing informed consent for the web-based study, participants were introduced to a target with Black and White grandparents, who, interested in knowing more about their ancestry (one of the most common reasons why people take an ancestral DNA test; NIH, 2017), takes a biogeographical ancestry test from a 23&Me. 23&Me, a popular direct-to-consumer ancestry testing company, provides biogeographical ancestry testing along with other forms of genetic testing (Wolinsky, 2006). The gender, sex, gender identity, along with other demographic information about target (e.g., age), were not specified. After reading this information, participants are then randomly assigned to view the target's biogeographical ancestry test results, which vary in their reported percentage of sub-Saharan African and European biogeographical ancestries. Participants then made a series of judgments about the target and responded to basic demographic questions.

Biogeographical ancestry. Participants were randomly assigned to one of the following biogeographical test result conditions: (1) 25.8% sub-Saharan African, 73.1% European; (2) 35.8% sub-Saharan African, 63.1% European; (3) 45.8% sub-Saharan African, 53.1% European; (4) 55.8% sub-Saharan African, 43.1% European; (5) 65.8% sub-Saharan African, 33.1% European. All results had 1.1% of the biogeographical ancestry "unassigned". The biogeographical ancestry proportions are based on real 23&Me tests results of Black/White biracial individual (See Figure 5 for an example of how these biogeographic ancestry results are presented).

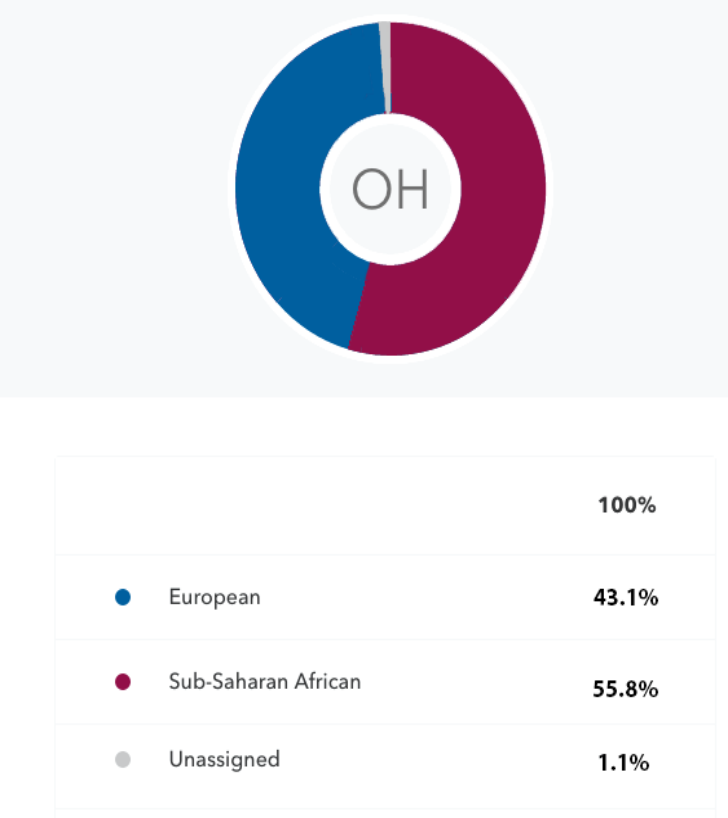


Figure 5. Example of 23&Me test results used in Study 1 biogeographical ancestry manipulation.

Complex racial categorization. Using a validated, 4-item measure of complex racial categorization (Sanchez et al., 2011; Young et al., 2017), participants rated the extent to which they considered the target to be White ("To what extent do you view this applicant as White,"

"To what extent do you think of this applicant as White"; r = .74, $p < .001$). Using the same two items with "Black" replacing "White", participants separately rated the extent to which they considered the target to be Black (r = .78, $p < .001$). Each item was rated from 1 (*not at all)* to 5 (*very much*). The two White items were averaged with each other, as were the two Black items, creating two separate composite measures where higher scores on each measure represent greater racial categorization for the relevant racial group.

Black cultural practices. Using an adapted measure from Young et al. (2007), participants reported the extent to which they believe the target engaged in the following Black-associated cultural practices: "prefers to listen to radio programs that have mostly Black/African Americans hosts", "celebrates Black History month", and "watches TV shows that have mostly Black/African American characters." Participants rated each items on a 7 point Likert scale (1 = *not likely* to 7= *highly likely*). The three items were averaged, with higher scores reflecting greater beliefs that the target engages in Black-associated cultural practices ($\alpha = .87$)**.**

Black racial stereotyping. Using items adapted from a previously established Black stereotype measure (Bonam, Yantis & Taylor, 2017), participants rated the extent to which the target is likely to be (1) athletic (3-items: *plays sports*, *athletic*, *has increased stamina*), (2) criminally inclined (3-items: *not have a criminal record* (R), *be a good person* (R), *engage in criminal activity*), and (3) academically oriented (*excel in school*, *fall behind in school* (R), *go to college*). Participants rated each item on a 7 point Likert scale (1 = *not likely* to 7= *highly likely*). Per each stereotyping sub-scale, all items were averaged, with higher numbers indicating greater Black racial stereotyping in the respective domain ($\alpha_{athletic} = .77$; $\alpha_{criminally\ inclined} = .62$; $\alpha_{academically\ oriented} = .65$).

Racial biological difference. Using 7 false belief items from Hoffman et al. (2016), participants rated the extent to which the target is biologically different from White people (see Appendix B for all 7 verbatim items). Participants rated each item on a 7 point Likert scale (1 =*definitely untrue* to 7 = *definitely true*). All 7 items were averaged, with higher scores indicating greater beliefs that the target is biologically different from White people ($\alpha = .96$).

Biological racial essentialism. Participants completed the Race Conception Scale (RCS; Williams & Eberhardt, 2008; $\alpha = .79$).

Manipulation check. Participants selected the target's test results from a list of five images showing each level of the biogeographical ancestry manipulation.

2.2.2 Results

Preliminary analyses. For ease of presentation, and considering the focus of this research (see Current Studies section), results are presented in reference to sub-Saharan biogeographical ancestry: (1) 25.8%, (2) 35.8%, (3), 45.8%, (4), 55.8%, and (5) 65.8%. Unless otherwise specified, conclusions do not change when participants are excluded for failing the manipulation check ($n = 70$)[8]. The following results include all participants. Table II presents correlations among main study variables, along with their means and standard deviations. Table III presents the means and standard deviations of dependent variables as a function of experimental condition.

[8]12 participants in the 25.8% condition, 21 participants in the 55.8% condition, 8 participants in the 45.8% condition, 16 participants in the 55.8% condition, and 13 participants in the 65.8% condition failed to correctly respond to the manipulation check question.

	1	**2**	**3**	**4**	**5**	**6**	**7**	8
1. Black Racial Categorization	-							
2. White Racial Categorization	-.32***	-						
3. Black Cultural Practices	.46***	-.25***	-					
4. Athletic	.19***	.10	.24***	-				
5. Criminally Inclined	.03	-.11*	.10	-.49***	-			
6. Academically Oriented	-.06	.13*	-.09	.59***	-.76***	-		
7. Biological Difference	.14**	.03	.31**	.08	.32***	-.23***	-	
8. Biological Racial Essentialism	.14**	-.09	.25*	.12*	.16**	-.11*	.13*	-
M (SD)	3.11 (.79)	3.16 (.75)	4.13 (1.13)	4.67 (.95)	3.21 (1.05)	4.70 (.96)	2.63 (1.46)	4.33 (.83)

Note.$^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$

Table II. STUDY 2: CORRELATIONS (*r*), MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR DEPENDANT VARIABLES

	M (*SD*)				
Sub-Saharan Biogeo. Ances.	**25.8** *n* = 75	**35.8** *n* = 84	**45.8** *n* = 67	**55.8** *n* = 70	**65.8** *n* = 76
Black Racial Categorization	2.71 (.81)[cde]	2.91 (.77)[de]	3.10 (.57)[ae]	3.34 (.66)[ab]	3.55 (.80)[abc]
White Racial Categorization	3.52 (.78)[de]	3.38 (.67)[de]	3.23 (.50)[e]	2.98 (.58)[ab]	2.71 (.87)[abc]
Black Cultural Practices	3.80 (1.23)[de]	3.87 (1.09)[e]	4.12 (.92)	4.35 (1.01)[a]	4.57 (1.17)[ab]
Athletic	4.60 (1.10)	4.68 (.96)	4.70 (.91)	4.66 (.88)	4.70 (.92)
Criminally Inclined	3.10 (1.14)	3.23 (1.09)	3.01 (1.09)	3.32 (.96)	3.39 (.94)
Academic Orientation	4.85 (.99)	4.67 (.93)	4.84 (.98)	4.58 (.85)	4.54 (1.02)
Biological difference	2.21 (1.42)[b]	2.94 (1.43)[ac]	2.22 (1.33)[b]	2.85 (1.46)	2.87 (1.48)[ac]

Note. Biogeo. = Biogeographical. Ances. = Ancestry
a represents a difference ($p < .05$) between the marked mean and mean in 25.8/73.1 condition using post-hoc Bonferroni contrasts; *b* represents a difference ($p < .05$) between the marked mean and mean in 35.8/63.1 condition using post-hoc Bonferroni contrasts; *c* represents a difference ($p < .05$) between the marked mean and mean in 45.8/53.1 condition using post-hoc Bonferroni contrasts; *d* represents a difference ($p < .05$) between the marked mean and mean in 55.8/43.1 condition using post-hoc Bonferroni contrasts; *e* represents a difference ($p < .05$) between the marked mean and mean in 65.8/33.1 condition using post-hoc Bonferroni contrasts.

Table III. STUDY 2: MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR DEPENDANT VARIABLES BY CONDITION

Primary analyses: Biogeographical ancestry effects. I tested the effect of biogeographical ancestry on (1) racial categorization, (2) cultural practices, (3) racial stereotyping, and (4) biological difference using linear regression analyses. I included manipulated biogeographical ancestry as a continuous predictor in each regression, as it increases by equal increments across the five conditions (see Pasta, 2009; Rhemtulla, Brosseau-Liard & Savalei, 2012; Williams, 2016), and because hypotheses are centered on understanding how increasing percentage of sub-Saharan biogeographical ancestry influences this study's dependent outcomes. Unless specified otherwise, controlling for biological racial essentialism did not change result patterns (see Appendix G).

Complex racial categorization. Consistent with hypotheses, increasing sub-Saharan biogeographical ancestry lead to greater Black racial categorization, $b = .21$, $SE = .03$, $p < .001$,

CI [.160, .264] (see Figure 6). Also consistent with hypotheses, increasing sub-Saharan biogeographical lead to decreasing White racial categorization, b = -.20, SE = .03, p < .001, CI[-.252, -.153] (see Figure 7).

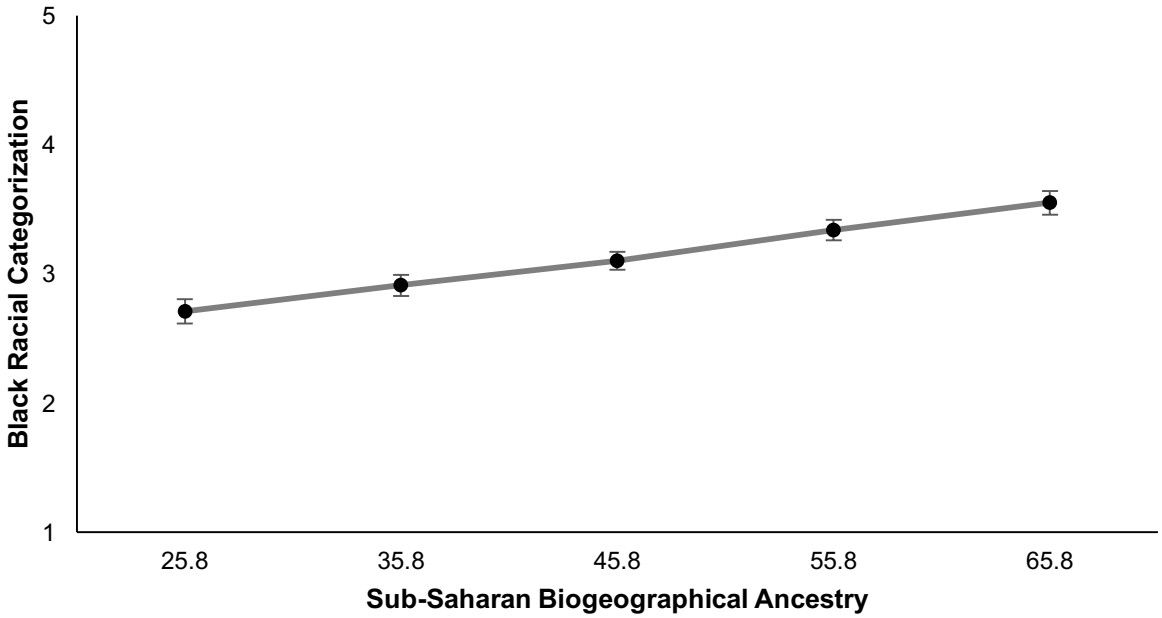


Figure 6. Effect of sub-Saharan Biogeographical ancestry on Black racial categorization. Error bars represent standard errors.

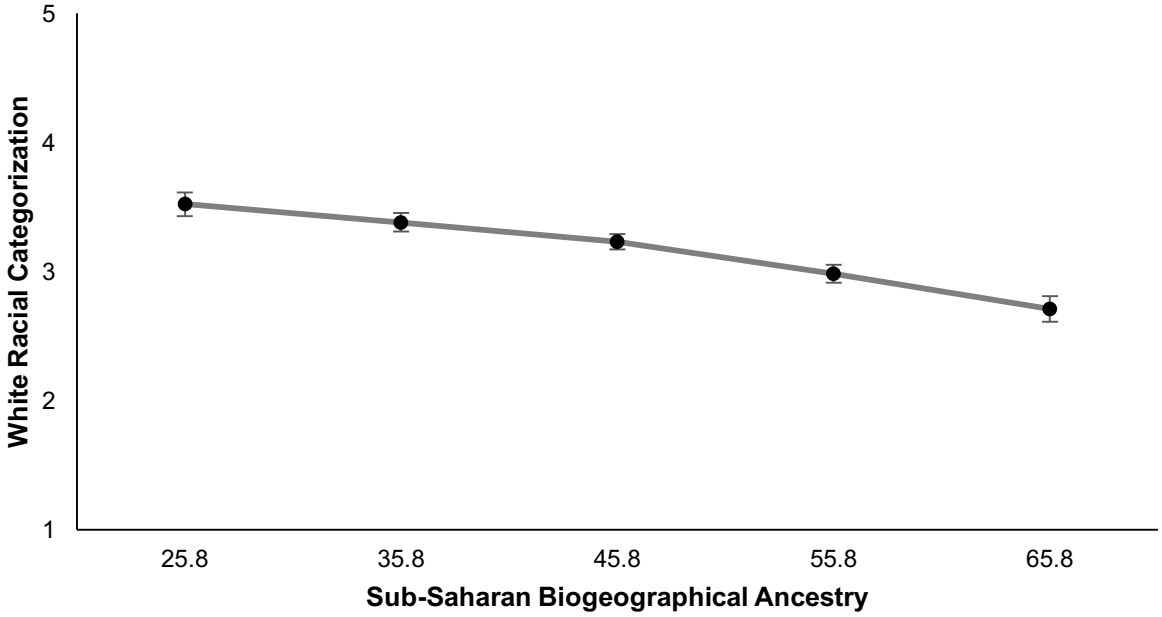


Figure 7. Effect of sub-Saharan Biogeographical ancestry on White racial categorization. Error bars represent standard errors.

Black cultural practices. Consistent with hypotheses, increasing sub-Saharan biogeographical ancestry increased beliefs that the target engaged in Black-associated cultural practices, $b = .20$, $SE = .04$, $p < .001$, CI [.125, .281] (see Figure 8).

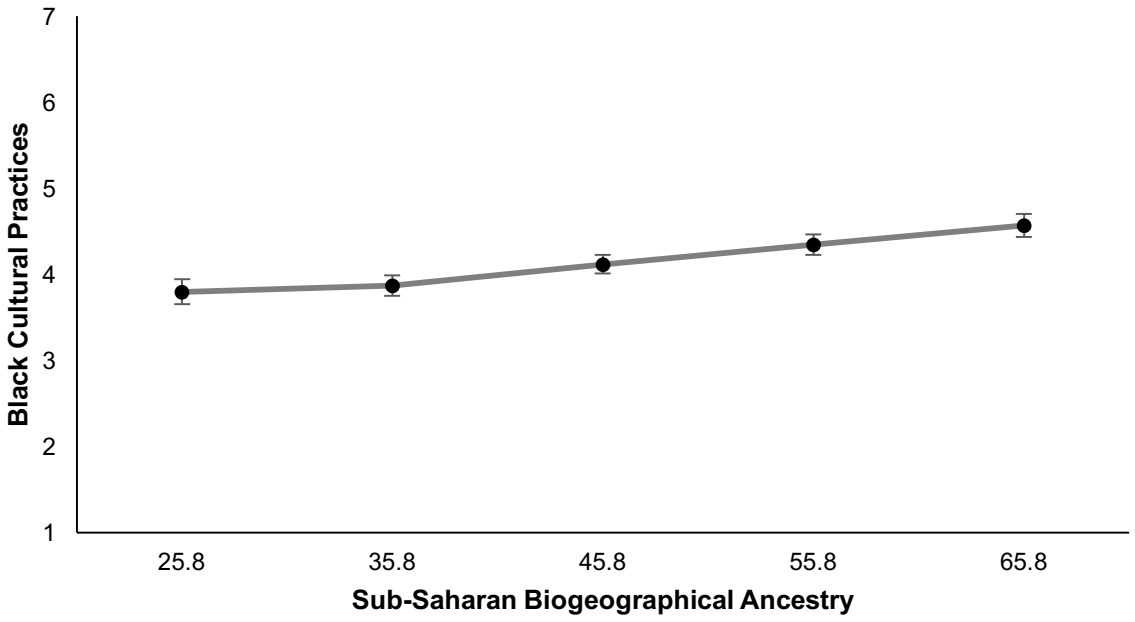


Figure 8. Effect of sub-Saharan Biogeographical ancestry on Black Cultural practices. Error bars represent standard errors.

Black racial stereotyping. Consistent with hypotheses, increasing sub-Saharan biogeographical ancestry decreased beliefs that the target is academically oriented, $b = -.07$, $SE = .04$, $p = .045$, CI [-.139, -.002][9] (see Figure 9). However, inconsistent with hypotheses, sub-Saharan biogeographical ancestry did not shift beliefs about the target's athletic ability, $b = .02$, $SE = .04$, $p = .601$, CI [-.050, .087], or criminal inclination , $b = .07$, $SE = .04$, $p = .080$, CI [-.008, .142].

[9]The effect of biogeographical ancestry on academic orientation becomes non-significant when participants failing the manipulation check are excluded from the analysis, $b = -.05$, $SE = .04$, $p = .189$, 95%CI [-.131, .026].

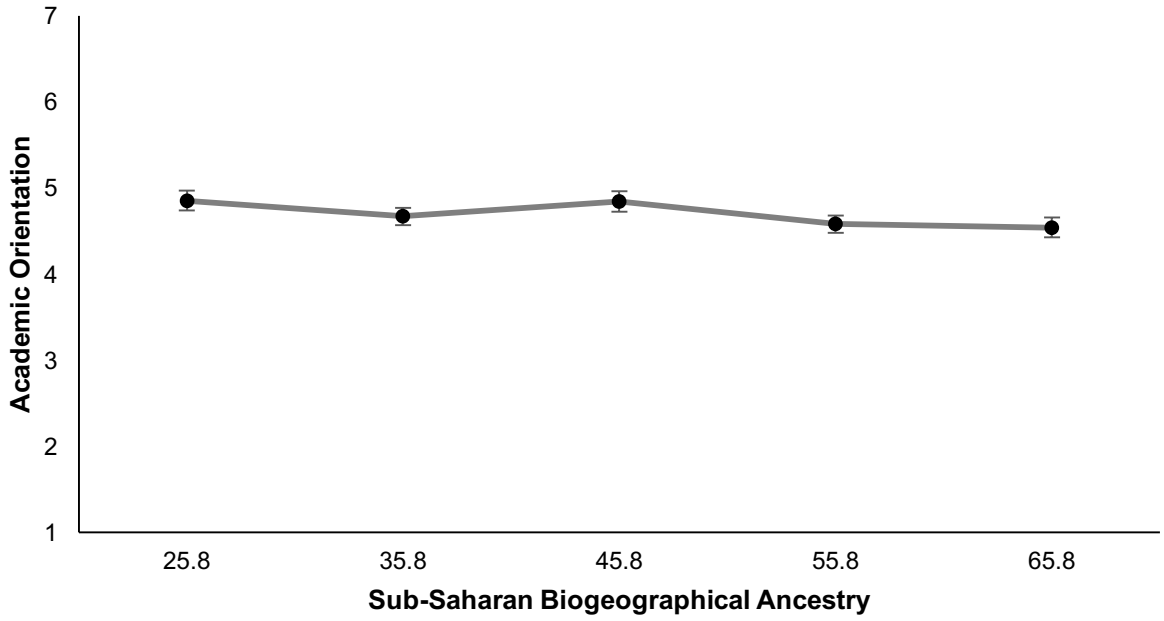


Figure 9. Effect of sub-Saharan Biogeographical ancestry on Academic Orientation. Error bars represent standard errors.

Biological difference. Consistent with hypotheses, increasing sub-Saharan biogeographical ancestry increased beliefs that the target is biologically different from White people, $b = .12$, $SE = .05$, $p = .029$, CI [.012, .220] (see Figure 10).

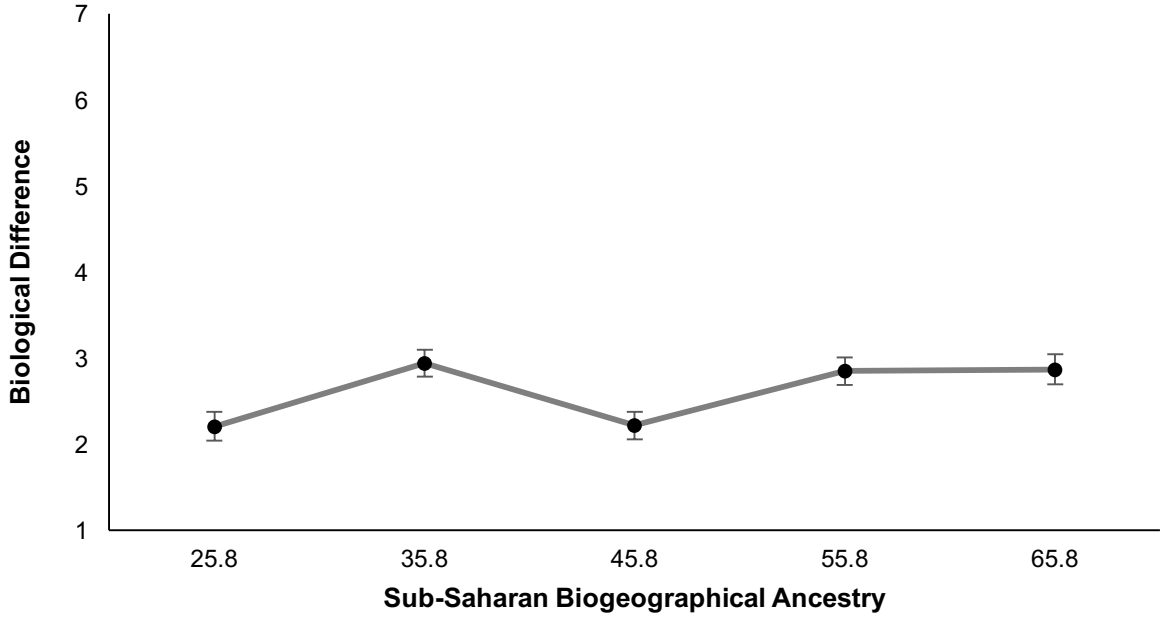


Figure 10. Effect of sub-Saharan Biogeographical ancestry on Biological difference. Error bars represent standard errors.

Mediation analyses: Racial categorization as a mediator. Across 10 separate mediation analyses (same procedure as Study 2), I tested racial categorization (first Black, then White) as a mediator between biogeographical ancestry and (1) cultural practices, (2) racial stereotyping, and (3) biological difference. I entered biogeographical ancestry into the regressions as a continuous predictor, with conditions coded from 1 to 5 (i.e., 1= 25.8%, 2 = 35.8%, 3 = 45.8%, 4 = 55.8%, 6 = 65.8%; see Pasta, 2009; Rhemtulla et al., 2012; Williams, 2016). Unless specified otherwise, controlling for biological racial essentialism did not change result patterns.

Black racial categorization as a mediator

Black cultural practices. Consistent with predictions, Black racial categorization mediated the relationship between biogeographical ancestry and Black cultural practices, b = .13, SE = .03, CI [.082, .190]. Specifically, as expected, increasing sub-Saharan biogeographical ancestry increased Black racial categorization, which in turn predicted increasing beliefs that the target engaged in Black-associated cultural practices (see Figure 11 for path estimates).

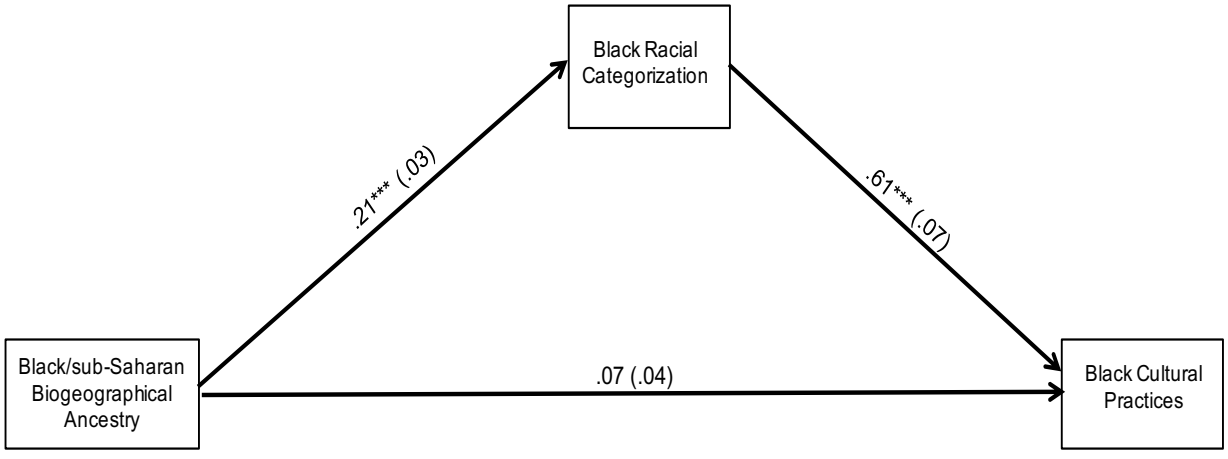


Figure 11. Indirect effect of biogeographical ancestry on cultural practices through Black racial categorization. Reports direct effect between biogeographical ancestry and Black cultural practices. All coefficients are unstandardized. Standard errors in parentheses.
***$p < .001$

Black racial stereotyping. *Athleticism*. Consistent with predictions, Black racial categorization mediated the relationship between biogeographical ancestry and athleticism, b = .05, SE = .02, CI [.023, .096]. Specifically, as expected, increasing sub-Saharan biogeographical ancestry increased Black racial categorization, which in turn predicted increasing beliefs that the target is athletic (see Figure 12 for path estimates).

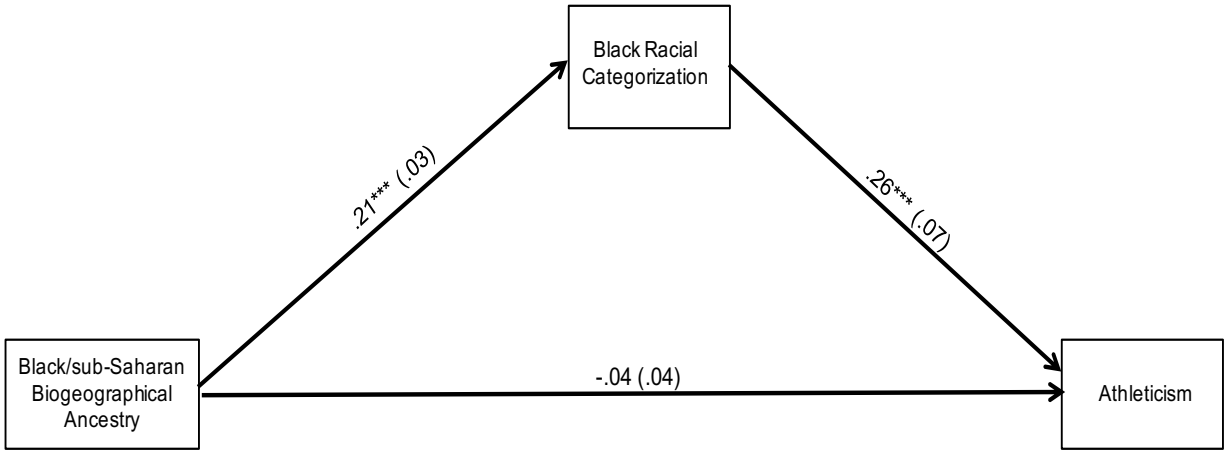


Figure 12. Indirect effect of biogeographical ancestry on athleticism through Black racial categorization. Reports direct effect between biogeographical ancestry and athleticism. All coefficients are unstandardized. Standard errors in parentheses.
***$p < .001$

Academic orientation. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and academic orientation, b = -.01, SE = .02, CI [-0.39, .010].

Criminal inclination. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and criminal inclination, b = .00, SE = .02, CI [-.038, .034].

Biological difference. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and biological difference, b = .04, SE = .02, CI [-.001, .097].

White racial categorization as a mediator

Black cultural practices. Consistent with predictions, White racial categorization mediated the relationship between biogeographical ancestry and cultural practices, b = .05, SE = .02, CI [.011, .234]. Specifically, as expected, more sub-Saharan biogeographical ancestry decreased White racial categorization, which in turn predicted decreasing beliefs that the target engaged in Black-associated cultural practices (see Figure 13 for path estimates).

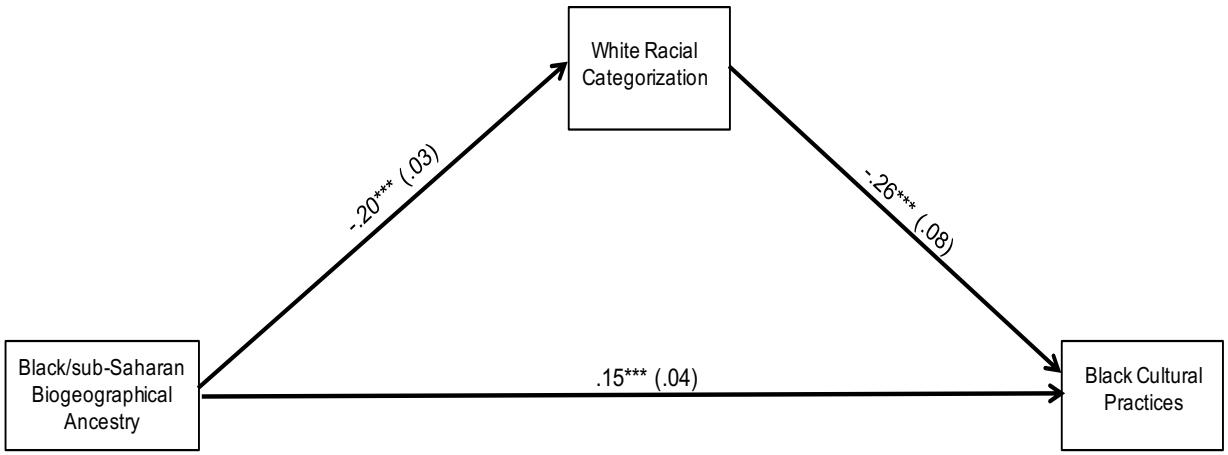


Figure 13. Indirect effect of biogeographical ancestry on cultural practices through White racial categorization. Reports direct effect between biogeographical ancestry and Black cultural practices. All coefficients are unstandardized. Standard errors in parentheses.
***p < .001

Black racial stereotyping. *Athleticism*. Consistent with predictions, White racial categorization mediated the relationship between biogeographical ancestry and athleticism, b = -.03, SE = .02, CI [-.068, -.002]. Specifically, as expected, more sub-Saharan biogeographical

ancestry decreased White racial categorization, which in turn predicted decreasing beliefs that the target is athletic (see Figure 14 for path estimates).

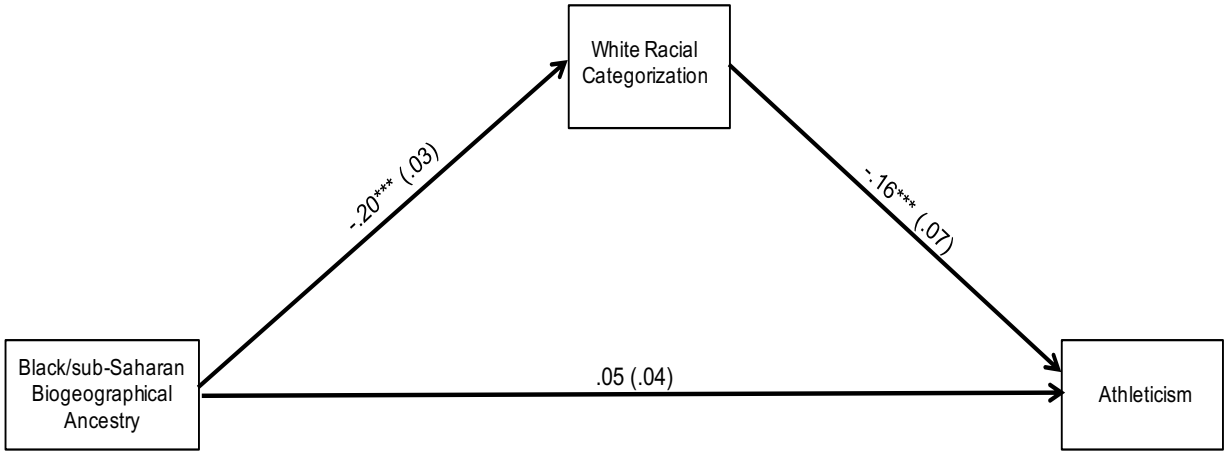


Figure 14. Indirect effect of biogeographical ancestry on athleticism through White racial categorization. Reports direct effect between biogeographical ancestry and athleticism. All coefficients are unstandardized. Standard errors in parentheses.
***$p < .001$

Academic orientation. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and academic orientation, b = -.03, SE = .02, CI [-.063, .002].

Criminal inclination. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and criminal inclination, b = .03, SE = .02, CI [-.008, .064].

Biological difference. Inconsistent with hypotheses, Black racial categorization did not mediate the relationship between biogeographical ancestry and biological difference, b = -.03, SE = .03, CI [-.084, .018].

2.2.3 Study 1 Discussion

Overall, results from Study 1 show that people use biogeographical ancestry to racially categorize and make consequential racialized judgments about targets. These results show that the effect of biogeographical ancestry is impactful even when participants have information

about the target's racial background (even if not as specific as the racial ancestry info presented in the Pilot Study). Indeed, this evidence is consistent with my prediction that people engage in component racialism, by misrepresenting biogeographical ancestry as "race genes". For example, increasing sub-Saharan biogeographical ancestry causes people's perceptions of the target to become more aligned with Black racial stereotypes (i.e., less academically oriented). People are also more likely to assume the target will engage in Black cultural practices and be biologically different from White people. People therefore see biogeographical ancestry as a source of not only Black-White racial biological difference, but also as a predictor of important behavioral patterns (i.e., athletics, Black cultural engagement). Critically, the study also presents more supporting evidence for component racialism as distinct from, and holding predictive power over and above, biological racial essentialism as these relationships remain even when controlling for biological racial essentialism

Furthermore, results show that racial categorization mediates the relationship between biogeographical ancestry and beliefs about the target's athletic ability and the extent to which the target engages in Black cultural practices. This demonstrates that in addition to directly influencing these race-based target judgments, biogeographical ancestry also influences these judgments via perceptions of the target's racial group category. This reveals that people sometimes use both genetic and social (i.e., racial categorization) information to make race-based judgments; results that conceptually replicate work showing that people use both cultural and biological essentialist beliefs to explain and describe racial groups (Morning, 2009; Soylu Yalcinkaya et al., 2017; Zeromskyte & Wagner, 2017). This also highlights that people think of racial differences as arising from both genetic and social factors (e.g., "A person is more likely to watch T.V. shows with Black casts, not only because of their genetic makeup, but because their

genetic makeup places them in a racial category, that, more often than not, watches shows with Black casts"). Interestingly, however, racial ancestry did not mediate the relationship between biogeographical ancestry and either academic orientation, criminal inclination, or biological difference. In these instances, it is conceivable that more pronounced beliefs that the aforementioned outcomes result from racial genetic differences (academic orientation [i.e., intelligence] and criminality have often though to result from biological/genetic factors; Rushton, 1990; Rushton & Jensen, 2005; 2010) drove direct effects from biogeographical ancestry to these outcomes. Together, these results suggest that not only might people believe that racial differences are due to racial genetic differences, they also hold ideas about how racial group membership can lead to disparate outcomes, potentially because of or in tandem with genetic differences.

Despite this study's contributions, one question that is left unanswered is: To what extent, if at all, do these biogeographical ancestry effects shift in the presence of information about the target's racial ancestry? In other words, will I find evidence that people still engage in component racialism when they have more specific information about the target's racial background? In this study, participants are only told that the target has Black and White grandparents and did not know the proportion Black vs. White ancestry (as in the Pilot Study). In addition, although controlling for biological racial essentialism serves as a test of component racialism's distinctiveness in predicting race-based judgments, doing so is limited because biological racial essentialism represents a more abstract form of biological race conception. Consequently, to strengthen tests of component racialism, it is important to control for ideas about racialism (i.e., specific ideas that genes determine race). Further, though this study provides evidence suggesting that people misrepresent biogeographical ancestry as "race genes",

it does not include a measure that explicitly (or less ambiguously) captures this (mis)representation. Administering such a measure would provide a more conservative and direct test of the component racialism hypothesis. Altogether, Study 2 addresses these limitations to provide a more conservative test of component racialism than the Pilot Study and Study 1.

2.3 Study 2: Biogeographical ancestry and racial ancestry influence (racialized) target perception

Study 2 combines the Pilot Study and Study 1 procedures, with three primary changes to provide a more conservative and direct test of component racialism: First, I measure and co-vary for racialist ideologies. This analysis will test my prediction that component racialism holds predictive power beyond racialist beliefs, which are more concrete and closely related to component racialism than biological racial essentialism (i.e., Study 1's covariate, which I again measure in Study 2). Second, I add a more direct measure of "race genes". Specifically, compared to Study 1, I more directly attempt to capture the component racialism phenomenon by testing my prediction that increasing a target's sub-Saharan biogeographical ancestry will lead perceivers to believe that the target's genetic profile is more similar to the prototypical Black person's genetic profile; this will indicate that people misrepresent biogeographic ancestry as "race genes".

Third, I simultaneously manipulate racial ancestry and biogeographical ancestry to examine whether biogeographical ancestry still drives perceptions of the target when observers have detailed knowledge of the target's racial ancestry. Manipulating racial ancestry will provide greater insight into component racialism, as it will allow for clearer examination of how, and the extent to which, people use biogeographical information to make race-based judgments, even with racial ancestry information present (unlike in Study 1). Further, including a condition with

no racial ancestry information (unlike Study 1 and the Pilot, which both specified the target's racial ancestry) will highlight how biogeographical ancestry information influences target judgments when it is completely unconstrained by racial ancestry information.

Along with providing a more conservative and direct test to capture component racialism, I also broaden my examination of this phenomenon by testing its implications for a wider range of key racialized perceptions and judgments. Specifically, along with measures from the Pilot Study and Study 1, I examine presumptions about the target's skin tone, superhuman physical capabilities and ability to overcome pain, and susceptibility to illnesses and diseases that have become racialized. I investigate these outcomes because: (1) people perceive Black people (relative to White people) as possessing greater superhuman abilities (e.g., greater pain tolerance; Waytz et al., 2015) due to Black-White genetic and biological differences (e.g., Hoffman et al., 2016); (2) endorsing beliefs about Black-White genetics is related to the increased use of skin tone to racially categorize targets (i.e., darker skin tones are associated with increased Blackness; Plaks et al., 2012); and (3) both lay people and medical health professionals believe that racial biological and genetic variations explain racial differences in illness/disease susceptibility (Condit et al., 2004a; Condit et al., 2004b; Institute of Medicine, 2003; Shields et al., 2005).

Following results from the Pilot Study and Study, I expect a main effect of biogeographical ancestry, such that increasing sub-Saharan (i.e., Black) biogeographical ancestry will lead to increasing beliefs that the target: is susceptible to illness/disease; possesses superhuman abilities and Black-associated stereotypical characteristics; has a darker skin tone; engages in Black-associated cultural practices; is biologically different from White people; and is genetically similar to Black people. I also expect this main effect of biogeographical ancestry because some tentative evidence suggests it will supersede racial ancestry information in

participants' race-based target judgments and evaluations (see example of Sgt. Brown; Eligon, 2017).

Additionally, I expect this main effect (of biogeographical ancestry) will be carried to target judgments and evaluations through perceptions of the target's "race genes", and through perceptions of the target's "race genes" and racial categorization in serial. Specifically, I expect to find evidence that, as a target's level of sub-Saharan African biogeographical ancestry increases, participants will increasingly perceive the target as having more "Black race genes", which in turn will lead to increasing Black-associated judgments and evaluations. Further, in serial, I expect to find evidence that increasing perceptions that the target has more "Black race genes" will be associated with increased Black (decreased White) racial categorization, which in turn will lead to increasing Black-associated (decreasing White-associated) judgments and evaluations (see Figure 15 for a conceptual mediation model).

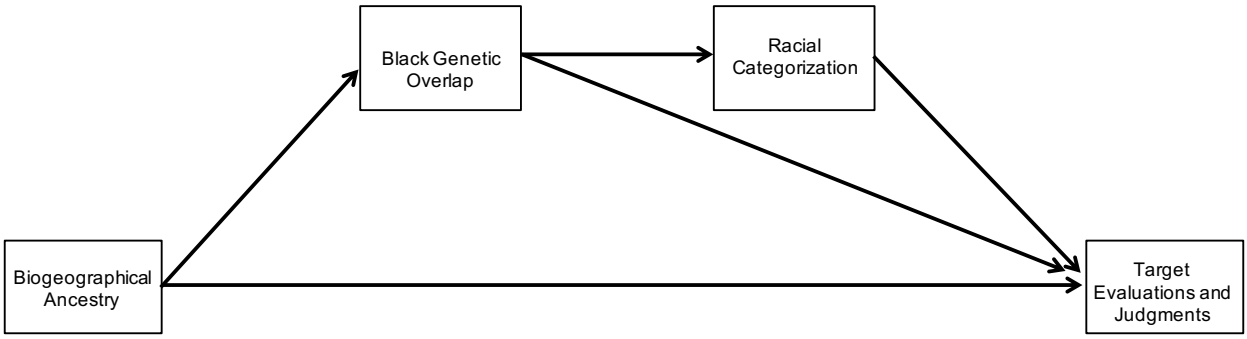


Figure 15. Conceptual Model for Study 2 mediations.

Alternatively, it is also likely that I could find evidence of an interaction between biogeographical ancestry and racial ancestry. In other words, I could expect participants to use both biogeographical and racial ancestry information (i.e., operating simultaneously) when making target judgments and evaluations. If this interaction does occur, I would expect participants to make judgments and evaluations of the target that are consistent with the target's Black racial and biogeographical ancestries (e.g., Study 1; Good et al., 2013; Young et al., 2017),

as a way to police the White racial border (Lewis, 2003). With this hypothesis, I expect participants to use whichever cue that indicates greater Black ancestry to racially categorize, perceive, and judge the target in a way that more closely matches the prototypical image of a Black person.

In particular, when the target has more Black than White racial ancestry, I do not expect an effect of biogeographical ancestry. However, when the target has less Black than White racial ancestry, I expect participants to use biogeographical ancestry to racially categorize, perceive, and judge the target in a way that more closely matches the prototypical image of a Black person. Specifically, I expect that when the target has less Black than White racial ancestry, increasing Sub-Saharan African biogeographical ancestry will lead to increased Black-associated race-based target judgments and evaluations. Similarly, when the target's racial ancestry is unspecified, I expect increasing Sub-Saharan African biogeographical ancestry will lead to increased Black-associated race-based target judgments and evaluations; however, I anticipate this relationship to be relatively stronger than when the target has less Black than White racial ancestry.

Additionally, I expect the interaction between racial and biogeographical ancestry will be carried to the target judgments and evaluations through perceptions of the target's "race genes", and through perceptions of the target's "race genes" and racial categorization in serial. However, I anticipate these indirect effects will be strongest when no racial ancestry information is provided, present but relatively weaker when the target has less Black than White racial ancestry, and absent when the target has more Black than White racial ancestry (see Figure 16 for a conceptual moderated mediation model).

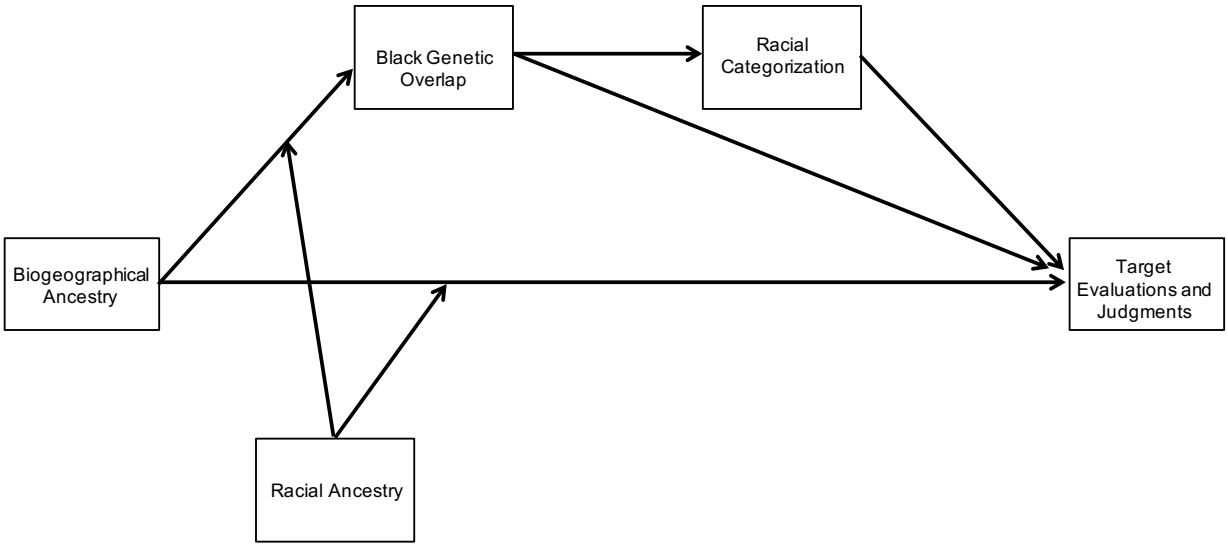


Figure 16. Conceptual Model for Study 2 moderated mediations and moderated serial mediations.

Last, I expect all the aforementioned relationships to remain significant even after independently controlling for biological racial essentialist and racialist ideologies. Moreover, I emphasize now that total and indirect effects of biogeographical ancestry (on target judgments and evaluations) that are not moderated by racial ancestry would actually provide the strongest evidence for component racialism, as they would show that the evaluative consequences of biogeographical ancestry are not dependent on an individual's racial ancestry.

2.3.1 Method

2.3.1.1 Participants

Following participant recruitment methods and sample size determination procedures detailed in the Pilot Study, and effects sizes obtained in Study 1, I recruited 647 (58.1% female; M_{age}= 38.81 years, SD_{age} = 12.59) White U.S. citizen adults from MTurk.

2.3.1.2 Materials and Procedure

After providing informed consent for the web-based study, participants were introduced to a target who, interested in understanding more about their susceptibility to illness and disease, decided to take an ancestral DNA test from 23&Me. After reading the cover story, participants

were randomly assigned to view a target profile providing the target's racial ancestry (mostly Black, mostly White, or unspecified). Next, they were randomly assigned to view a set of DNA test results providing filler results and the target's biogeographical ancestry (more sub-Saharan African than European, equally sub-Saharan African and European, or less sub-Saharan African than European). Participants then made judgments about the target and responded to basic demographic questions.

Target profile with racial ancestry manipulation. The number of Black and White grandparents vary in each version of the target profile (see Ho et al., 2015; see figure 17); condition abbreviations are in parentheses): 3 Black and 1 White grandparents (3B/1W); 1 Black and 3 White grandparents (1B/3W); or a control profile where the number of grandparents is "Not Set" (unspecified).

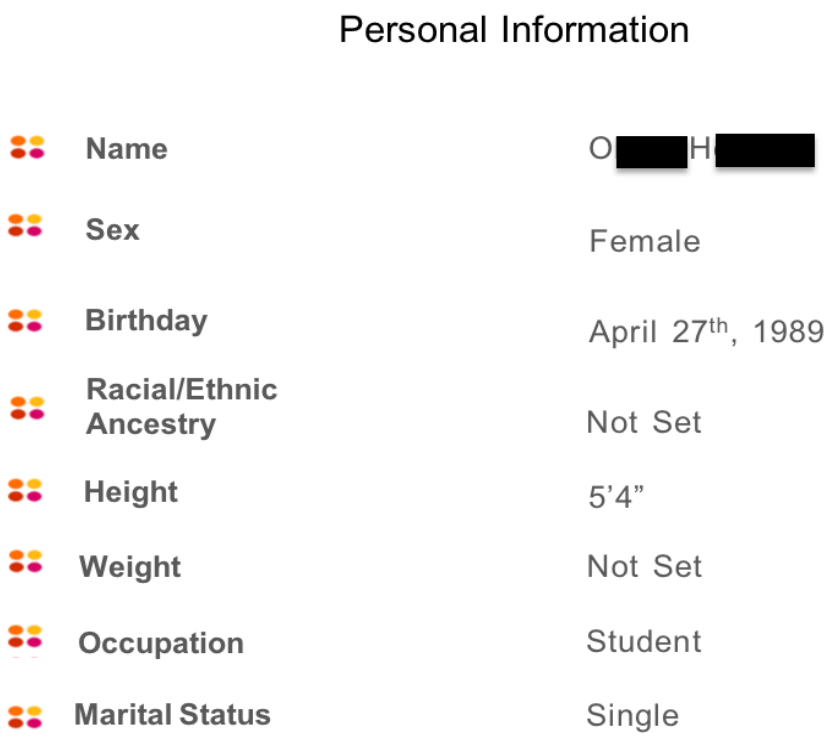


Figure 17. Target profile example used in Study 2 for Racial Ancestry manipulation.

Biogeographical ancestry manipulation and filler information. There were three versions of biogeographical ancestry test results, which were always paired with a set of 12 filler test results (see figure 18 for example presentation of both target and filler test results; condition abbreviations are in parentheses): (1) 24.7% Sub-Saharan African, 74.2% European, 1.1% Unassigned (25A/75E); (2) 49.4% Sub-Saharan African, 49.5% European, 1.1% Unassigned (50A/50E)[10]; and (3) 74.2% Sub-Saharan African, 24.7% European, 1.1% Unassigned (75A/25E).

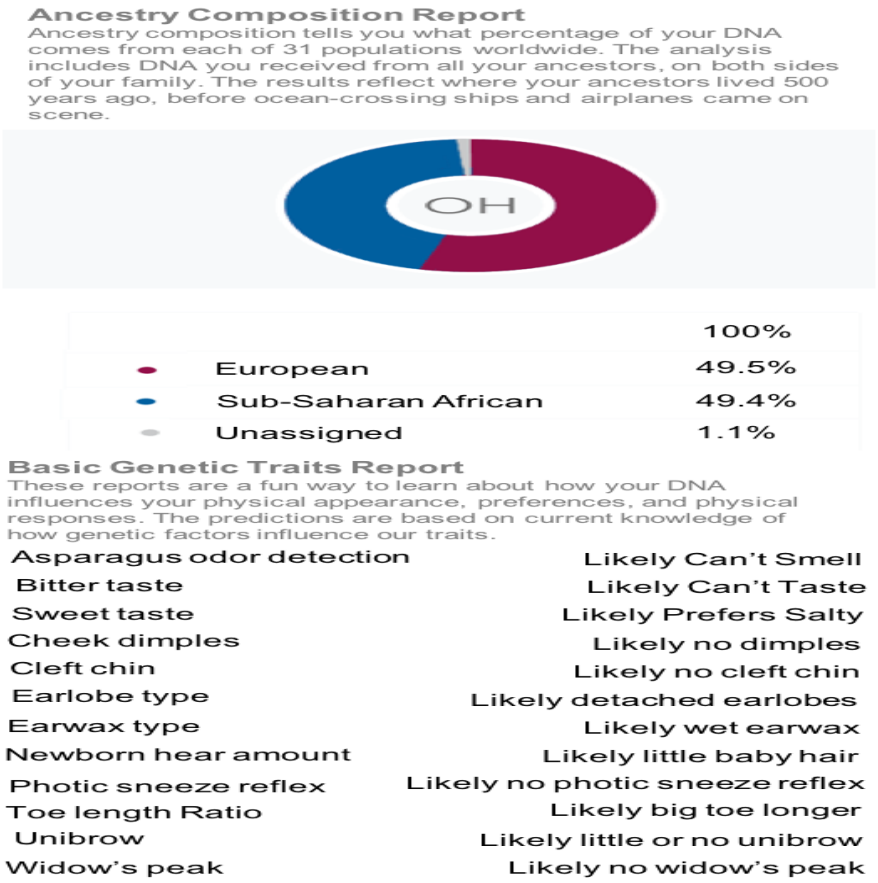


Figure 18. Target ancestry DNA test results used in Study 2 for Biogeographical Ancestry manipulation.

[10]A second version of the 50A/50E condition was formatted as 49.5% Sub-Saharan African, 49.4% European, 1.1% Unassigned. With a one-way ANOVA, I tested whether there were differences between these two versions. No differences emerged on any of the dependent measures, *p*s range from .142 to .684. Following this result, I combined these two conditions into one 50A/50E biogeographic ancestry condition.

Black genetic overlap. Following Plaks et al. (2012), participants rated the percentage of genetic material the target shared with the average Black/African American person (0–100%).

Superhumanization. Using items adapted from Waytz and colleagues (2015), participants rated the extent to which the target displays superhuman pain tolerance and physicality.

*Pain tolerance***.** Specifically, from on a 7-point Likert scale (1= *none* to 7 = *an extreme amount*), participants rated how much pain medication the target would need after experiencing the following: (1) were involved in a car crash, (2) dislocated a shoulder playing sports, (3) were burnt by touching a hot dish, (4) had their wisdom teeth removed, (5) stapled their fingers at work, (6) got shingles, and (7) hurt themselves while assembling furniture. All items were averaged, with higher values representing greater beliefs that the target is pain tolerant ($\alpha = .85$).

*Physicality***.** Participants rated the extent to which the target is capable of: (1) "surviving a fall from an airplane without breaking a bone", (2) "running faster than a cheetah", and (3) lifting up a tank". Participants rated each item on a 7-point Likert scale (1 = *very unlikely* to 7 = *very likely*). Responses to all items were averaged to create a *physicality* measure. All items were averaged, with higher values represent greater beliefs that the target possesses superhuman physical abilities ($\alpha = .94$).

Illness and disease susceptibility. Participants rated the extent to which the target is likely to develop mental illnesses (depression, post-traumatic stress disorder [PTSD], generalized anxiety disorder), physical illness (hypertension/high blood pressure, diabetes, stroke, obesity), and sexually transmitted diseases/infections ([STDs/STIs]; HIV/AIDS, gonorrhea, chlamydia, syphilis), from 1 (*very unlikely*) to 7 (*very likely*). For all illness and disease categories, empirical work has demonstrated non-Hispanic Black and non-Hispanic White prevalence disparities:

mental illness (Asnaani, Richey, Dimaite, Hinton, & Hofmann, 2010; Centers for Disease Control and Prevention [CDC], 2014; Roberts, Gilman, Breslau, Breslau, & Koenen, 2011); physical illness (National Center for Health Statistics [NCHS], 2016); STDs/STIs (CDC, 2015; 2016). Three different composite measures were created: *physical illness*, *mental illness*, and *STD/I*. Higher values represent a greater belief that the target is likely to develop each category of illness or disease ($\alpha_{\text{mental illness}} = .80$; $\alpha_{\text{physical illness}} = .85$; $\alpha_{\text{STD/STI}} = .94$).

Skin tone. Using an adapted version of Massy and Martin Skin Color Scale (Massey & Martin, 2003), participants selected the tone that best represents how they imagine the target's skin. The ten shades of skin color corresponding to the points 1 to 10 are depicted in a chart, with each point represented by a hand, of identical form, but differing in color. Increasing values are associated with increased skin tone darkness (See Appendix C for image).

Black racial stereotyping. Following Bonam, Yantis & Taylor (2018), participants rated the extent to which the target possesses characteristics that are associated with Black people on a 7-point Likert scale (1 = *very unrepresentative* to 7 = *very representative*). Sample items include: *uneducated*, *poor*, *clean* (R), *dangerous* (see Appendix D for full list of items). The appropriate items were reverse coded and all items were then averaged. Greater numbers represent more negative evaluations of the target and a greater belief that the target possessed stereotypically Black characteristics ($\alpha = .92$).

Racialism. Adapting and combining items from the Race Conception Scale (Williams & Eberhardt, 2008) and Belief in Genetic Determinism Scale (i.e., a general measure of biological essentialism; Keller, 2005), I constructed a measure of *racialism* (i.e., genetic racial essentialism). Unlike other measures, this measure attempts to capture people's specific beliefs about the extent to which genes determine race and racial differences—a more specific and

concrete measure of biological racial essentialism. Participants rated the extent to which they agree to the following items, from 1(*strongly disagree*) to 7 (*strongly agree*): (1) It's impossible to determine how a person will be racially categorized by examining their DNA (R); (2) Genetic differences is an important cause for the differences in abilities between individuals from racial groups; (3) Very few behavioral traits of racial groups can be traced back to their genes; (4) Differences between in behavior and personality between racial groups are largely determined by genetic predisposition; (5) The chief reason why members of the same racial group are so alike in behavior and character is that they possess a shared genetic inheritance. The appropriate items were reverse coded and all items were then averaged. Greater numbers represent a greater endorsement of racialist ideologies (i.e., genes determine race; $\alpha = .75$).

Pilot Study and Study 1 measures. Participants completed the following measures from Study 1 or the Pilot Study: complex racial categorization ($r_{Black} = .90$; $r_{White} = .91$), cultural practices ($\alpha = .89$), biological difference ($\alpha = .93$), biological racial essentialism ($\alpha = .89$).

Manipulation checks. From a list of photos, participants selected the ancestral DNA test results and the target's racial ancestry they viewed.

2.3.2 Results

Preliminary analyses. Table IV presents correlations among main study variables, along with their means and standard deviations. Table V presents the means and standard deviations of dependent variables as a function of experimental condition. Unless otherwise specified, conclusions do not change when participants are excluded for failing the manipulation check ($n_{ancestry\ test} = 89$; $n_{racial\ ancestry} = 139$)[11]. The following results include all participants.

[11] Participants failing the ancestral DNA test manipulation check question: 25A/75E = 29; 50A/50E = 26; 75A/25E= 34. Participants failing the racial ancestry manipulation check question: 1B/3W = 52; 3B/1W = 61; unspecified = 26.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1.Black Gen. Overlap	-													
2.Pain Tolerance	.03	-												
3.Physicality	.03	.08	-											
4.Mental Illness	.09*	.19***	.16***	-										
5.Physical Illness	.14***	.21***	.04	.67***	-									
6.STD/STIs	.06	.16***	.22***	.63***	.50***	-								
7.Skin Tone	.37***	.04	.15***	.04	.09*	.09*	-							
8.Stereotyping	.04	-.02	.20***	.07	.11**	.38***	.08	-						
9.Black Racial Cat.	.37***	.04	-.02	.03	.14**	.09*	.52***	-.01	-					
10. White Racial Cat.	-.36***	.05	.13**	.04	-.03	.00	-.43***	-.06	-.54***	-				
11. Cultural Practices	.31***	.16***	.09*	.15***	.25***	.19***	.39***	.04	.53***	-.35***	-			
12. Bio. Difference	-.07	.10**	.35***	.17***	.03	.27***	.08*	.13**	.04	.04	.28***	-		
13. Racialism.	-.02	.09*	.34***	-.01	.04	.11**	.10**	.07	.10*	-.03	.18***	.28***	-	
14. Bio. Racial Essent.	-.03	.11**	-.01	.01	.06	.11**	.11**	.02	.16***	-.12**	.21***	.19***	.47***	-
M(*SD*)	54.59 (25.67)	3.82 (1.08)	1.69 (1.33)	3.84 (.94)	4.06 (.93)	3.50 (1.13)	7.25 (1.53)	3.27 (.84)	3.24 (1.06)	2.75 (1.08)	4.36 (1.08)	3.41 (1.15)	3.36 (1.03)	4.30 (.86)

Note. Gen. = Genetic; Cat. = Categorization; Bio. = Biological. Essent. = Essentialism

*$p < .05$ **$p < .01$ ***$p < .001$

Table IV. STUDY 2: CORRELATIONS (*r*), MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR MAIN STUDY VARIABLES

	Mean (Standard Deviation)													
Condition	Racial Ancestry			Biogeographical Ancestry			Racial Ancestry X Biogeographical Ancestry							
	1B/3W n = 216	3B/1W n = 210	Unspe. n = 221	25A/75E n = 225	50A/50/E n = 217	75A/25E n = 205	1B/3W X 25A/75E n = 74	1B/3W X 50A/50/E n = 73	1B/3W X 75A/25E n = 69	3B/1W X 25A/75E n = 60	3B/1W X 50A/50/E n =70	3B/1W X 75A/25E n =80	Unspe. X 25A/75E n =91	Unspe. X 50A/50/E n =74
Dependent Measure:														
Black Gen. Overlap	56.21 (27.63)	58.32 (23.72)	49.46 (24.64)	42.91 (27.38)	54.30 (21.58)	67.71 (21.02)	49.01 (30.58)	53.95 (25.83)	66.34 (23.19)	48.44 (27.22)	54.64 (19.35)	68.94 (20.20)	34.30 (22.27)	54.33 (19.12)
Pain Tolerance	3.79 (1.04)	3.83 (1.08)	3.85 (1.13)	3.90 (1.04)	3.76 (1.09)	3.81 (1.12)	3.88 (1.03)	3.65 (1.12)	3.83 (.97)	3.85 (.97)	3.87 (1.05)	3.78 (1.19)	3.95 (1.11)	3.77 (1.11)
Physicality	1.56 (1.21)	1.89 (1.46)	1.63 (1.31)	1.76 (1.38)	1.57 (1.24)	1.75 (1.37)	1.70 (1.35)	1.56 (1.26)	1.42 (.97)	2.01 (1.39)	1.60 (1.31)	2.05 (1.60)	1.64 (1.38)	1.54 (1.16)
Mental Illness	3.93 (.89)	3.76 (1.00)	3.83 (.93)	3.83 (.92)	3.90 (.89)	3.79 (1.02)	3.78 (1.00)	4.11 (.79)	3.89 (.84)	3.75 (.88)	3.76 (.95)	3.76 (1.13)	3.92 (.88)	3.82 (.89)
Physical Illness	4.18 (.91)	4.06 (.96)	3.93 (.90)	3.96 (.95)	4.10 (.83)	4.12 (.98)	4.03 (1.09)	4.34 (.79)	4.18 (.81)	3.84 (.93)	4.05 (.81)	4.24 (1.07)	3.98 (.85)	3.92 (.86)
STD/STIs	3.56 (1.13)	3.41 (1.22)	3.51 (1.02)	3.44 (1.09)	3.56 (1.08)	3.50 (1.21)	3.40 (1.25)	3.72 (1.05)	3.55 (1.07)	3.35 (1.04)	3.33 (1.21)	3.53 (1.36)	3.53 (.99)	3.60 (.95)
Skin Tone	7.21 (1.46)	7.65 (1.60)	6.90 (1.44)	6.67 (1.41)	7.22 (1.25)	7.93 (1.65)	6.82 (1.38)	7.32 (1.30)	7.52 (1.62)	7.18 (1.54)	7.31 (1.30)	8.30 (1.67)	6.20 (1.20)	7.03 (1.15)
Stereotyping	3.20 (.87)	3.24 (.86)	3.38 (.78)	3.25 (.84)	3.28 (.80)	3.30 (.88)	3.16 (.95)	3.22 (.83)	3.23 (.83)	3.28 (.80)	3.16 (.88)	3.27 (.91)	3.29 (.77)	3.45 (.69)
Black Racial Cat.	3.24 (.99)	3.60 (.99)	2.90 (1.06)	2.79 (1.01)	3.27 (.91)	3.70 (1.05)	2.92 (.99)	3.25 (.91)	3.57 (.97)	2.28 (.99)	3.54 (.93)	3.91 (1.00)	2.37 (.86)	3.04 (.85)
White Racial Cat.	2.81 (1.04)	2.42 (1.01)	3.01 (1.11)	3.21 (1.12)	2.74 (.89)	2.26 (1.02)	3.07 (1.11)	2.79 (.95)	2.56 (1.01)	2.78 (1.11)	2.49 (.86)	2.09 (.99)	3.62 (.99)	2.94 (.81)
Cultural Practices	4.38 (.98)	4.62 (1.02)	4.09 (1.11)	4.08 (1.12)	4.32 (1.03)	4.70 (.98)	4.24 (1.11)	4.42 (.87)	4.49 (.93)	4.46 (1.00)	4.49 (1.12)	4.86 (1.05)	3.70 (1.10)	4.08 (1.06)
Bio. Difference	3.36 (1.23)	3.45 (1.10)	3.42 (1.13)	3.51 (1.14)	3.34 (1.11)	3.36 (1.21)	3.45 (1.28)	3.39 (1.09)	3.23 (1.30)	3.50 (.94)	3.34 (1.15)	3.49 (1.17)	3.57 (1.15)	3.29 (1.10)

Note. Unspe. = Unspecified; Gen. = Genetic; Cat. = Categorization; Bio. = Biological

Table V. STUDY 2: MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR MAIN DEPENDANT VARIABLES BY CONDITION

Primary analyses: Biogeographic and racial ancestry effects. Using two-way ANOVAs, I tested the effect of biogeographical ancestry, racial ancestry, and their interaction on each outcome measure. Unless specified otherwise, independently controlling for biological racial essentialism and racialism did not change result patterns (see Appendix G).

Black genetic overlap. Results revealed a main effect racial ancestry, $F(2, 638) = 3.06$, $p = .048$, $\eta_p^2 = 0.01$. Specifically, planned contrasts revealed that participants reported the unspecified target shared less genetic material with Black people than both the 1B/3W and 3B/1W targets, $p = .056$ and $p = .022$, respectively.

Results also revealed a main effect of biogeographical ancestry effect, $F(2, 638) = 54.27$, $p < .001$, $\eta_p^2 = 0.15$. Specifically, planned contrast revealed that participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared less genetic material with Black people than the 75A/25E target, $ps < .001$.

Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 638) = 3.34$, $p = .010$, $\eta_p^2 = 0.02$. Specifically, planned contrasts showed that when the target's racial ancestry was unspecified, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared less genetic material with Black people than the 75A/25E target, $ps < .001$: $F(2, 638) = 48.61$, $p < .001$. Planned contrasts also revealed that when the target had 1B/3W racial ancestry, participants reported the 50A/50E target shared genetic material with Black people no differently from the 25A/75E target, $p = .265$, but shared less genetic material with Black people than the 75A/25E target, $p < .001$: $F(2, 638) = 7.87$, $p < .001$. Last, planned contrasts revealed that when the target had 3B/1W racial ancestry, participants reported the 50A/50E target shared genetic material with Black people no

differently from the 25A/75E target, $p = .114$, but shared less genetic material with Black people than the 75A/25E target, $p < .001$: $F(2, 638) = 16.09$, $p < .001$ (see figure 19).

In addition, when the target had 25A/75E biogeographical ancestry, participants reported that the unspecified target shared less genetic material with Black people than both the 1B/3W and 3B/1W targets, $p < .001$ and $p = .002$, respectively: $F(2, 638) = 8.03$, $p < .001$. However, there were no differences across racial ancestries when the target had 50A/50E or 75A/25E biogeographical ancestry, $F(2, 638) = .02$, $p = .982$, and $F(2, 638) = .28$, $p = .756$, respectively. (see figure 19).

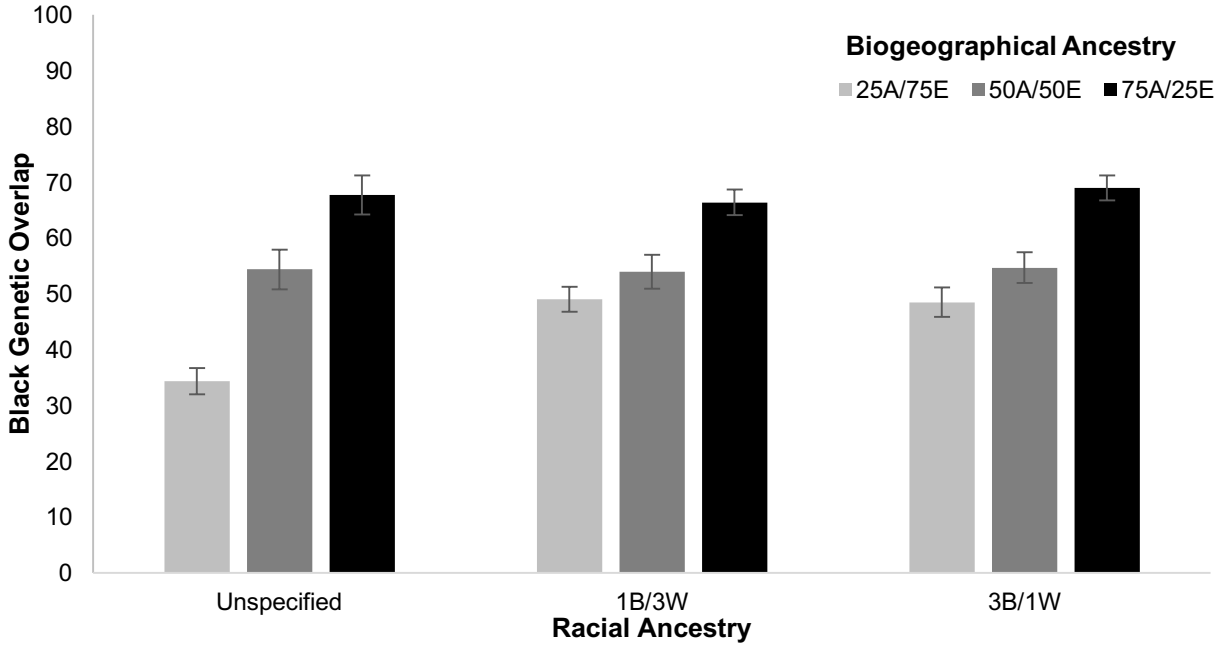


Figure 19. Effect of Biogeographical ancestry and Racial Ancestry on Black Genetic Overlap.

Complex Racial Categorization. *Black racial categorization*. Results revealed a main effect of biogeographical ancestry, $F(2, 638) = 38.81$, $p < .001$, $\eta_p^2 = 0.11$. Specifically, planned contrast reveal that participants racially categorized the unspecified target as less Black than both the 3B/1W and 1B/3W targets, $p = .001$ and $p = .006$, respectively. Results also revealed a a main effect of racial ancestry, $F(2, 638) = 19.49$, $p < .001$, $\eta_p^2 = 0.06$. Specifically, planned

contrast reveal that participants racially categorized the 50A/50E target as more Black than 25A/75E, and less Black than the 75A/25E target, *ps* < .001. However, inconsistent with hypotheses, results revealed no biogeographical by racial ancestry interaction, $F(4, 638) = 2.11$, $p = .078$, $\eta_p^2 = 0.01$[12].

White racial categorization. Results revealed a main effect of racial ancestry, $F(2, 637) = 11.79$, $p < .001$, $\eta_p^2 = 0.04$. Specifically, planned contrast revealed that participants racially categorized the unspecified target as White no differently from the 1B/3W target, $p = .349$, but more White than the 3B/1W target, $p < .001$.

Results also revealed a main effect of biogeographical ancestry, $F(2, 637) = 43.11$, $p < .001$, $\eta_p^2 = 0.12$. Specifically, planned contrast reveal that participants racially categorized the 50A/50E target as more White than 25A/75E, but less White than the 75A/25E target, *ps* < .001.

Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 637) = 4.96$, $p = .001$, $\eta_p^2 = 0.03$. Specifically, planned contrasts showed that when the target's racial ancestry was unspecified, participants racially categorized the 50A/50E target as less White than the 25A/75E target, but more White than the 75A/25E target, *ps* < .001: $F(2, 637) = 43.46$, $p < .001$. Planned contrasts also revealed that when the target had 1B/3W racial ancestry, participants racially categorized the 50A/50E no differently from both the 25A/75E and 75A/25E targets, $p = .098$ and $p = .182$, respectively: $F(2, 637) = 4.47$, $p = .013$. Last, planned contrasts revealed that when the target had 3B/1W racial ancestry, participants racially categorized the 50A/50E target as White no differently from the 25A/75E target, $p = .097$, but more White than the 75A/25E target, $p = .016$: $F(2, 637) = 8.40$, $p < .001$ (See Figure 20).

[12] Biogeographical ancestry x racial ancestry interaction was significant when controlling for biological racial essentialism (see Appendix G).

In addition, when the target had 25A/75E biogeographical ancestry, participants racially categorized the unspecified target as more White than both the1B/3W and 3B/1W targets, $p <$ .001 and $p < .002$, respectively: $F(2, 637) = 12.27$, $p < .001$. When the target had 50A/50E biogeographical ancestry, participants racially categorized the unspecified target as White no differently from the 1B/3W target, $p = .295$, but more White than the 3B/1W target, $p = .002$: $F(2, 637) = 4.98$, $p = .008$. Last, when the target had 75A/25E biogeographical ancestry, participants racially categorized the unspecified target as more White than 1B/3W target, $p =$ .017, but no differently from the 3B/1W target, $p = .858$: $F(2, 637) = 4.65$, $p = .011$ (See Figure 20).

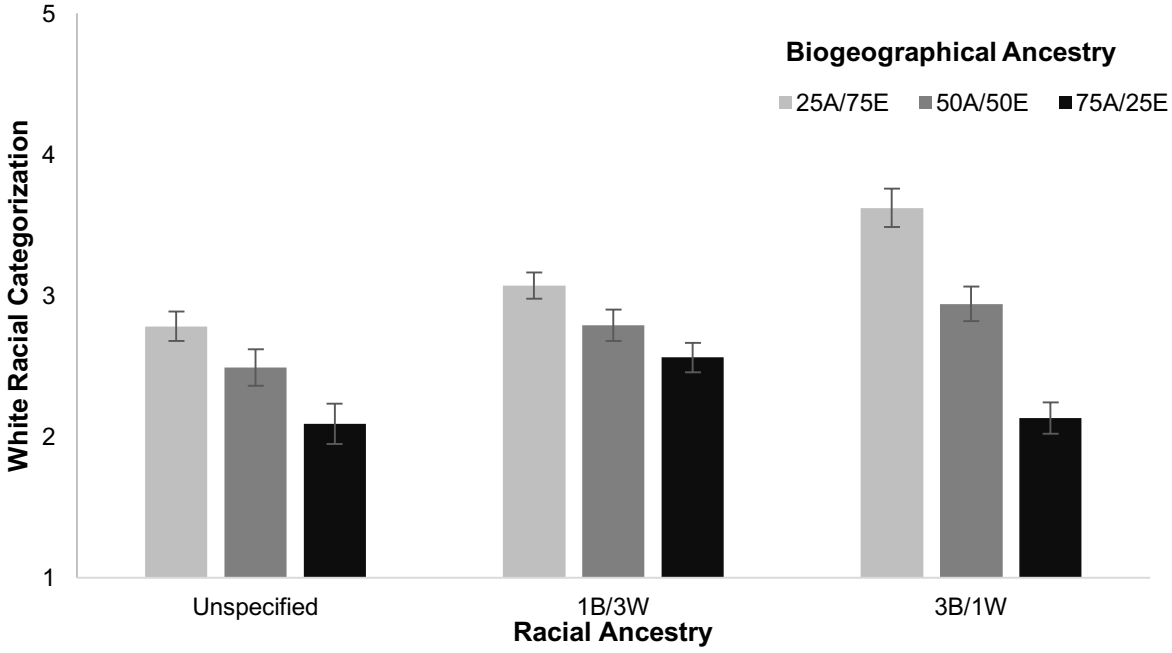


Figure 20. Effect of Biogeographical ancestry and Racial Ancestry on White Racial Categorization.

Cultural Practices. Results revealed a main effect of racial ancestry, $F(2, 638) = 9.18$, $p < .001$, $\eta_p^2 = 0.03$. Specifically, planned contrast revealed that participants believed that the

unspecified target engaged in Black-associated cultural practices less than both the 1B/3W and 3B/1W targets, $p = .034$ and $p < .001$, respectively.

Results also revealed a main effect of biogeographical ancestry, $F(2, 638) = 16.15$, $p < .001$, $\eta_p^2 = 0.05$. Specifically, planned contrast revealed that participants believed that the 50A/50E target engaged in Black-associated cultural practices more than the 25A/75E target, $p < .001$, but less than the 75A/25E target, $p = .047$.

Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 638) = 3.07$, $p = .016$, $\eta_p^2 = 0.02$. Specifically, planned contrasts showed that when the target's racial ancestry was unspecified, participants believed that the 50A/50E target engaged in Black-associated cultural practices more than the 25A/75E target, $p = .020$, but less than the 75A/25E target, $p < .001$: $F(2, 638) = 17.08$, $p < .001$. Planned contrasts revealed that when the target had 3B/1W racial ancestry, participants believed the 50A/50E target engaged in Black-associated cultural practices no differently from the 25A/75E target, $p = .872$, but less than the 75A/25E target, $p = .032$: $F(2, 638) = 3.34$, $p = .037$. Last, planned contrasts revealed no differences across biogeographical ancestry when the target had 1B/3W racial ancestry, $F(2, 638) = 1.28$, $p = .281$ (See figure 21).

In addition, when the target had 25A/75E biogeographical ancestry, participants believed that the unspecified target engaged in Black-associated cultural practices more than both the 1B/3W and 3B/1W targets, $ps < .001$: $F(2, 638) = 10.13$, $p < .001$. When the target had 50A/50E biogeographical ancestry, participants believed that the unspecified target engaged in Black-associated cultural practices more than both the 1B/3W and 3B/1W targets, $p = .048$ and $p = .040$, respectively: $F(2, 638) = 3.26$, $p = .018$: $F(2, 638) = 3.26$, $p = .040$. However, there were

no differences across racial ancestries when the target had 75A/25E biogeographical ancestry, $F(2, 638) = 2.68$, $p = .071$ (See figure 21).

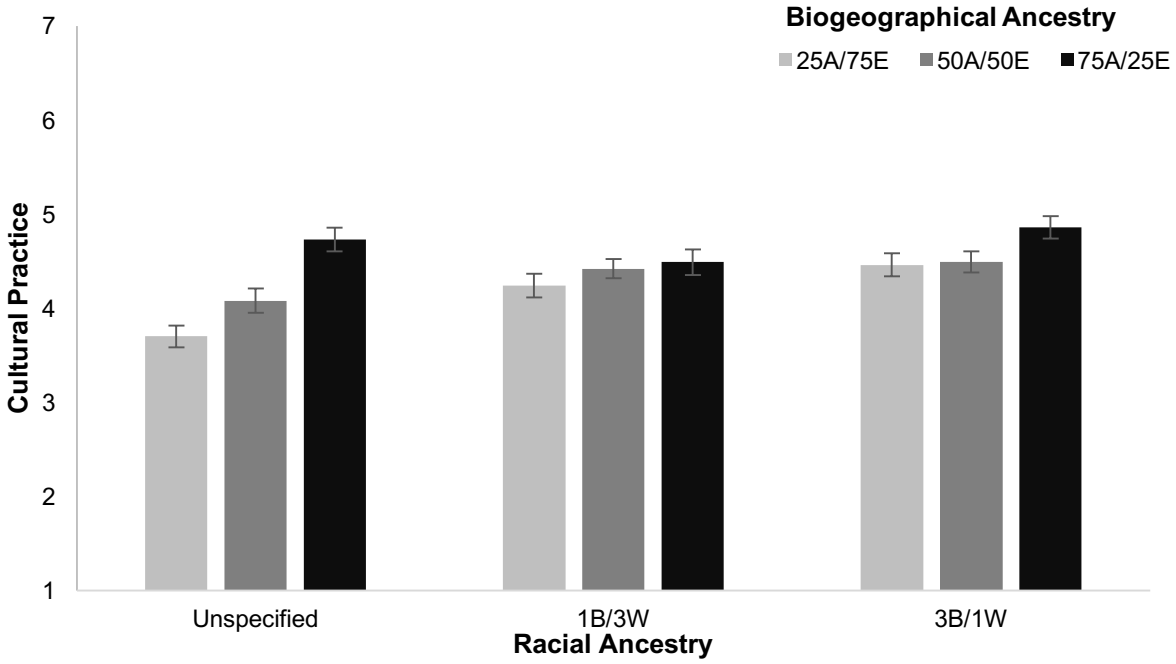


Figure 21. Effect of Biogeographical ancestry and Racial Ancestry on Cultural Practice.

Skin tone. Results revealed a main effect of racial ancestry, $F(2, 638) = 8.56$, $p < .001$, $\eta_p^2 = 0.03$. Specifically, planned contrast revealed that participants rated the unspecified target's skin tone no differently from the 1B/3W, $p = .185$, but lighter than the 3B/1W target**,** $p < .001$.

Results also revealed a main effect of biogeographical ancestry, $F(2, 638) = 36.04$, $p < .001$, $\eta_p^2 = 0.10$. Specifically, planned contrast revealed that participants rated the 50A/50E target as having a darker skin tone than 25A/75E target, but a lighter skin tone than the 75A/25E target, *ps* < .001.

Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 638) = 3.27$, $p = .012$, $\eta_p^2 = 0.02$. Specifically, planned contrasts showed that when the target's racial ancestry was unspecified, participants reported that the 50A/50E had a darker skin tone than the 25A/75E target, but a lighter skin tone than the 75A/25E target, *ps* < .001: $F(2, 638) =$

30.85, $p < .001$. Planned contrasts also revealed that when the target had 1B/3W racial ancestry, participants reported that the 50A/50E had a darker skin tone than the 25A/75E target, $p = .040$, but a skin tone no different from the 75A/25E target, $p = .393$: $F(2, 638) = 4.49$, $p = .012$. Last, planned contrasts revealed that when the target had 1B/3W racial ancestry, reported that the 50A/50E target's skin tone was no different from the 25A/75E target, $p = 0.624$, but lighter than the 75A/25E target's skin tone, $p < .001$: $F(2, 638) = 11.88$, $p < .001$ (See figure 22).

In addition, when the target had 25A/75E biogeographical ancestry, participants rated the unspecified as having a lighter skin tone than both the 1B/3W and 3B/1W targets, $p = .003$ and $p < .001$, respectively: $F(2, 638) = 10.35$, $p < .001$. When the target had 75A/25E biogeographical ancestry, participants rated the unspecified as having a lighter skin tone than the 1B/3W target, $p = .051$, but rated the 3B/1W target as having a darker skin tone, $F(2, 638) = 4.27$, $p = .015$. However, there were no differences across racial ancestries when the target had 50A/50E biogeographical ancestry, $F(2, 638) = 1.29$, $p = .277$ (See figure 22).

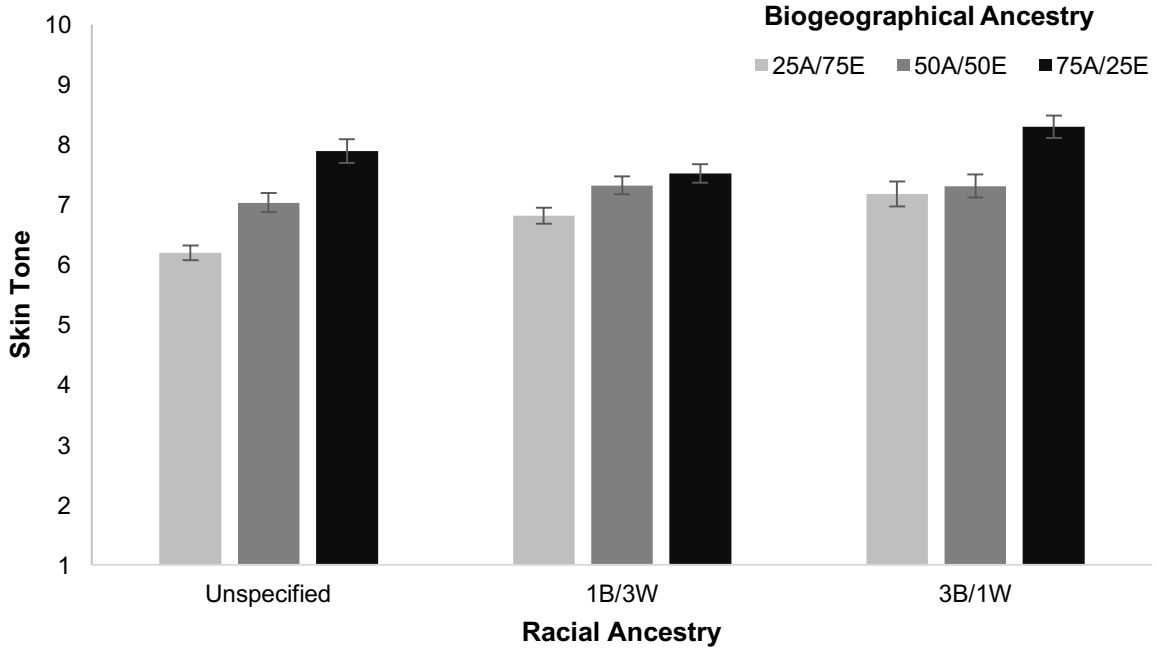


Figure 22. Effect of Biogeographical ancestry and Racial Ancestry on Skin Tone.

Racial Stereotyping. Inconsistent with hypotheses, results revealed no main effect of biogeographical ancestry, $F(2, 636) = 0.29$, $p = .752$, $\eta_p^2 < 0.01$, no main effect of racial ancestry, $F(2, 636) = 2.95$, $p = .053$, $\eta_p^2 = 0.01$, or their two-way interaction, $F(4, 636) = 0.47$, $p = .757$ $\eta_p^2 < 0.01$.

Superhumanization. *Physicality***.** Results revealed a main effect of racial ancestry, $F(2, 638) = 3.54$, $p = .030$, $\eta_p^2 = 0.01$. Specifically, inconsistent with hypotheses, planned contrast revealed no differences in reported physicality between the unspecified target and the 1B/3W and 3B/1W targets, $p = .053$ and $p = .541$, respectively[13]. Also inconsistent with hypotheses, results revealed no main effect of biogeographical ancestry, $F(2, 638) = 1.56$, $p = .210$, $\eta_p^2 < 0.01$, or a racial ancestry by biogeographical ancestry interaction, $F(4, 638) = 1.08$, $p = .368$, $\eta_p^2 < 0.01$.

Pain tolerance. Inconsistent with hypotheses, results revealed no main effect of racial ancestry, $F(2, 638) = 0.16$, $p = .856$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 638) = 0.81$, $p = .444$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 638) = 0.37$, $p = .834$, $\eta_p^2 < 0.01$.

Illness/disease perceptions. *Physical illness*. Results revealed a main effect of racial ancestry, $F(2, 637) = 4.48$, $p = .012$, $\eta_p^2 = 0.01$, Specifically, planned contrast revealed that participants rated the unspecified target's as more susceptible to physical illnesses than 1B/3W, $p = .003$, but no differently from the 3B/1W target**,** $p = .166$. However, inconsistent with hypotheses, results revealed no main effect of biogeographical ancestry, $F(2, 637) = 1.91$, $p = .149$, $\eta_p^2 = 0.01$. or a biogeographical ancestry by racial ancestry interaction, $F(4, 637) = 1.99$, $p = .094$ $\eta_p^2 = 0.01$.

[13] Main effect of racial ancestry become non-significant when controlling for racialism, $F(2, 637) = 2.40$, $p = .092$, $\eta_p^2 = 0.01$.

Mental illness. Inconsistent with hypotheses, results revealed no main effect of racial ancestry, $F(2, 637) = 1.81$, $p = .165$, $\eta_p^2 = 0.01$, no main effect of biogeographical ancestry, $F(2, 637) = 0.81$, $p = .448$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 637) = 1.30$, $p = .270$, $\eta_p^2 = 0.01$.

STD/I. Inconsistent with hypotheses, results revealed no main effect of racial ancestry, $F(2, 637) = 0.97$, $p = .381$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 637) = 0.69$, $p = .502$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 637) = 1.05$, $p = .383$, $\eta_p^2 = 0.01$.

Biological Difference. Results revealed no main effect of racial ancestry, $F(2, 638) = 0.97$, $p = .335$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 638) = 1.35$, $p = .261$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 638) = 0.49$, $p = .740$, $\eta_p^2 < 0.01$.

Moderated-mediations: The indirect effect of biogeographical ancestry X racial ancestry interaction through Black genetic overlap and Racial Categorization

Next, I tested whether the biogeographical ancestry by racial interaction was carried to the target judgments and evaluations through perceptions of the target's "race genes", and through perceptions of the target's "race genes" and racial categorization in serial. I tested these mediations using moderated mediation procedures: Model Template 86 of PROCESS version 3.0 in SPSS, with 10,000 bootstrap resamples (Hayes 2018; see Figure 16 for conceptual model). I used effect coding to examine specific contrasts for biogeographical ancestry: D1 compared 25A/75E to 50A/50E (25A/75E = 1, 50A/50E = -1, 75A/25E = 0), and D2 compared 75A/25E to 50A/50E (25A/75E = 0, 50A/50E = -1, 75A/25E = 1). I also used effect coding to examine specific contrasts for racial ancestry: Z1 compared 1B/3W to Unspecified (Unspecified = -1, 1B/3W = 1, 3B/1W = 0), and Z2 compared 3B/1W to Unspecified (Unspecified = -1, 1B/3W = 0,

3B/1W = 1). Unless specified otherwise, independently controlling for biological racial essentialism and racialism did not change result patterns.

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and Black racial categorization

Cultural practice. Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and Black racial categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.052, -.009]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.07, *SE* = .02, 95%CI [-.100, -.041]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.061, -.020], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .02, 95%CI [-.040, .020]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with increased Black racial categorization, which in turn lead to increased beliefs that the target engaged in Black-associated cultural activities.

Also consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.046, -.006]. Specifically, consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.05, *SE* = .02, 95%CI [-

.090, -.022]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.03, *SE* = .01, 95%CI [-.054, -.011], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.032, .016] (see Figure 23 for path estimates).

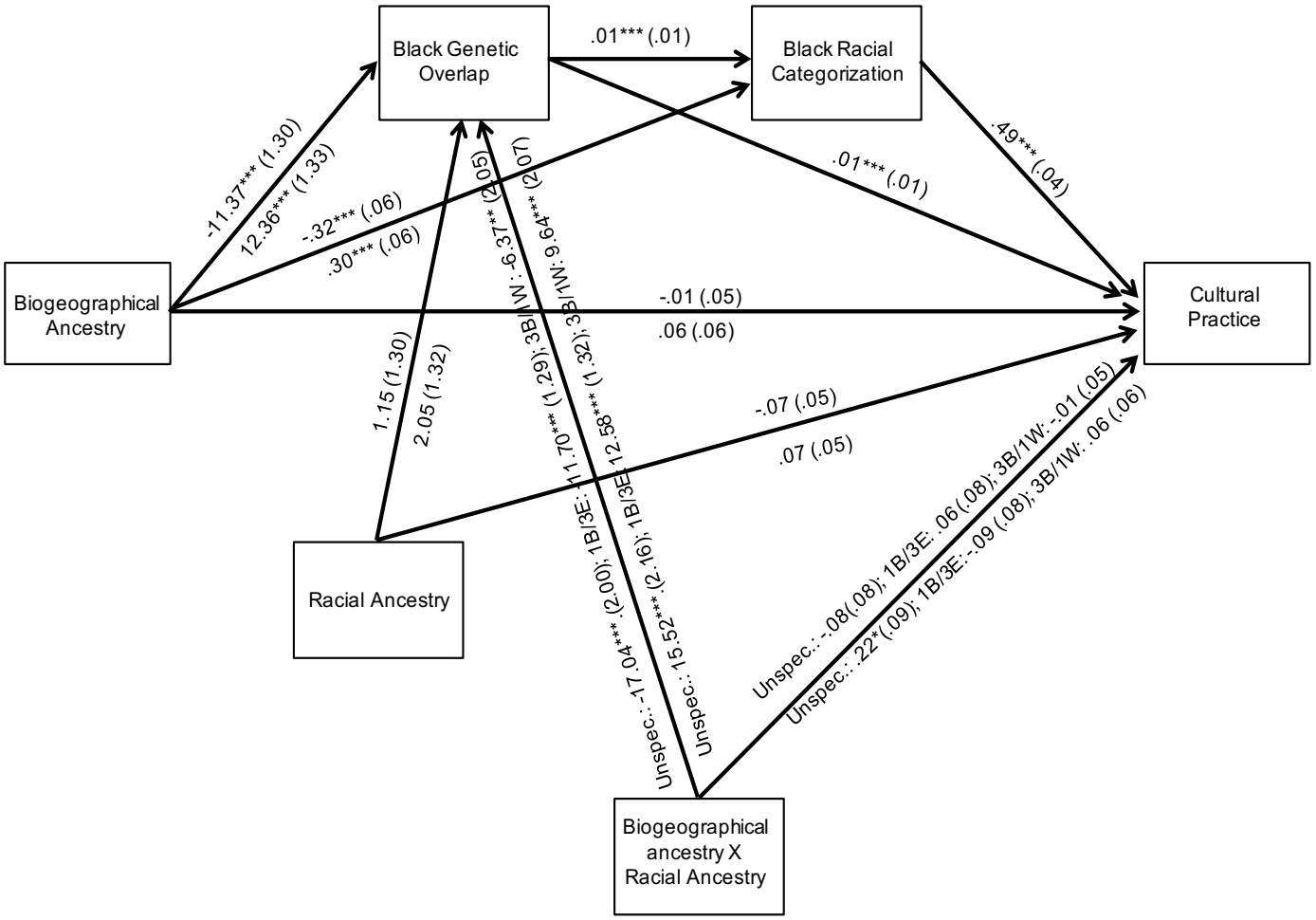


Figure 23. Indirect effect of biogeographical ancestry and racial ancestry on cultural practices through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on cultural practices. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001, **p < .01, ***p < .001

Skin tone. Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and Black racial categorization in serial, *index* = -.04, *SE* = .01, 95%CI [-.069, -.012]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.09, *SE* = .02, 95%CI [-.130, -.052]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.05, *SE* = .01, 95%CI [-.078, -.026], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .02, 95%CI [-.052, .027]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with increased Black racial categorization, which in turn lead to increased beliefs that the target had a darker skin tone.

Also consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap, *index* = -.05, *SE* = .02, 95%CI [-.097, -.016]. Specifically, consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.12, *SE* = .03, 95%CI [-.188, -.064]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.07, *SE* = .02, 95%CI [-.113, -.032], and absent when the target had 1B/3W racial ancestry, *index* = -.02, *SE* = .03, 95%CI [-.073, .036] (see Figure 24 for path estimates).

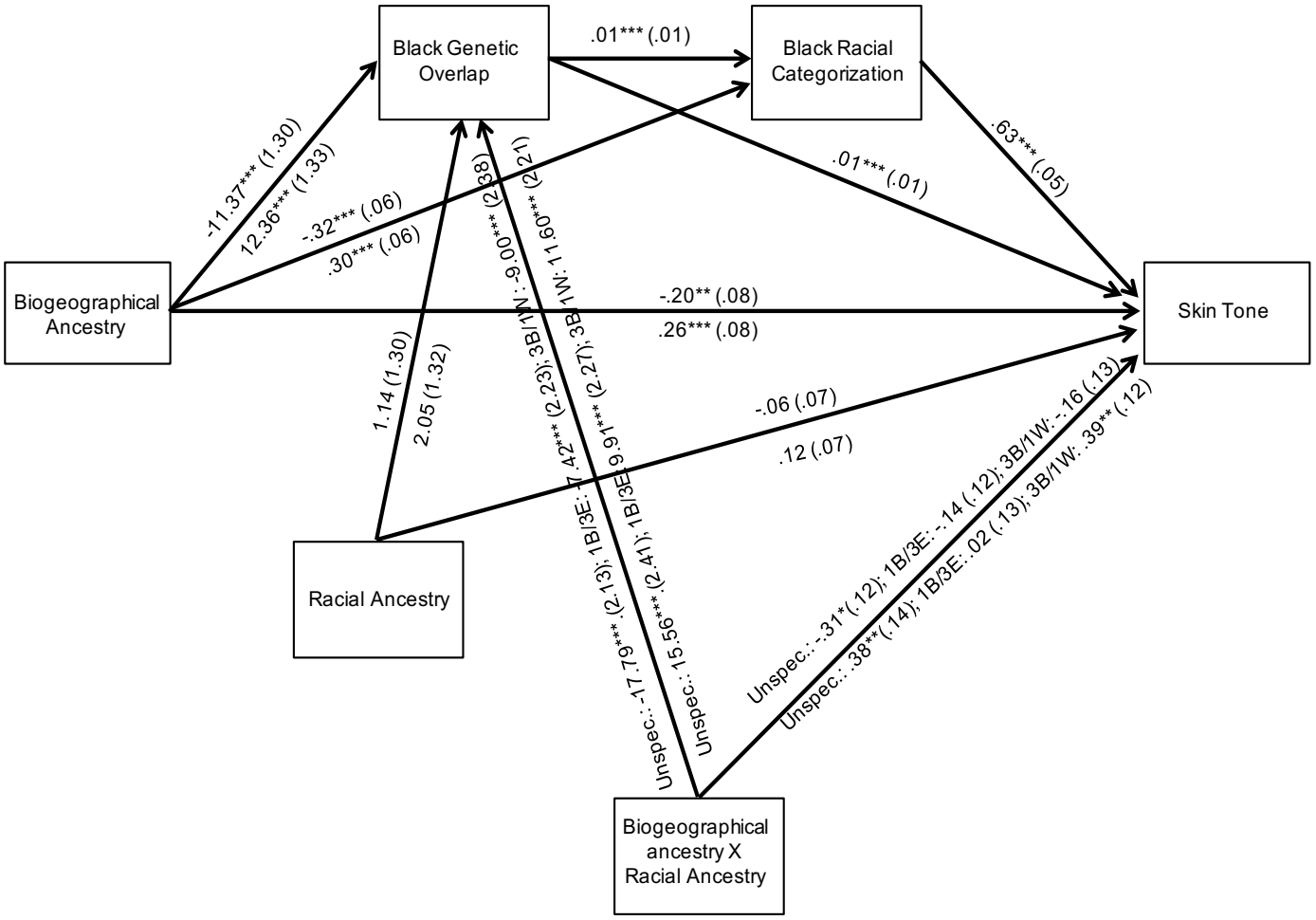


Figure 24. Indirect effect of biogeographical ancestry and racial ancestry on skin tone through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on skin tone. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001 **p < .01 ***p < .001

Racial stereotyping. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.003, .006]; or the

indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.025, .005].

Superhumanization. *Physicality.* Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.005, .008]; or the indirect effect of biogeographical ancestry on physicality through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.036, .008].

Pain tolerance. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.009, .002]; or the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.024, .010].

Illness/disease perceptions. *Physical Illness.* Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on perceptions of physical illness susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.037, -.001][14]. Specifically, consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .02, 95%CI [-.076, -.002]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.044, -.001], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.025, .012]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared

[14] Racial ancestry did not moderate the indirect effect of biogeographical ancestry on perceptions of physical illness susceptibility through Black genetic overlap when controlling for racialism, *index* = .00, *SE* = .01, 95%CI [-.025, .001].

genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with increased Black racial categorization, which in turn lead to increased beliefs that the target was susceptible to physical illness. However, inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.013, .000] (see Figure 25 for path estimates)**.**

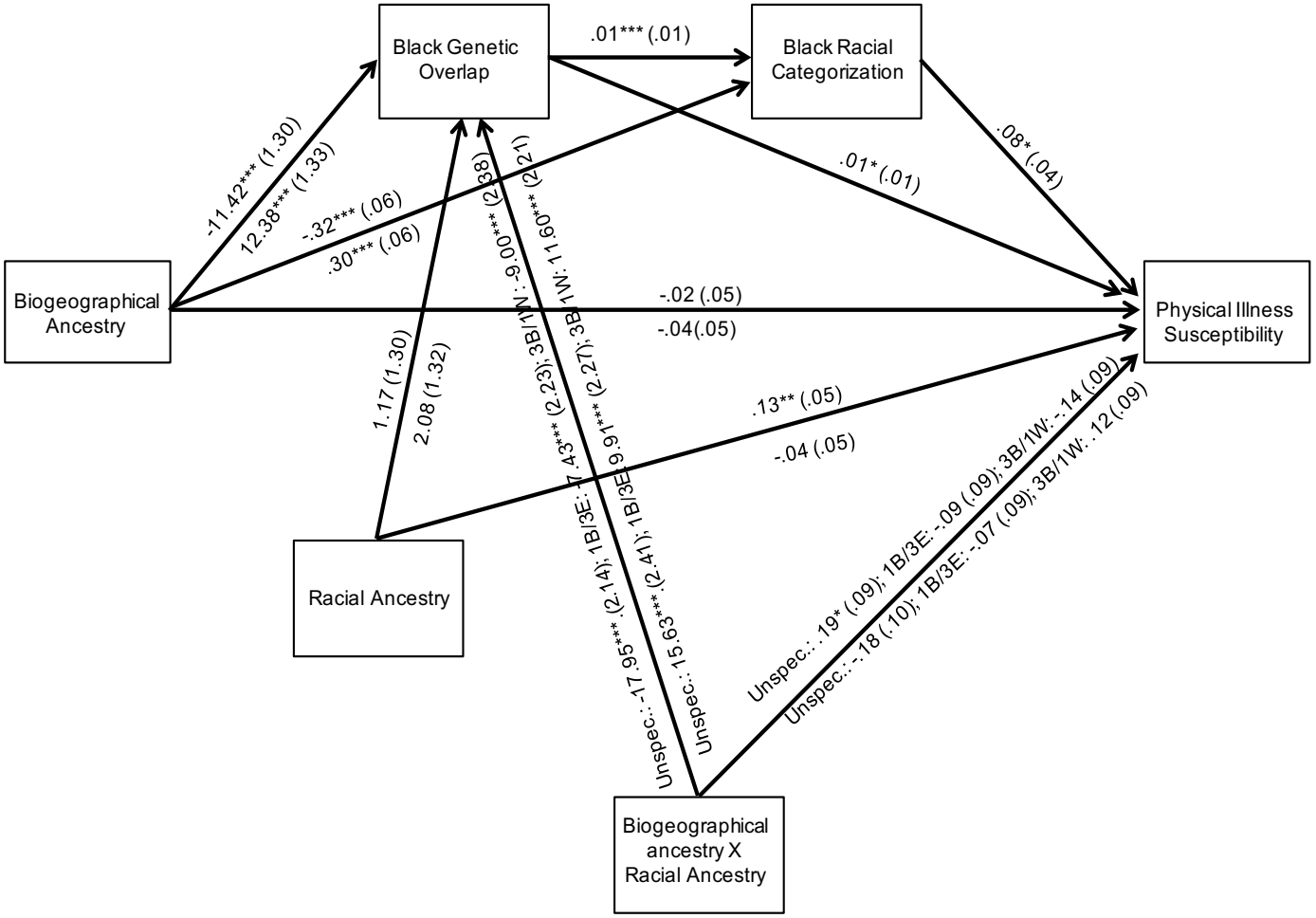


Figure 25. Indirect effect of biogeographical ancestry and racial ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -

1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on physical illness susceptibility. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001 **p < .01 ***p < .001

Mental Illness. Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on perceptions of mental illness susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.035, -.001]. Specifically, consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .02, 95%CI [-.071, -.003]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.042, -.002], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.025, .011]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with increased Black racial categorization, which in turn lead to increased beliefs that the target was susceptible to mental illness. However, inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.006, .006] (see Figure 26 for path estimates).

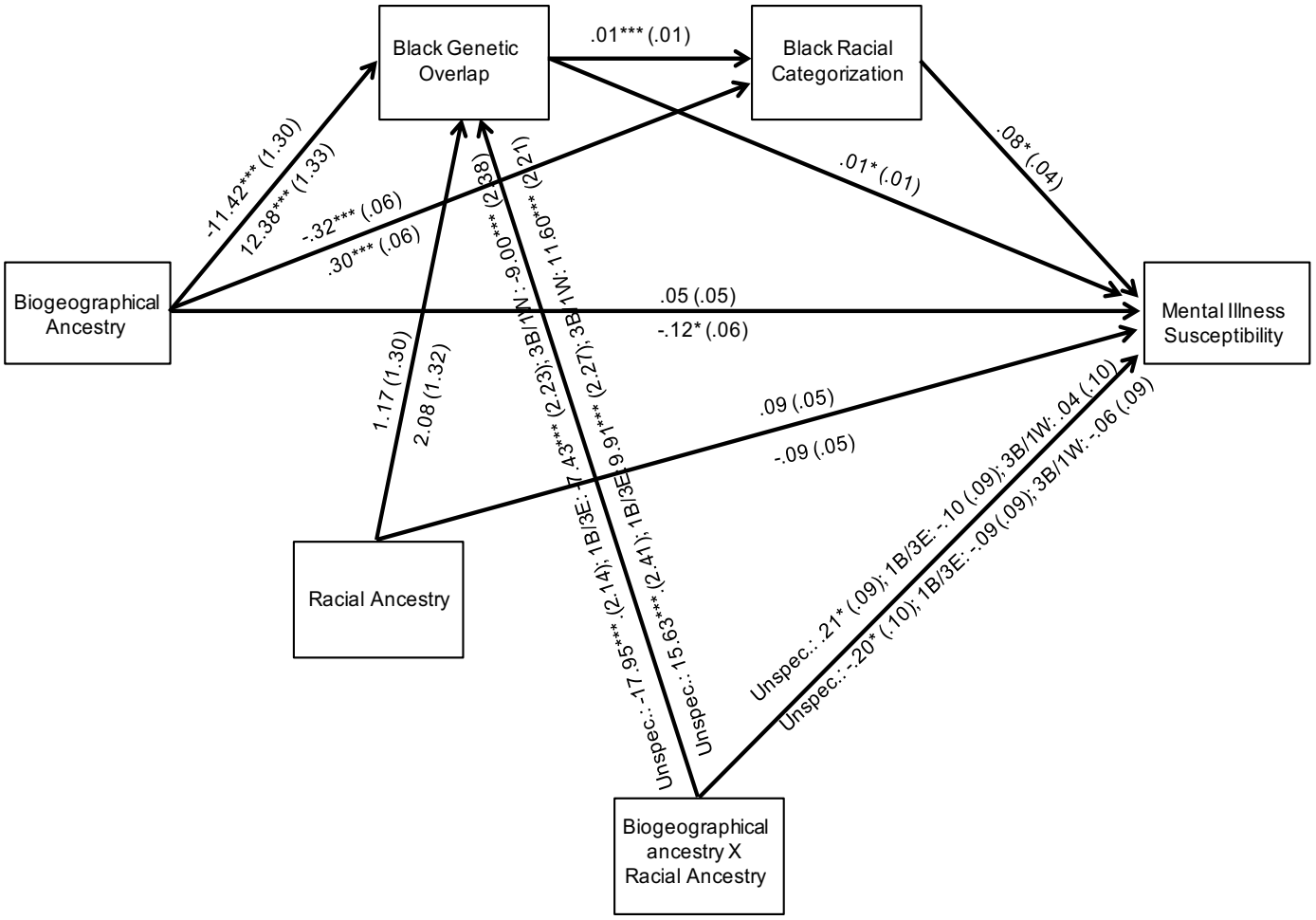


Figure 26. Indirect effect of biogeographical ancestry and racial ancestry on mental illness susceptibility through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on mental illness susceptibility. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001 **p < .01 ***p < .001

STD/I. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.014, .001]; or the indirect effect of

biogeographical ancestry on STD/I susceptibility through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.027, .011].

Biological difference. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and Black racial categorization in serial, *index* = -.01, *SE* = .00, 95%CI [-.014, .001]; or the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.004, .040].

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and White racial categorization

Cultural practice. Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and White racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.027, -.004]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .01, 95%CI [-.060, -.020]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.036, -.010], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.022, .011]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with increased White racial

categorization, which in turn lead to decreased beliefs that the target engaged in Black-associated cultural activities (see Figure 27 for path estimates).

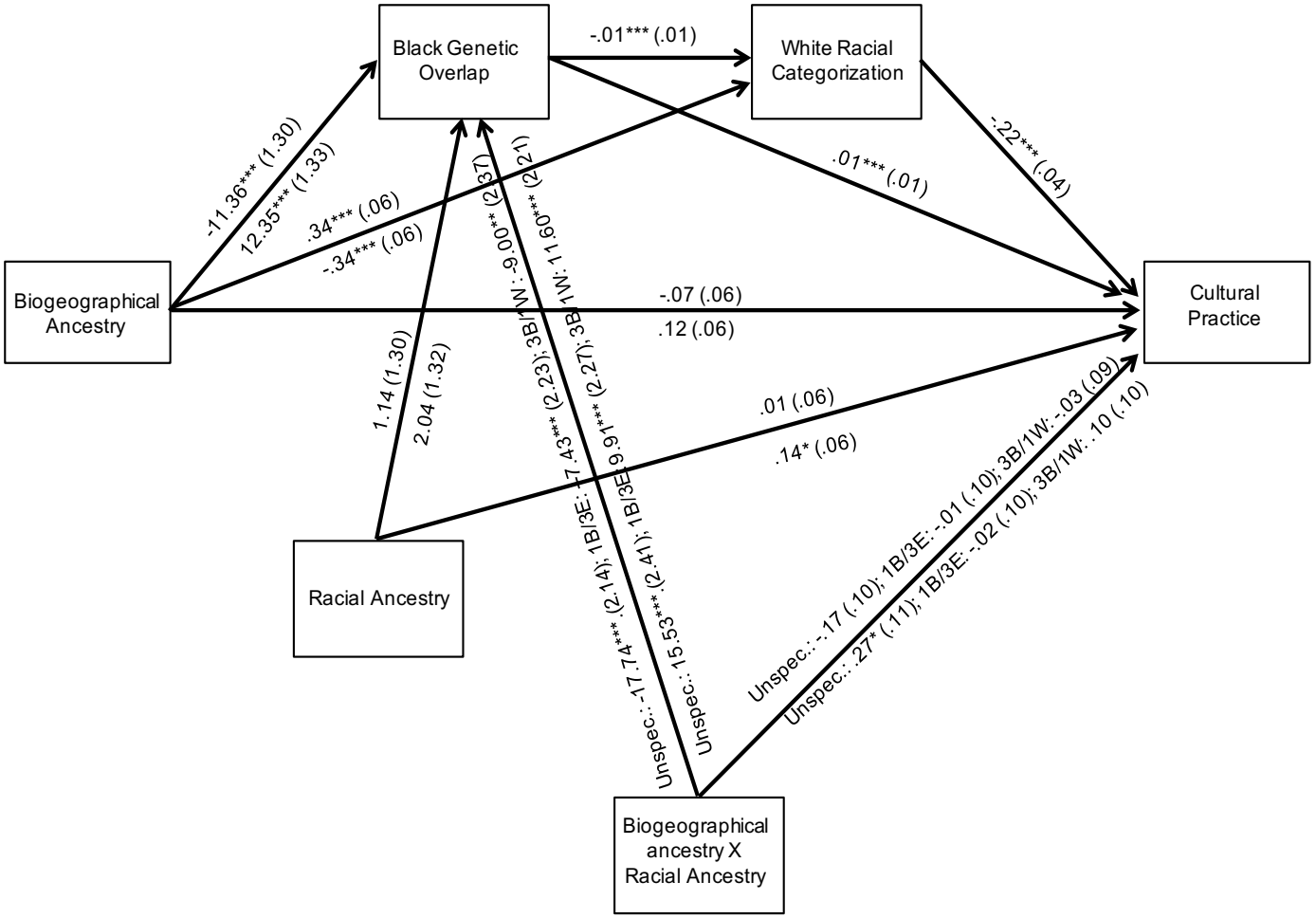


Figure 27. Indirect effect of biogeographical ancestry and racial ancestry on cultural practices through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on cultural practices. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001 **p < .01 ***p < .001

Skin tone. Consistent with hypotheses, racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and White racial

categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.053, -.008]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.06, *SE* = .02, 95%CI [-.100, -.036]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.060, -.019], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.040, .020]. In particular, when the target had 3B/1W or an unspecified racial ancestry, participants reported the 50A/50E target shared more genetic material with Black people than the 25A/75E target, but shared genetic material with Black people no differently from the 75A/25E target (there were no differences in reported shared genetic material with Black people when the target had 1B/3W). Increased reports that the target shared genetic material with Black people was associated with decreased White racial categorization, which in turn lead to increased beliefs that the target had a lighter skin tone (see Figure 28 for path estimates).

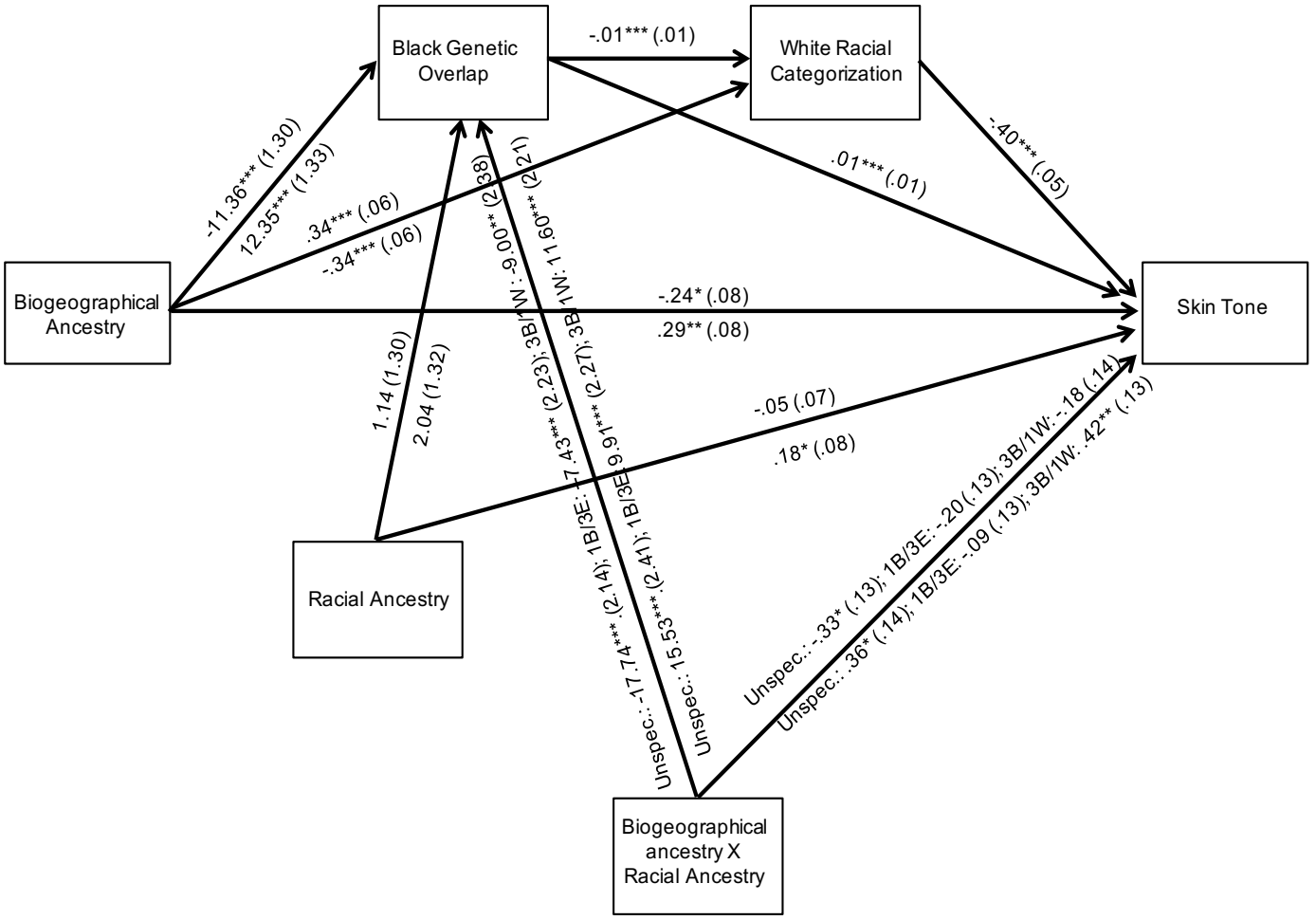


Figure 28. Indirect effect of biogeographical ancestry and racial ancestry on skin tone through Black genetic overlap and Black racial categorization. Biogeographical ancestry: Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line; Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Racial ancestry: Statistics for Z1 (1B/3W vs. Unspecified [Unspecified = -1, 1B/3W = 1, 3B/1W = 0]) are presented above the line; Statistics for Z2 (3B/1W vs. Unspecified [Unspecified = -1, 1B/3W = 0, 3B/1W = 1]) are presented below the line. Biogeographical Ancestry X Racial Ancestry: Statistics for D1 are presented above the line; Statistics for D2 are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on skin tone. All coefficients are unstandardized. Standard errors in parentheses. Unspec. = Unspecified Racial Ancestry condition.
*p <.001 **p < .01 ***p < .001

Racial stereotyping. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.008, .001].

Superhumanization. *Physicality.* Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.000, .022][15].

Pain tolerance. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.002, .010].

Illness/disease perceptions. *Physical Illness.* Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .007].

Mental Illness. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [.000, .011].

STD/I. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.005, .008].

Biological difference. Inconsistent with hypotheses, racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.006, .007].

Mediation: The indirect effect of biogeographical ancestry through genetic overlap and racial categorization.

[15] Racial ancestry moderated the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and White racial categorization in serial when independently controlling for racialism and biological racial essentialism (see Appendix G).

Last, where racial ancestry did not moderate the indirect effect of biogeographical ancestry Black genetic overlap, or through Black genetic overlap and racial categorization in serial, I conducted mediation analysis imputing biogeographical ancestry as the predictor and racial ancestry as a covariate. These tests examined the indirect effect of biogeographical ancestry on these outcomes. I conducted this analysis with Model Template 6 of PROCESS version 3.0 in SPSS, with 10,000 bootstrap resamples (Hayes 2018; see Figure 15 for conceptual model). I used effect coding to examine specific contrasts for biogeographical ancestry: D1 compared 25A/75E to 50A/50E (25A/75E = 1, 50A/50E = -1, 75A/25E = 0), and D2 compared 75A/25E to 50A/50E (25A/75E = 0, 50A/50E = -1, 75A/25E = 1). Unless specified otherwise, independently controlling for biological racial essentialism and racialism did not change result patterns.

Indirect effect of biogeographical ancestry through Black genetic overlap and Black racial categorization

Racial stereotyping. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.022, .005], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .008].

Superhumanization. *Physicality.* Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to physicality through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.049, .008], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.005, .011].

Pain tolerance. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01,

95%CI [-.031, .011], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.012, .003].

Illness/disease perceptions. *Physical Illness.* Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.031, .000].

Mental Illness. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.012, .015].

STD/I. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = .02, *SE* = .03, 95%CI [-.031, .085], or through Black genetic overlap and Black racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [-.001, .032].

Biological difference. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .02, *SE* = .01, 95%CI [-.002, .051], or through Black genetic overlap and Black racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.036, .004].

Indirect effect of biogeographical ancestry through Black genetic overlap and White racial categorization

Racial stereotyping. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.026, .011], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.011, .002].

Superhumanization. *Physicality.* Consistent with hypotheses, the indirect effect of biogeographical ancestry was carried to physicality through Black genetic overlap, *index* = -.03, *SE* = .02, 95%CI [-.069, -.003]. Consistent with predictions, this indirect effect was present when comparing the 50A/50E and 25A/75E targets, *index* = .03, *SE* = .01, 95%CI [.010, .044], and when comparing the 50A/50E and 75A/25E targets, *index* = -.03, *SE* = .01, 95%CI [-.047, -.011]. Specifically, participants reported that the 50A/50E target shared more genetic materials with Black people relative to the 25A/75E, and less genetic material with the 75A/25E target. Increased reporting that the target shared more genetic material with Black people was then associated with categorizing the target as less White. However, against predictions, increasingly categorizing the target as White was then related to increased beliefs that the target possessed superhuman physical abilities (see figure 29 for path estimates)

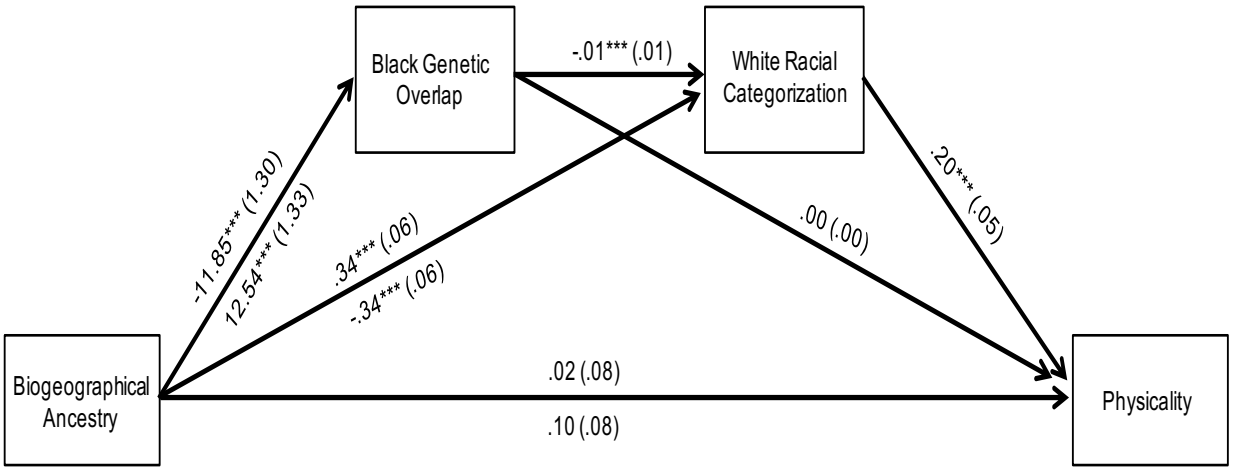


Figure 29. Indirect effect of biogeographical ancestry and racial ancestry on physicality susceptibility through Black genetic overlap and Black racial categorization. Statistics for D1 (25A/75E vs. 50A/50E [25A/75E = 1, 50A/50E = -1, 75A/25E = 0]) are presented above the line. Statistics for D2 (75A/25E vs. 50A/50E [25A/75E = 0, 50A/50E = -1, 75A/25E = 1]) are presented below the line. Reports direct effect of biogeographical ancestry and racial ancestry on physicality. All coefficients are unstandardized. Standard errors in parentheses.
*p <.001 **p < .01 ***p < .001

Pain tolerance. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.02, *SE* = .01,

95%CI [-.043, .004], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.002, .014].

Illness/disease perceptions. *Physical Illness.* Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.004, .010].

Mental Illness. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.000, .027].

STD/I. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.046, .005], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.005, .010].

Biological difference. Inconsistent with hypotheses, the indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.012, .041], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.007, .010].

2.3.3 Study 2 Discussion

Overall, results from Study 2 show that people use racial ancestry and biogeographical ancestry information, sometimes independently and sometimes simultaneously, to make race-based judgments about targets in ways that police the White racial border (Lewis, 2003). For example, when making a Black racial categorization of the target, participants independently used biogeographical and racial ancestry information. They did do in such a way as to racially

categorize the target as Black depending on which information (racial or biogeographical ancestry) communicated more Blackness. However, when making a White racial categorization of the target, participants used the biogeographical and racial ancestry information simultaneously. Across all racial ancestry conditions, participants categorized targets with more sub-Saharan biogeographical ancestry as less White, however, these differences appear greater when the target had more Black than White (3B/1W) racial ancestry. Thus, when categorizing a target as White, participants seem to use all available information simultaneously (compared to when making a Black categorization), possibly as a way to police Whiteness.

Further, results show that participants also used biogeographical and racial ancestry simultaneously when reporting the amount of genetic material that they believe the target shares with Black people. Specifically, when the target had 25A/75E biogeographical ancestry, participants reported that the unspecified target shared less genetic material with Black people than both the 1B/3W and 3B/1W targets; however, there were no differences across racial ancestries when the target had 50A/50E or 75A/25E biogeographical ancestry. It is conceivable, in agreement with empirical work on the "one-drop rule" or "hypodescent" (e.g., Guo et al., 2014; Ho et al., 2015), that, as the target's "Black blood" increased, participants paid less attention to the target's racial ancestry when making this judgment. As a consequence the potential for racial ancestry information to shift judgments about "race genes" become increasingly difficult with increasing biogeographical ancestry (similar to the experience Sgt. Brown; Eligon, 2017). Alternately, when the target has less "Black blood", participants seem to use racial ancestry to make judgments about the target's genetics, again, possibly as a way to protect and police the White racial border.

Additionally, results show that the interaction between racial and biogeographical ancestry on perceptions of "Black race genes" influenced other target-related judgments and evaluations. Specifically, when the target had 3B/1W or an unspecified racial ancestry (but not 1B/3W), increased reports that the target shared genetic material with Black people (between targets with 50A/50E and 25A/75E biogeographical ancestry) was associated with increased beliefs that the target (1) had a darker skin tone, (2) engaged in Black associated cultural practices, (3) was susceptible to physical illnesses, and (4) was susceptible to mental illnesses. Consistent with ideas about component racialism, these results suggest the extent to which people perceive a target to have "Black genes" leads to increased racialized judgments of the target, especially when the target's racial ancestry is unknown or unspecified (stronger mediation effect). This result conceptually replicates Study 1, along with previous work showing that people believe that racial genetic variations explain racial differences in illness/disease susceptibility (Condit et al., 2004a; Condit et al., 2004b; Institute of Medicine, 2003; Shields et al., 2005).

Inconsistent with hypotheses, however, there were no main effect of biogeographical ancestry, or a biogeographical X racial ancestry interaction on: racial stereotyping, illness/disease perception, superhumanization, or biological difference. For these outcomes, it is possible that participants perceive any target with any amount of Black racial ancestry (or "Black blood) as similar across these outcomes. Accordingly, for these outcomes, perhaps it is not the proportion of Black ancestry (racial, biogeographical) that influences judgments, but rather the fact of having any Black ancestry (i.e., "one drop") that makes people perceive an individual as indistinguishably Black. Similar reasoning can also explain the relevant, non-significant indirect effects. In addition, inconsistent with hypotheses, increased White racial categorization was

associated with increased beliefs that the target possessed superhuman abilities—although results showed, consistent with hypotheses, that Black genetic overlap and White racial categorization mediated the relationship between biogeographical ancestry and physicality in serial. In this instance, it is likely that participants perceived the physical superhuman outcomes as positive outcomes, which facilitated their associating it with Whiteness. Therefore, it is conceivable that the positive association between White categorization and physicality resulted from White participants making positive in-group attributions (Leyens et al., 2001; Stephan, 1977).

Last, this study presents more evidence supporting component racialism by showing that independently controlling for biological racial essentialism and racialism does not change conclusions. In addition, it shows, more directly than before, that people believe that the extent to which a target shared genetic material with Black people can predict several racialized outcomes. However, as with the previous two studies, this study tests ideas of component racialism by statistically controlling for more abstract biological conceptualizations of racial essences. Thus, to address this limitation, and to provide even stronger empirical support for component racialism, in the final study I manipulate the concreteness of racial conceptualization (i.e., biological essentialism, racialism, component racialism) to more directly test how it influences racialized perceptions and judgments. In addition, going beyond perceptions of singular individuals, in the following study I investigate how race conceptions influence racialized perceptions and judgments on the group level (i.e., perceptions of entire racial groups).

2.4 Study 3: Concreteness of biological race conceptions predict group-level judgments

With Study 3, I further investigate the idea of component racialism by manipulating—rather than controlling for—biological race conceptions (i.e., different forms of biological racial essentialism). In particular, I examine how, and the extent to which, the concreteness of

biological race conceptions influences judgments and evaluations at the racial-group level (see Figure 1). These manipulations try to capture the different levels of abstractness/concreteness of biological racial essentialism. Specifically, I examine how biological racial essentialist, racialist, and component racialist ideas influence perceptions of Black-White racial group differences (i.e., Black-White disease and illness rates; Black-White biological, genetic, and socio-behavioral differences), in addition to perceptions of Black people as a whole (e.g., racial stereotyping).

Following results from Studies 1 and 2, along with previous work (e.g., Shiloh et al., 2002; Williams & Eberhardt, 2008), I expect increasing concreteness of biological race conceptions to lead to greater beliefs that Black-White biological, genetic, and socio-behavioral differences are due to natural (vs. environmental causes). I also expect increasing concreteness of biological race conceptions to lead to similar beliefs concerning the differences in illness/diseases between Black and White people. Further, at the group level, I expect increasing concreteness of biological race conceptions to lead to greater racialized perceptions of Black people.

Last, I expect these relationships to be mediated by a "magnitude of essentialist bias". That is, I expect to find evidence that increasing concreteness of biological race conceptions will be associated with increased beliefs that Black and White people are genetically different, which in turn will lead to greater beliefs that Black-White differences (biological, socio-behavioral) are natural (vs. environmental). I also anticipate finding evidence that increasing concreteness of biological race conceptions will be associated with increased beliefs that Black and White people are genetically different, which in turn will increase racialized perceptions of Black people.

2.4.1 Method

2.4.1.1 Participants

Following participant recruitment methods, and post-hoc power analyses conducted in a Pilot Study (see Appendix E), I recruited 303 (53.1% female; M_{age}= 40.07 years, SD_{age} = 13.66) White U.S. citizen adults from MTurk.

2.4.1.2 Materials and Procedure

After providing informed consent for the web-based study, participants were randomly assigned to read one of three articles, each discussing research on a "recent scientific discovery". Participants were told that the purpose of the study was to develop materials for future research that aimed to "more effectively present scientific discoveries to the general population". In reality, the articles were used to shift (i.e., manipulate) participants' biological race conception. After reading each article, participants completed a series of race-related measures, along with some basic demographic questions.

Biological race conception manipulation. Participants were assigned to read one of three articles. Each article was examined for complexity, ease of reading, ease of understanding, engagement, interestingness, believability, and clarity, along with ability to shift biological race conception, in a previous Pilot Study (see Appendix E). The articles were adaptations of the "Scientists Reveal That Race Has No Genetic Basis, But Does Have a Biological One" article used in Williams & Eberhardt (2008) to shift people into a biological conception of race. In particular, I adapted the articles in a way that one article discussed the scientific discovery of a general *biological*, but not genetic, basis of race; this represented the *biological essentialism* condition. The other article discussed the scientific discovery of a *genetic* basis of race; this represented the *racialism* condition. The final article discussed the discovery of racial genetic differences; this represented the *component racialism* condition (see Appendix F for full articles).

Genetic difference. Participants rated the extent to which genetic differences between Black and White people predict racial differences (0 -100%).

Perceived difference. Following Williams and Eberhardt (2008), participants were prompted with: "There are racial disparities between Black and White Americans in this country in a number of areas (e.g., inequalities in education, employment, income). On many different dimensions, Black Americans do not do as well as White Americans." Following this prompt, participants completed the following measures:

*Illness/Disease perceptions***.** Using items from Study 2, participants reported the extent to which they think Black-White differences in mental illness (depression, post-traumatic stress disorder [PTSD], generalized anxiety disorder), physical illness (hypertension/high blood pressure, diabetes, stroke, obesity), and sexually transmitted diseases/infections ([STDs/STIs]; HIV/AIDS, gonorrhea, chlamydia, syphilis) rates are due to "environmental" or "natural" factors on a 7-point Likert scale (1 = *very likely environmental* to 7 = *very likely natural*). Items specific to each illness/disease category were averaged to create *physical illness*, *mental illness*, and *STD/I* measures, with higher values representing a greater belief that Black-White racial differences in the respective disease/illness category are due to "natural" causes ($\alpha_{\text{mental illness}}$ = .86; $\alpha_{\text{physical illness}}$ = .82; $\alpha_{\text{STD/STI}}$ = .93).

*Socio-behavioral difference***.** Participants reported the extent to which they perceived Black-White racial differences in (1) income, (2) education, (3) wealth, (4) athletic ability, (5) intelligence, and (6) criminal behavior resulted from "environmental" or "natural" factors on a 7-point Likert scale (1 = *very likely environmental* to 7 = *very likely natural*). All items were averaged, with higher values representing a greater belief that these Black-White socio-behavioral racial differences are due to "natural" causes (α = .87).

*Biological difference***.** Participants completed the "Biological difference" measure used in Study 1 and Study 2; however, they rated differences between "Black people" and "White people" ($\alpha = .94$).

Racial stereotyping. Same as Study 2; however, participants responded to the items with "Black people" as the focus ($\alpha = .95$).

Super-humanization. Same as Study 2; however, participants responded to the items with "Black people" as the focus ($\alpha_{\text{pain tolerance}} = .88$; $\alpha_{\text{physicality}} = .90$).

Cultural Practices. Same as Study 1 and Study 2; however, participants responded to the items with "Black people" as the focus ($\alpha = .81$).

Conceptual manipulation checks. To assess the extent to which the articles shifted biological race conceptions, participants completed the Racialism measure (Study 2; $\alpha = .72$), which assess endorsement of genetic views of race; and the Component racialism measure (2-item; see Appendix E), which captures beliefs that racial groups possess unique genetic compositions ($r = .55$).

Article evaluations. Participants rated the articles along three dimensions on 7-point bipolar Likert scales: (1) difficulty reading (1 = *extremely easy* to 7 = *extremely difficult*); (2) difficulty understanding (1 = *extremely easy* to 7 = *extremely difficult*); and (3) complexity (1 = *extremely simple* to 7 = *extremely complex*).

Reading comprehension/Attention checks. Participants responded to five reading comprehension questions that gauged their level of attention while reading the article. The reading comprehension questions, all multiple choice, included selecting: (1) the name of the interviewed scientist; (2) the interviewed scientist's university; (3) the scientific journal in which the research paper was published; (4) the researchers' other lines of work; (5) the name of the

gene mentioned in the article.

2.4.2 Results

Preliminary analyses. First**,** I conducted several one-way ANOVAs to investigate the extent to which the articles shifted biological race conceptions, and participants' recollection and evaluation of each article.

Article evaluations. *Difficulty reading***.** Results revealed no effect of article on reading difficulty, $F(2, 299) = .01$, $p = .993$, $\eta_p^2 < 0.01$ 01 (Component Racialism: $M = 2.71.$; $SD = 1.61$; Racialism: $M = 2.69$; $SD = 1.44$; Biological Essentialism: $M = 2.68$; $SD = 1.58$).

*Difficulty understanding***.** Results revealed no effect of article on difficulty to understand, $F(2, 297) = .78$ $p = .462$, $\eta_p^2 = 0.02$ (Component Racialism: $M = 2.54.$; $SD = 1.37$; Racialism: $M = 2.81$; $SD = 1.51$; Biological Essentialism: $M = 2.69$; $SD = 1.62$).

Complexity. Results revealed no effect of article on complexity, $F(2, 300) = .74$ $p = .476$, $\eta_p^2 = 0.01$ (Component Racialism: $M = 3.67.$; $SD = 1.60$; Racialism: $M = 3.93$; $SD = 1.67$; Biological Essentialism: $M = 3.69$; $SD = 1.67$).

Reading comprehension/Attention checks. I summed (total = 5) the number of correct responses to the reading comprehension questions and performed a one-way ANOVA to investigate differences in information recollection per each article. Scores were high, indicating satisfactory article comprehension and attention, and results revealed no condition effect on information recollection, $F(2, 300) = .56$, $p > .250$, $\eta_p^2 < 0.01$ (Component Racialism: $M = 3.82.$; $SD = 1.18$; Racialism: $M = 3.94$; $SD = 1.33$; Biological Essentialism: $M = 3.99$; $SD = 1.09$).

Conceptual manipulation checks. *Racialism***.** Results revealed a condition effect on racialist beliefs, $F(2, 300) = 12.26$, $p < .001$, $\eta_p^2 = 0.08$. Consistent with predictions, planned

contrast revealed that participants endorsed genetic essentialist views of race to a greater extent in the Component Racialism condition (M = 3.76.; SD = 1.13) and Racialism conditions (M = 3.84.; SD = 1.23) relative to the Biological racial essentialism condition (M = 3.12.; SD = 1.11), both $ps < .001$.

*Component racialism***.** Results revealed a condition effect on component racialist beliefs, $F(2, 300) = 11.65$, $p < .001$, $\eta_p^2 = 0.07$. Consistent with predictions, planned contrast revealed that participants endorsed component racialist ideologies to a greater extent in the Component Racialism (M = 3.90.; SD = 1.53) and Racialism (M = 3.85.; SD = 1.23) conditions relative to the Biological racial essentialism condition (M = 2.99.; SD = 1.54), both $ps < .001$. However, against predictions, there was no difference between the Component Racialism and Racialism conditions, $p = .842$.

Primary analyses. Next, I conducted several one-way ANOVAs to investigate the effect of biological race conception on the main dependent outcomes. Table VI presents correlations among main study variables, along with their means and standard deviations. Table VII presents the means and standard deviations of dependent variables as a function of experimental condition.

Condition	Mean (Standard Deviation)		
	Component Racialism n= 101	**Racialism** n= 91	**Biological Essentialism** n= 111
Genetic Difference	52.91 (93.57)	45.36 (34.56)	24.39 (31.44)
Physical Illness	4.00 (1.46)	3.79 (1.38)	3.54 (1.36)
Mental Illness	3.27 (1.47)	3.09 (1.42)	2.82 (1.24)
STD/I	2.61 (1.60)	2.61 (1.62)	2.36 (1.35)
Socio-Behavioral Difference	3.16 (1.45)	3.16 (1.36)	2.91 (1.30)
Biological Difference	3.04 (1.49)	2.92 (1.46)	2.72 (1.31)
Pain Tolerance	4.28 (1.14)	4.40 (1.23)	4.39 (1.09)
Physicality	2.00 (1.56)	2.12 (1.59)	1.92 (1.36)
Cultural Practice	4.82 (1.10)	4.86 (1.07)	4.79 (.97)
Racial Stereotyping	3.58 (1.01)	3.55 (1.01)	3.45 (1.08)

Table VI. STUDY 3: CORRELATIONS (*r*), MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR DEPENDANT VARIABLES

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
1. Genetic Diff.	-									
2. Physical Illness	.14*	-								
3. Mental Illness	.19**	.59***	-							
4. STD/I	.20***	.43***	.56***	-						
5. Socio-Behavioral Diff.	.21***	.47***	.55***	.49***	-					
6. Biological Diff.	.24***	.33***	.46***	.48***	.49***	-				
7. Pain Tolerance	-.02	.07	-.01	-.01	.00	-.10	-			
8. Physicality	.19**	.12*	.24***	.43***	.42***	.52***	.00	-		
9. Cultural Practice	.08	.18**	.15*	.02	.10	.17**	.17**	.01	-	
10. Racial Stereotyping	.04	.09	.11	.12*	.16**	-.09	.02	.02	.12*	-
M (*SD*)	40.08 (61.10)	3.78 (1.41)	3.05 (1.38)	2.51 (1.52)	3.07 (1.38)	2.89 (1.42)	4.36 (1.15)	2.00 (1.50)	4.82 (1.05)	3.52 (1.03)

Note. Diff. = Difference
*$p < .05$, **$p < .01$, ***$p < .001$

Table VII. STUDY 3: MEANS (*M*), AND STANDARD DEVIATIONS (*SD*) FOR MAIN DEPENDANT VARIABLES BY CONDITION

Genetic Difference. Consistent with hypotheses, results revealed an effect of biological race conception on genetic difference, $F(2, 298) = 6.37$, $p = .002$, $\eta_p^2 = 0.04$. Consistent with predictions, planned contrast revealed that participants were more likely to believe that Black-White genetic differences explained more differences between Black and White people in the Racialism condition relative to the Biological essentialism condition, $p = .014$; however, against predictions, there was no difference between the Component racialism and Racialism conditions, $p = .394$.

Illness/disease difference. *Physical illness*. Consistent with hypotheses, results revealed an effect of biological race conception on physical illness, $F(2, 300) = 2.78$, $p = .064$, $\eta_p^2 = 0.02$. Against predictions, there were no differences between the Component Racialism and Racialism conditions, $p = .309$, or between the Racialism and Biological essentialism conditions, $p = .215$. However, post-hoc analysis revealed that participants were more likely to believe that Black-White differences in physical illness rates were due to "natural" causes in the Component racialism condition relative to the Biological essentialism condition, $p = .019$.

Mental Illness. Consistent with hypotheses, results revealed an effect of biological race conception on mental illness, $F(2, 300) = 2.91$, $p = .056$, $\eta_p^2 = 0.02$. Against predictions, there were no differences between the Component Racialism and Racialism conditions, $p = .393$, or between the Racialism and the Biological essentialism conditions, $p = .151$. However, post-hoc analysis revealed that participants were more likely to believe that Black-White differences in physical illness rates were due to "natural" causes in the Component racialism condition relative to the Biological essentialism condition, $p = .018$.

STD/I. Inconsistent with hypotheses, results revealed no effect of biological race conception on STD/I, $F(2, 300) = .99$, $p = .372$, $\eta_p^2 = 0.01$.

Socio-behavioral difference. Inconsistent with hypotheses, results revealed no effect of biological race conception on racial difference, $F(2, 300) = 1.17$, $p = .310$, $\eta_p^2 = 0.01$.

Biological Difference. Inconsistent with hypotheses, results revealed no effect of biological race conception on biological difference, $F(2, 300) = 1.44$, $p = .239$, $\eta_p^2 = 0.01$.

Superhumanization. Inconsistent with hypotheses, results revealed no effect of biological race conception on superhumanization: *Pain tolerance*: $F(2, 300) = .37$, $p = .693$, $\eta_p^2 < 0.01$; *Physicality*: $F(2, 299) = .45$, $p = .636$, $\eta_p^2 < 0.01$.

Cultural Practices. Inconsistent with hypotheses, results revealed no effect of biological race conception on cultural practice, $F(2, 299) = .13$, $p = .88$, $\eta_p^2 < 0.01$.

Racial Stereotyping. Inconsistent with hypotheses, results revealed no effect of biological race conception on racial stereotyping, $F(2, 300) = .44$, $p = .643$, $\eta_p^2 < 0.01$.

Mediation analyses: Black-White genetic difference as a mediator. Next, I tested the extent to which perceptions of Black-White genetic difference mediated the relationship between race conception and the other dependent outcomes using mediation procedures with Model Template 4 of the SPSS macro PROCESS version 2.16 and 10,000 bias-corrected bootstrap resamples, where a 95% confidence interval (CI) excluding zero indicates a significant effect at the $p < .05$ level (Hayes, 2012; 2016). I used effect coding to examine specific contrasts: D1 compared Component racialism to Racialism (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0), and D2 compared Racialism to Biological Essentialism (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1).

Illness/disease difference. *Physical Illness*. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of physical illness, *index* = -.04, *SE* = .04 95%CI [-.131, -.009]. Specifically, as expected, compared

to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in physical illness rates are due to natural (less environmental) causes b = .03, SE = .03, 95%CI [.008, .563] (see Figure 30 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in physical illness rates are due to natural causes, b = .05, SE = .05, 95%CI [.103, .177] (see Figure 30 for path estimates).

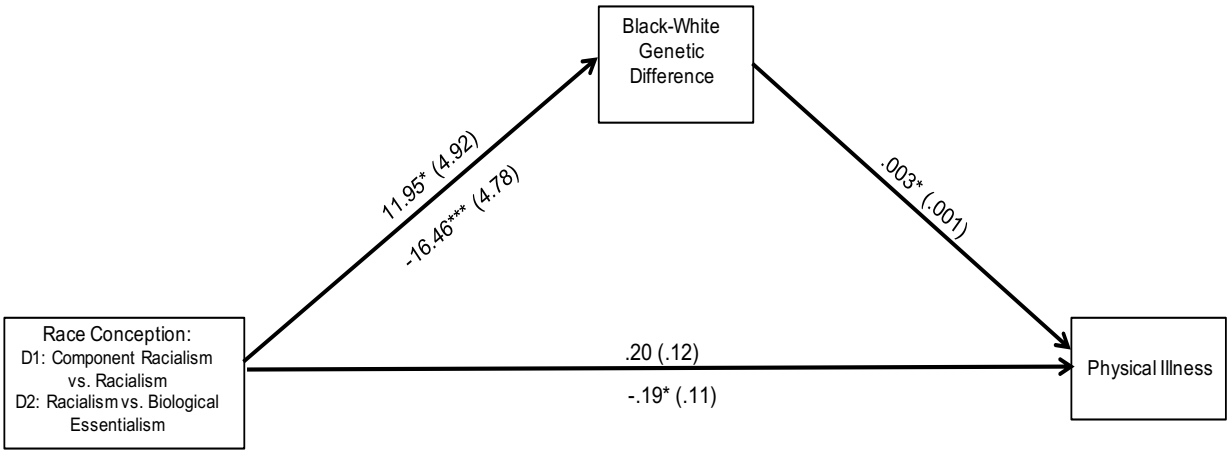


Figure 30. Statistical model depicting the indirect effect of race conception on physical illness through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and physical illness. All coefficients are unstandardized. Standard errors in parentheses.
*p < .05, ***p < .001

Mental Illness. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of mental illness, *index* = -.06, SE = .03 95%CI [-.134, -.027]. Specifically, as expected, compared to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater

cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in mental illness rates are due to natural causes, *b* = .05, *SE* = .02, 95%CI [.005, .099] (see Figure 31 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in mental illness are due to natural causes, *b* = .06, *SE* = .05, 95%CI [.031, .184] (see Figure 31 for path estimates).

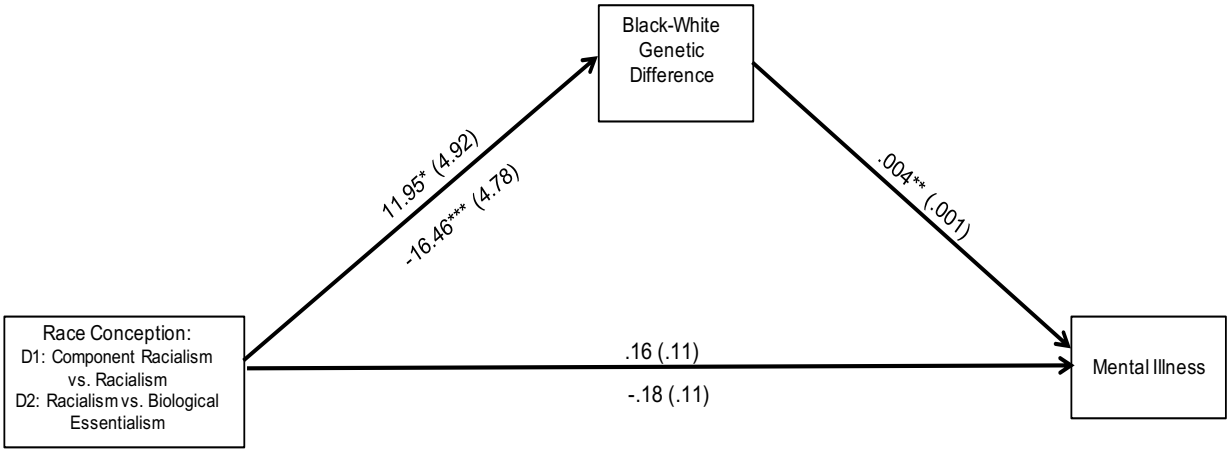


Figure 31. Statistical model depicting the indirect effect of race conception on mental illness through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and mental illness. All coefficients are unstandardized. Standard errors in parentheses.
*$p < .05$, ** $p < .01$, ***$p < .001$

STD/I. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of STD/I, *index* = -.07, *SE* = .03 95%CI [-.120, -.029]. Specifically, as expected, compared to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in STD/I rates are due to natural causes, *b* = .06, *SE* = .03, 95%CI [.002, .101] (see

Figure 32 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White differences in STD/I rates are due to natural causes, *b* = .08, *SE* = .04, 95%CI [.042, .179] (see Figure 32 for path estimates).

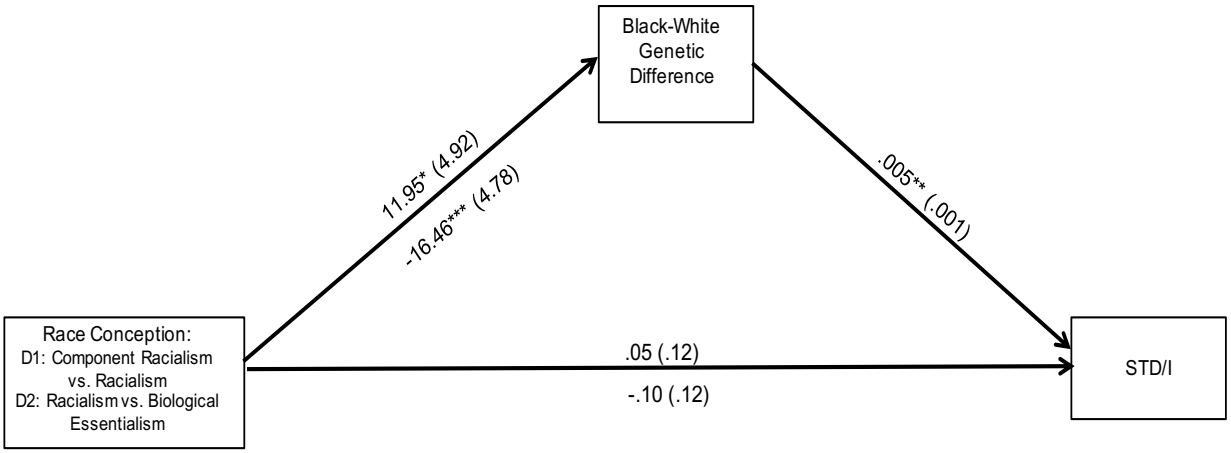


Figure 32. Statistical model depicting the indirect effect of race conception on STD/I through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and STD/I. All coefficients are unstandardized. Standard errors in parentheses.
*$p < .05$, ** $p < .01$, ***$p < .001$

Socio-behavioral difference. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of socio-behavioral differences, *index* = -.07, *SE* = .04 95%CI [-.157, -.035]. Specifically, as expected, compared to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White socio-behavioral differences are due to natural causes, *b* = .05, *SE* = .03, 95%CI [.007, .116] (see Figure 33 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in

the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White socio-behavioral differences are due to natural causes, b = .07, SE = .06, 95%CI [.038, .216] (see Figure 33 for path estimates).

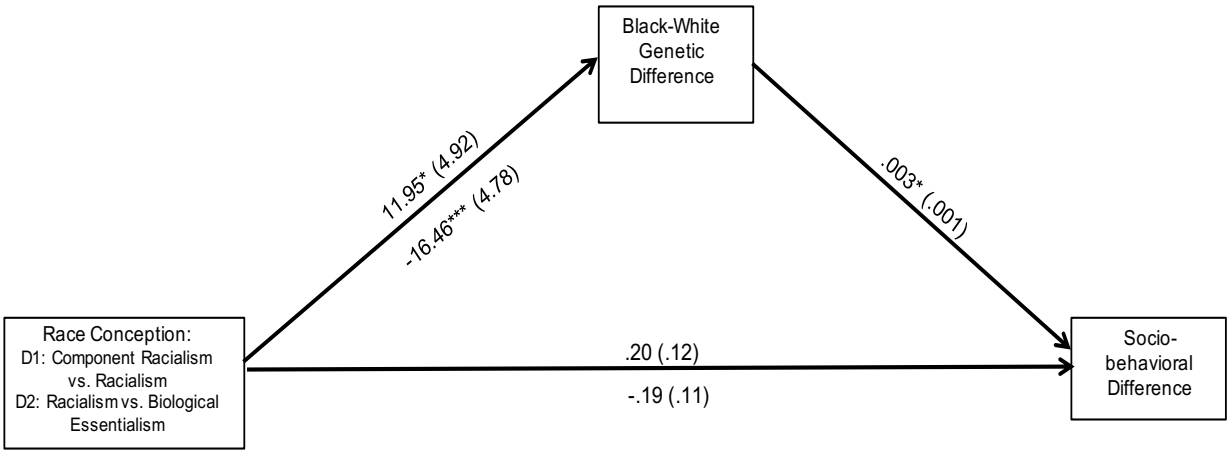


Figure 33. Statistical model depicting the indirect effect of race conception on socio-behavioral difference through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and socio-behavioral difference. All coefficients are unstandardized. Standard errors in parentheses.
*p < .05, ***p < .001

Biological difference. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of biological differences, *index* = -.08, SE = .04 95%CI [-.189, -.045]. Specifically, as expected, compared to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black-White biological differences are due to natural causes, b = .06, SE = .03, 95%CI [.010, .140] (see Figure 34 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was

then associated with an increased belief that Black-White biological differences are due to natural causes, $b = .005$, $SE = .001$, $p < .001$, 95%CI [.003, .008] (see Figure 34 for path estimates).

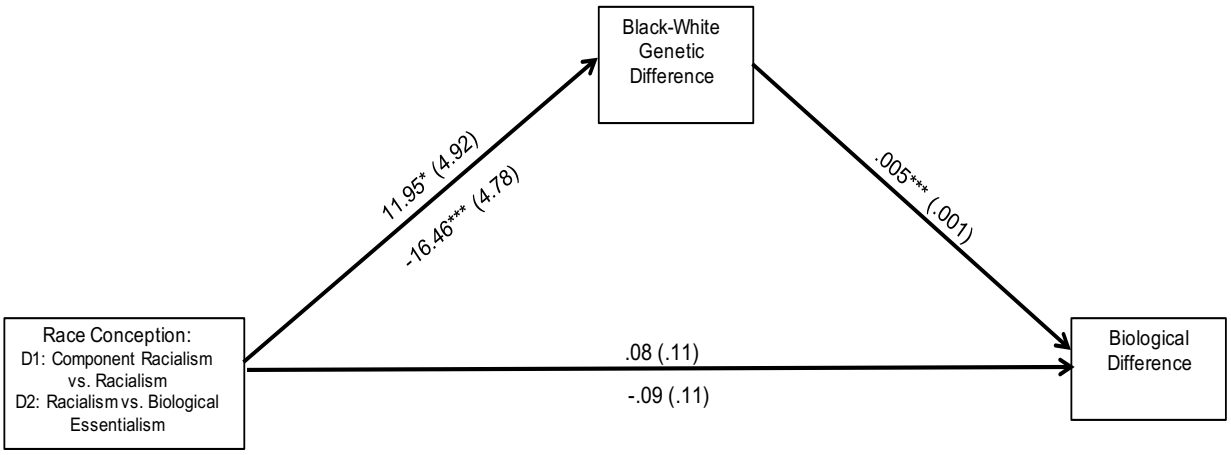


Figure 34. Statistical model depicting the indirect effect of race conception on biological difference through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and biological difference. All coefficients are unstandardized. Standard errors in parentheses.
*$p < .05$, ***$p < .001$

Superhumanization. *Physicality*. Confirming hypotheses, Black-White genetic difference mediated the relationship between biological race conception and perceptions of physicality, *index* = -.07, *SE* = .07 95%CI [-.117, -.028]). Specifically, as expected, compared to the Racialism condition, participants in the Component Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was then associated with an increased belief that Black people possess superhuman physical abilities, $b = .06$, $SE = .02$, 95%CI [.005, .101] (see Figure 35 for path estimates). Also consistent with hypotheses, compared to the Biological Essentialism condition, participants in the Racialism condition believed that genetic differences are a greater cause of Black-White racial differences, which was

then associated with an increased belief that Black people possess superhuman physical abilities, *b* = .08, *SE* = .04, 95%CI [.038, .169] (see Figure 35 for path estimates).

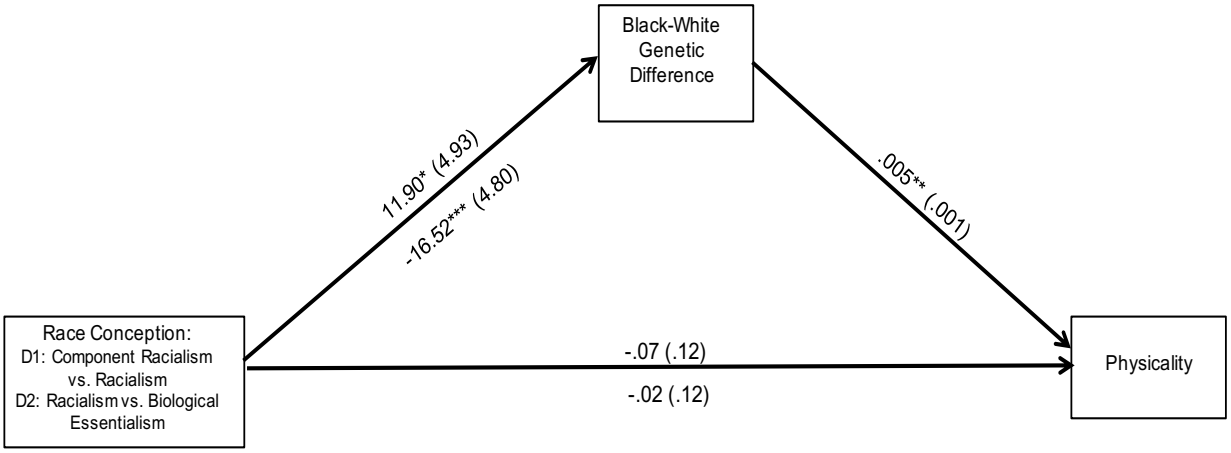


Figure 35. Statistical model depicting the indirect effect of race conception on physicality through Black-White genetic difference. Statistics for D1 (Component Racialism = 1, Racialism = -1, Biological Essentialism = 0) are presented above the line. Statistics for D2 (Component Racialism = 0, Racialism = -1, Biological Essentialism = -1) are presented below the line. Reports direct effect between race conception and physicality. All coefficients are unstandardized. Standard errors in parentheses.
*$p < .05$, ** $p < .01$, ***$p < .001$

Pain tolerance. Inconsistent with predictions, Black-White genetic difference did not mediate the relationship between biological race conception and pain tolerance, *b* = .00, *SE* = .01, 95%CI [-.031, .031].

Cultural practice. Inconsistent with predictions, Black-White genetic difference did not mediate the relationship between biological race conception and cultural practice, *b* = -.02, *SE* = .03, 95%CI [-.100, .014].

Racial stereotyping. Inconsistent with predictions, Black-White genetic difference did not mediate the relationship between biological race conception and racial stereotyping, *b* = -.01, *SE* = .01, 95%CI [-.041, .018].

2.4.3 Study 3 Discussion

Collectively, results show no effect of biological race conception on perceptions of Black-White racial differences or group-level perceptions of Black people. However, supporting hypotheses, results show that beliefs about Black-White genetic differences mediated the relationship between race conceptions and these outcomes. Specifically, results show that, as the concreteness of biological race conception increased (see Figure 1), participants increasingly believed that Black and White people are genetically different, which in turn lead them to perceive Black-White differences in illness/disease rates, and Black-White biological, genetic, and socio-behavioral differences as increasingly due to "natural" causes, as well as increased beliefs that Black people possess superhuman physical abilities.

Interestingly, the mediations highlight one significant effect of component racialist ideologies: its influence on perceptions of racial group differences. Specifically, beliefs about Black-White differences mediated all relationships between race conceptions and Black-White racial difference outcomes, yet only mediated one outcome specific to Black people (i.e., physicality). This suggests that people believe that genetic variation between racial groups (or the unique genetic composition of racial groups, i.e., component racialism) explain (perceived) racial group differences. In addition, consistent with results of the Study 1 and Study 2, these results demonstrate, conceivably, that increasingly endorsing component racialist ideologies can have negative consequences for people's perception of racial disparities (they see these disparities as "natural"), which might further decrease efforts to correct racial disparities, broadly defined. It also shows, though to a lesser extent, that beliefs about racial genetic differences can influence perceptions of racial groups, which can also reinforce rigid ideas about race. Altogether, these results suggest that the concreteness of biological race conception can influence perceptions of racial group disparities through beliefs about racial genetic difference.

However, notwithstanding the strengths of this study, there is a key limitation: despite previous attempts to pilot the effectiveness of the manipulations (see Appendix A), the Component Racialism manipulation failed to shift participants' endorsement of component racialist ideologies. On the one hand, it is likely that the "ineffectiveness" of this manipulation accounted for the null effect of race conception on all main dependent outcomes, in addition to the non-significant differences between the racialism and component racialism conditions. However, if this were the only reason, results would show differences between the racialism and biological essentialism conditions (given that the manipulations shifted people on the Racialism measure). On the other hand, results showing beliefs about Black-White genetic differences as a mediator of these relationships shows, conceivably, that in addition to the belief that racial groups possess unique genetic compositions, beliefs about racial group genetic differences (and the extent of these differences) are one underlying mechanism. Thus, together these results suggest that beliefs about "degree of racial genetic difference" (i.e., the magnitude of essentialism bias) and beliefs about racial group genetic differences are factors involved in component racialist ideologies. Nevertheless, future research should incorporate more explicit measures to capture the extent to which participants perceived the manipulations along the abstract-concrete continuum.

3. GENERAL DISCUSSION

This research investigates a novel concept, *component racialism*: the belief that racial groups possess unique genetic configurations that determine within-racial group similarities and between-racial group differences in behavioral, biological, and physical characteristics and outcomes. In particular, this research uses biogeographical ancestry, through its misrepresentation as "race genes", to examine how component racialist ideologies exacerbate biased perceptions of racial groups and targets. In particular, I explored the following research questions: (1) How does biogeographical ancestry information influence racial categorization, racial stereotyping, and perceived racial differences in illness/disease susceptibility? (2) To what extent do people misrepresent biogeographical ancestry as "race genes", and how does this misrepresentation explain perceptions of racial boundaries, racial differences, and illness/disease susceptibility? (3) To what extent is component racialism different from (or similar to) other forms of biological racial essentialism?

Results from the Pilot Study show that people use a target's racial ancestry information to make judgments about their biogeographical ancestry, such that more Black (less White) racial ancestry was associated with more sub-Saharan biogeographical ancestry. Moreover, biogeographical ancestry mediated the relationship between racial ancestry and racial categorization, such that increasing Black racial ancestry was related to increased beliefs that a target possessed sub-Saharan biogeographical ancestry, which in turn led to increasing Black racial categorization. This study provided initial evidence to suggest that people use biogeographical ancestry to make race-based target judgments. It also suggested that, perhaps, this process is fueled by people's misrepresentations of biogeographical ancestry as race genes,

given the almost proportional relationship between the proportion of Black racial ancestry and sub-Saharan biogeographical ancestry.

Next, building on these findings, in Study 1 I manipulated a target's biogeographical ancestry to investigate how the proportion of sub-Saharan ancestry influences racial categorization and other-raced based target judgments (e.g., racial stereotyping, Cultural practices). Results showed that increasing sub-Saharan Black biogeographical ancestry was related to increasing Black (decreasing White) racial categorization, increased beliefs that the target engaged in Black-associated cultural activities, increased beliefs that the target possesses Black-associated stereotypical traits (e.g., decreased academic orientation), and increased beliefs that the target was biologically different from White people. Further, results showed that racial categorization mediated the relationship between biogeographical ancestry and some raced-based outcomes. Specifically, as sub-Saharan biogeographical ancestry increased, participants increasingly categorized the target as Black, which in turn was associated with increased beliefs that the target engaged in Black-associated cultural activities, and increased beliefs that the target was athletic. Thus, conceptually replicating previous work, this study shows that people use both cultural and biological essentialist beliefs concepts to make sense of race-based outcomes, although not all outcomes.

Next, in Study 2, I further investigated component racialism by manipulating a target's biogeographical ancestry and their racial ancestry. I also more explicitly (less ambiguously) measured the extent to which people misrepresent biogeographical ancestry as "race genes". In particular, in this study I examined the extent to which people misrepresent sub-Saharan biogeographical ancestry as "Black genes", and how this misrepresentation influences race-based

judgments. Further, for the first time, this study examined how biogeographical ancestry influences race-based judgments when a target's racial ancestry is unspecified.

Providing more support for component racialism, results show that people misrepresent sub-Saharan biogeographical ancestry as "Black genes". Specifically, across all racial ancestry conditions (but to varying degrees), increasing sub-Saharan Black racial ancestry was associated with increased beliefs that the target shared genetic material with Black people. This misrepresentation of sub-Saharan biogeographical ancestry as Black genes then influenced raced-based outcomes. For example, among targets with more Black than White ancestry and targets with unspecified racial ancestry, increasing sub-Saharan biogeographical ancestry was associated with increased beliefs that the target shared genes with Black people, which in turn was associated with increased beliefs that the target (1) had a darker skin tone, (2) engaged in Black associated cultural practices, (3) was susceptible to physical illnesses, and (4) was susceptible to mental illnesses.

Together, the Pilot study, Study 1, and Study 2 also presented more evidence of component racialism. In particular, in all three studies, controlling for biological racial essentialism (a more abstract/less concrete form of biological racial conception) did not change any results patterns; thus providing evidence that component racialist ideologist influence race-based judgments above and beyond other, more abstract/less concrete forms of biological race conceptions. In addition, in Study 2, controlling for racialism (a more concrete form of biological racial conception than biological essentialism, but a less concrete form than component racialism) did not change any result patterns. However, although these studies provide strong support for component racialism, they are limited as the less concrete forms of biological racial conceptions were statistically controlled for, rather than manipulated. Another limitation of these

studies is that they examined how component racialist ideologies influence race-based judgments at the target (i.e., individual) level.

Thus, to address these limitations and provide even stronger support for component racialism, in Study 3 I manipulated the abstractness/concreteness of racial conceptualization to more directly test how it influences racialized perceptions and judgments at the group level (i.e., perceptions of entire racial groups). Specifically, I examined how the abstractness/concreteness of biological race conceptions influenced perceptions of between-racial group differences (Black-White racial group differences) and within-racial group similarities (among Black people).

Study 3 results showed no effect of race conception on perceptions of Black-White racial differences or group-level perceptions of Black people. However, providing even greater support for component racialism, results showed that, as the concreteness of race conception increased, participants increasingly believed that Black and White people are genetically different, which in turn lead them to increasingly believe that Black-White differences in illness/disease rates, and Black-White biological, genetic, and socio-behavioral differences are due to "natural" causes, as well as increasingly believe that Black people possess superhuman physical abilities. That is, component racialist beliefs (relative to biological essentialism and racialism) produced a greater magnitude of essentialist bias in race-based judgments. Accordingly, supporting ideas of component racialism, this study demonstrated that beliefs that racial groups possess unique genetic configurations explain perceptions of within-group racial similarities and between-group racial differences more than other forms of biological racial conceptions.

Altogether, these studies provide strong experimental support for component racialism. Particularly, they show that people believe in complex genetic distinctions between racial

groups; a distinction they believe is the root cause of between-group racial differences and within-group racial similarities. Critically, they also show that shifting people to a component racialist view may further concretize mental representations of racial genetic essences, in a way that more strongly predicts beliefs about both racial differences (Heine et al., 2017; Simons & Keil, 1995). Providing empirical support to past theorizing (e.g., Feldman, 2010; Koenig, 2010), this research shows that people often misinterpret the science of biogeographical ancestry as evidence that genes determine race and racial differences, even though only the former is empirically supported (Bamshad, et al., 2004; Feldman, 2010; Hadler et al., 2008; Koenig, 2010; Risch et al., 2002; Rosenberg et al., 2002; Tishkoff & Kidd, 2004). Indeed, they also show that people further misrepresent biogeographical ancestry as "race genes" (Bolnick et al., 2007; Duster, 2006).

This research also provides experimental support to claims that biogeographical ancestry represents a contemporary form of racial formation and racialization (Murji & Solomos, 2005; Omi & Winant, 1994). Further, consistent with Duster (2006), they show that biogeographical ancestry—via genetic tests—can reinforce genetic determinist beliefs about race and racial differences (Bolnick et al., 2007). They also highlight how genetic ancestry tests might provide a mechanism for racism and racialization by reviving ideas of racial categories as proxies for biological differences (Hirschman & Panther-Yates, 2008; Scodari, 2017). Certainly, from these studies one can conclude that espousing component racialist beliefs can reinforce already exaggerated beliefs about innate differences between racial groups and the idea that racial groups each possess unique genetic compositions (see also Koenig, 2010).

For example, replicating past work (e.g., Lawton & Foeman, 2017; Morning, 2017; Nelson, 2008) these studies show that the misrepresentation of biogeographical ancestry as race

genes has negative consequences for how people perceive racial groups, such as in-group preference and out-group stereotyping (Keller, 2005; Schmalor, Cheung, & Heine, *unpublished*; as cited in Heine et al., 2017). These current studies also add to this growing literature in novel ways, by showing that, in addition to racial stereotyping, the misrepresentation of biogeographical ancestry as race genes also affects perceptions of illness/disease susceptibility, biological differences, behaviors (e.g., cultural practices), super-humanization, and racial categorization.

Collectively, these results have broad social implications for understand the perpetuation of racial disparities in health outcomes, healthcare quality, academic outcomes, and treatment within the criminal justice system. These results also have implications for racial ancestry testing, and for the study of biological racial essentialism.

3.1 Implications

Previous work shows that in addition to having a generally limited understanding of genetics—specially as it relates to understanding race and racial differences (Bolnick et al., 2007; Dar-Nimrod & Heine, 2011; Dubriwny et al., 2004)—people are very receptive to genetic ancestry tests (Hochschild & Sen, 2011), even though these tests do not adhere to scientific evidence on, and standards related to, genetic testing (Bolnick et al., 2007; Fullwiley, 2014). Previous work also shows that people use biogeographical information to inform ideas about their own racial identities and group affiliations (Lawton & Foeman, 2017; Morning, 2017; Nelson, 2008; see also Nelson, 2016). This evidence suggest that people also use biogeographical ancestry information to make judgments about others, often in racialized ways. What then are the implications of biogeographical ancestry information, and tests that purport to show one's biogeographical ancestry?

Recently, the U.S. Food and Drug Administration (FDA) granted 23&Me, a direct-to-consumer testing company, authorization to provide information on an individual's genetic predisposition to ten illnesses (e.g., Celiac Disease, Parkinson's Disease; FDA, 2017), and breast cancer (FDA, 2018). This authorization is potentially problematic, given the results of the present work. First, it is conceivable that people might conflate their reported predisposition to illnesses (based on results from ancestry tests) with their biogeographical ancestry information. In doing so, people might erroneously believe that their risk of developing an illness/disease (e.g., breast cancer) is a result of their biogeographical ancestry. Coupled with the misrepresentation of biogeographical ancestry as "race genes", this conflation (of biogeographical ancestry with illness susceptibility) may have implications for how people understand their own illness/disease risk. Potentially more troubling, these erroneous assumptions may be heightened when people take biogeographical ancestry tests that provide no illness/disease risk information. In these cases, people might use their reported biogeographical ancestry information to make judgments about their own susceptibility to illness/disease, especially those that are racialized (e.g., HIV/AIDS: perceived as a "Black disease"; Bredström, 2006). For example, an individual with 12% sub-Saharan biogeographical ancestry might perceive their susceptibility to HIV/AIDS as lower than someone with 70% sub-Saharan biogeographical ancestry. Further work examining how people might use biogeographical ancestry information to form opinions of their own risk to illness/disease is important, given that these perceptions are related to health promoting behaviors (Brewer, 2007).

In the same way, as demonstrated in the present study, people can use biogeographical ancestry information to make judgments about another individual's susceptibility to illness/disease. For example, people in the present studies tended to believe that sub-Saharan

ancestry increases one's "Black genes", which can increase one's susceptibility to mental and physical illness. This false perception of biogeographical ancestry as "race genes" may further perpetuate racial disparities in health outcomes. For example, Hill, Rosentel, Bak, Hebert, and Bouris (2017) showed that young men who have sex with men rated a White target who engaged in insertive and receptive condomless anal sex with a Black partner as being at higher risk for HIV compared to Black/Black and White/White character dyads engaged in the same behaviors. In this example, it is likely (because the characters all engaged in the same behavior) that participants made judgments about the targets risk to HIV based on their implicit ideas about Black-White genetic/biological differences and racialized beliefs about HIV.

In addition, the misrepresentation of biogeographical ancestry as "race genes", along with component racialist ideologies, can further perpetuate racial disparities in healthcare quality, and treatment. For example, among medical and health professionals, the debate around the use of biogeographical ancestry to understand disease risk, progression, treatment, and susceptibility, especially within and between racial groups, is ongoing (Epstein, 200; Fujimura & Rajagopalan, 2010; Jorde & Wodding, 2004; Rotimi, 2004; Tishkoff & Kidd, 2004). This potential misrepresentation of biogeographical ancestry as "race genes" can be problematic given that medical health professionals tend to believe that biological differences explain racial differences in susceptibility to and manifestations of illness and disease (Institute of Medicine, 2003; Shields et al., 2005). Although research has not yet examined the extent to which biogeographical ancestry influences the treatment of patients, or the quality of care they receive, other work has shown that beliefs about Black-White biological differences influences the extent to which medical professionals assess the pain of Black (vs. White) people, and treatment recommendations as a result of this assessment (Hoffman et al., 2016). As both lay people and

medical professional express similar racial biases (Hoffman et al., 2016), it is conceivable that the use of biogeographical information by medical professionals will further exacerbate already existing racial disparities in patient care and treatment, and health care quality (Betancourt, Green, Carrillo, & Ananeh-Firempong II, 2003; Dotson, Bonam, & Jagers, 2017; Fiscella, Franks, Gold, & Clancy, 2000; Nelson, 2002; Saha, Arbelaez, & Cooper, 2003). Future work should, however, directly explore these predictions about component racialism among medical professionals.

Altogether, this study has important implications for ancestry tests (that purport to measure biogeographical ancestry) and representations of biogeographical ancestry in the public and private domains (e.g., mass media, medical and health fields). Duster (2006) argues that the rise of genetic testing, and its favorability among the general population, reinforces genetic determinist beliefs about race and racial differences (Bolnick et al., 2007). Similarly, scholars have argued that genetic ancestry tests provide a mechanism for racism and racialization by reviving ideas of racial categories as proxies for biological differences (Hirschman & Panther-Yates, 2008; Scodari, 2017). Following Lee et al's., (2008) and my own recommendations, ancestry testing companies and other entities that utilize biogeographical ancestry information should always emphasize that (1) there is no scientific basis for any claim that biogeographical ancestry explains racial and ethnic categories and/or their (perceived) similarities/differences (e.g., intelligence, athleticism) and (2) biogeographical ancestry is not a proxy of racial biological similarity/difference (e.g., illness and disease rates). In doing so, these entities can help reduce misinformation about biogeographical ancestry and misrepresentations of biogeographical ancestry as "race genes", thereby reducing the extent to which people use biogeographical ancestry information to form—often misleading and biased—impressions about

themselves and others. In addition, these entities should present the science of biogeographical ancestry and ancestry testing in ways that can be easily understood, while also actively educating consumers (and users) of biogeographical information), especially as it relates to the possible conclusions that can (and cannot) be drawn from this information.

In addition to health-related consequences, the misrepresentation of biogeographical ancestry as "race genes" can have other socially-relevant consequences (e.g., the extent to which people racially categorize and stereotype a target [e.g., academic orientation], as well as ideas about how prototypic members of racial groups should look [e.g., darker skin tone for Black people] and act [e.g., watching TV shows with Black actors]). Consistent with past scholarship, endorsing component racialist ideologies can further perpetuate and endorse racial stereotypes (e.g., Black people as unintelligent; Jayaratne et al., 2006), increasingly homogenize racial groups (e.g., Bastian & Haslam, 2006; 2007; Haslam, Rothschild, & Ernst, 2000), dehumanize members for racial out-groups (e.g., Leyens et al., 2001), and heighten negative attitudes and bias toward racial out-group members (e.g., Haslam, Rothschild, & Ernst, 2002). Such beliefs can also perpetuate ongoing racial disparities by further reducing people's unwillingness to tackle disparities due to heightened perceptions of "natural racial differences". For example, Byrd & Ray (2015) found that White people, who were more accepting of genetic explanations for Blacks' traits and behaviors, were also increasingly likely to oppose racial policies that might benefit Black people (e.g., being against preferential hiring and promotion of blacks). Similarly, Williams and Eberhardt (2008) found that a biological essentialist view of race is associated with less concern with racial inequality. Thus, these exaggerated beliefs of racial genetic differences can maintain, legitimize, and reinforce the racial hierarchy.

Last, these studies have implications for the study of psychological essentialism, broadly, and biological racial essentialism, specifically. In particular, results from this work suggest that, in addition to examining how the different forms of psychological essentialism influence perceptions of social groups, research should also address how the abstract-concreteness of these forms of psychological essentialism influence perception. For example, Dar-Nimrod & Heine (2011) introduced the *Genetic Essentialism Framework* (GEF), which broadly suggests that a genetic essentialist understanding of social groups is related to stereotyping, out-group derogation, and prejudice. Following the new ideas of component racialism presented in this research, the GEF can be reconfigured to include different types of genetic essentialism (e.g., racialism, component racialism) to discuss inferences about group behavior and characteristics that account for the abstractness-concreteness of each form.

3.2 Limitations and Future Work

Despite its strengths, this research has limitations that should be addressed in future work. First, in the Pilot Study and Studies 1 and 2, participants made judgments about Black/White multi-racial targets. As a result, how, and the extent, to which biogeographical ancestry and component racialist ideologies affect perceptions of mono-racial Black and White targets are unknown. However, it is probable that the multi-racial targets in these studies were categorized as either Black or White based on their (perceived) dominant racial ancestry (e.g., Franco & Holmes, 2017; Good et al., 2013; Peery, & Bodenhausen, 2008), thus providing some insight into how these processes might function among mono-racial Black and White targets. Nevertheless, future work needs to investigate these relationships among Black and White mono-racial targets given that their stereotypes differ from their Black/White multiracial counterparts (e.g., Shih, Bonam, Sanchez, & Peck, 2007).

In addition, future work should investigate these relationships outside the Black-White racial dichotomy, to examine the generalizability of these study's findings to other racial groups (e.g., Asian, Native American). In the same way, future work should also investigate how other types of biogeographical ancestries (e.g., East Asian, Native American) influence racialized target and group perceptions in similar (or different) ways. Another likely fruitful avenue for future research, then, is to investigate how seemingly mismatched biogeographical and racial ancestries influence target evaluations. Across these present studies, the target always possessed corresponding biogeographical (i.e., sub-Saharan African, European and racial [Black, White]) ancestries. As a result, the extent to which perceptions shift when the target's biogeographical and racial ancestries do not correspond at all are still unknown (e.g., a target with Native American and East Asian biogeographical ancestries but Black/White racial ancestry).

Third, only White American participants were recruited across all studies. Hence, it is conceivable that the effects of biogeographical ancestry and component racialism are unique to that demographic. However, other work shows that African Americans use this information to form impressions of their own racial identities and group affiliations (e.g., Nelson, 2008), and that non-Americans (i.e., Europeans) also use biogeographical ancestry to make judgments of out-group members (Keller, 2005). So, although previous research has not shown that non-White Americans (e.g., African Americans, Hispanic/Latinx Americans) use biogeographical ancestry information to make out-group racial judgments, previous work suggests it possible. Additionally, other work shows that non-White Americans are also influenced by shifts in racialism essentialism (e.g., Asian Americans; Williams & Eberhardt, 2008). However, the questions of how and the extent to which non-White Americans use biogeographical

information, as well as the extent to which component racialism influences their perceptions, are empirical questions.

Equally, subsequent work should investigate how component racialist ideologies influence perceptions, judgments, and evaluations of one's racial in-group (e.g., how racialist ideologies influence how a White person perceives White people and "White genes"). In doing so, this work will provide insight into the extent to which component racialism operates as an in-group/out-group racial process (see the GEF; Dar-Nimrod & Heine, 2011). Research should also examine how racialist ideologies influence perceptions of the self, as a racial group member. Work on internalized racism (i.e., the endorsement of negative in-group racial stereotypes; Williams, D., & Williams-Morris, 2000) shows that endorsing negative racial stereotypes can have negative implications for racial minorities (e.g., lower ethnic identity and self-esteem; James, 2017). Accordingly, it is possible that component racialist ideologies might exacerbate the negative effects of internalized racism via heighted beliefs that racial differences are "natural". Together, more work is needed to understand how component racialist ideologies influence perceptions of racial in-groups and self-perceptions.

In addition, it is likely that participants' knowledge of genetics broadly, and biogeographical ancestry, specifically, influenced how component racialist views influenced their judgments. However, past work suggests that these biased perceptions of racialized genetic differences might be fueled by racial biases, rather than lacking knowledge about genetics and ancestry. For example, Hoffman and colleagues (2016) found medical experts endorsed false beliefs of Black-White biological differences, and that endorsement of these false beliefs was related to racially biased outcomes. Thus, is probable that the racially biased consequences of biogeographical ancestry and component racialism will still emerge among people who

understand genetics and ancestry very well. Nonetheless, this question is an empirical one that needs to be investigated.

Another limitation of this research is the number of analyses and comparisons made—especially in study 2. With the large number of analysis, the probability of incorrectly rejecting the null hypothesis increased (Moran, 2003). To address this limitation, Bonferroni corrections should be conducted to correct (or compensate) for this increase testing (Armstrong, 2014; Moran, 2003). Future work should also replicate this research to provide more, and stronger, claims of these effects. In addition, to further investigate how, and when, people use racial ancestry and biogeographical ancestry information to make target judgments, future research should attempt to incorporate eye tracking, reaction time data, or hot spots measures. These types of data will allow more definitive inferences about the extent to which people use biogeographical ancestry information and racial ancestry information independently or simultaneously to make judgments.

Altogether, future work should continue to investigate component racialism. In addition to investigating how it influences perceptions of racial out-group members and targets, work should also examine how component racialism influences perceptions of racial in-group members and targets, as well as perceptions of the self. Moreover, subsequent work should examine how component racialist ideologies exacerbate current racial disparities, by investigating its association to racial prejudice and negative racial attitudes and bias. Further, additional work is needed to understand how component racialism functions as a novel and unique form of biological racial essentialism, and how it relates (similarly or differently) to other forms and types of psychological and biological essentialism, respectively.

4. CONCLUSION

This research investigated, and found empirical support, for the the novel concept of component racialism. In particular, this research found that component racialist (vs. racialist and biological essentialist) ideologies further exacerbate biased perceptions of racial groups and targets. In addition, results from this research show that people use biogeographical information to make race-based judgments about targets and that they misrepresent biogeographical information as "genes genes". Altogether, this research has broad social implications for understanding the perpetuation of racial disparities in health outcomes, healthcare quality, academic outcomes, and treatment within the criminal justice system, in addition to implications for racial ancestry testing, and for the study of biological racial essentialism.

REFERENCES

Angermeyer M, & Matschinger H. (2004). The stereotype of schizophrenia and its impact on discrimination against people with schizophrenia: Results from a representative survey in Germany. *Schizophrenia Bulletin*, 30, 1049–1061.

Armstrong, R. A. (2014). When to use the Bonferroni correction. *Ophthalmic and Physiological Optics*, *34*(5), 502-508.

Asnaani, A., Richey, J. A., Dimaite, R., Hinton, D. E., & Hofmann, S. G. (2010). A cross-ethnic comparison of lifetime prevalence rates of anxiety disorders. *The Journal of nervous and mental disease*, *198*(8), 551-555.

Bamshad, M., Wooding, S., Salisbury, B. A., & Stephens, J. C. (2004). Deconstructing the relationship between genetics and race. *Nature reviews genetics*, *5*(8), 598-609.

Bamshad, M. J., Wooding, S., Watkins, W. S., Ostler, C. T., Batzer, M. A., & Jorde, L. B. (2003). Human population genetic structure and inference of group membership. *The American Journal of Human Genetics*, *72*(3), 578-589.

Bar-Anan, Y., & Nosek, B. A. (2014). A comparative investigation of seven indirect attitude measures. *Behavior research methods*, *46*(3), 668-688.

Bastian, B., & Haslam, N. (2006). Psychological essentialism and stereotype endorsement. *Journal of Experimental Social Psychology*, *42*(2), 228-235.

Bastian, B., & Haslam, N. (2007). Psychological essentialism and attention allocation: Preferences for stereotype-consistent versus stereotype-inconsistent information. *The Journal of social psychology*, *147*(5), 531-541.

Betancourt, J. R., Green, A. R., Carrillo, J. E., & Ananeh-Firempong II, O. (2003). Defining Cultural Competence: A Practical Framework for Addressing Racial/Ethnic Disparities in Health and Health Care. *Public Health Reports*, *118*, 293-302.

Bolnick, D. A., Fullwiley, D., Duster, T., Cooper, R. S., Fujimura, J. H., Kahn, J., ... & Ossorio, P. (2007). Genetics. The science and business of genetic ancestry testing. *Science*, *318*(5849), 399-400.

Bonham, V. L., Warshauer-Baker, E., & Collins, F. S. (2005). Race and ethnicity in the genome era: the complexity of the constructs. *American Psychologist*, *60*(1), 9-15.

Bonam, C., Yantis, C., & Taylor, V.J. (2017). Invisible middle class Black space: Asymmetrical person and space stereotyping at the race-class nexus. Manuscript under revision.

Bowcock, A. M., Kidd, J. R., Mountain, J. L., Hebert, J. M., Carotenuto, L., Kidd, K. K., & Cavalli-Sforza, L. L. (1991). Drift, admixture, and selection in human evolution: a study with DNA polymorphisms. *Proceedings of the National Academy of Sciences*, *88*(3), 839-843.

Bredström, A. (2006). Intersectionality: A challenge for feminist HIV/AIDS research?. *European Journal of Women's Studies*, *13*(3), 229-243.

Brescoll, V., & LaFrance, M. (2004). The correlates and consequences of newspaper reports of research on sex differences. *Psychological Science*, *15*(8), 515-520.

Brewer, N. T., Chapman, G. B., Gibbons, F. X., Gerrard, M., McCaul, K. D., & Weinstein, N. D. (2007). Meta-analysis of the relationship between risk perception and health behavior: the example of vaccination. *Health Psychology*, *26*(2), 136-145.

Brug, J., Aro, A. R., Oenema, A., de Zwart, O., Richardus, J. H., & Bishop, G. D. (2004). SARS risk perception, knowledge, precautions, and information sources, the Netherlands. *Emerging Infectious Diseases*, *10*(8), 1486-1489.

Buhagiar, L. J., Sammut, G., Rochira, A., & Salvatore, S. (2018). There's no such thing as a good Arab: Cultural essentialism and its functions concerning the integration of Arabs in Europe. *Culture & Psychology*, doi: 10.1177/1354067X18763795

Buhrmester, M., Kwang, T., & Gosling, S. D. (2011). Amazon's Mechanical Turk: A new source of inexpensive, yet high-quality, data?. *Perspectives on psychological science*, *6*(1), 3-5.

Byrd, W. C., & Ray, V. E. (2015). Ultimate attribution in the genetic era: White support for genetic explanations of racial difference and policies. *The ANNALS of the American Academy of Political and Social Science*, *661*(1), 212-235.

Chandler, M. J., & Proulx, T. (2008). Personal persistence and persistent peoples: Continu- ities in the lives of individual and whole cultural communities. In F. Sani (Ed.), Self-continuity: Individual and collective perspectives (pp. 213–226). New York, NY: Psychology Press.

Ciaccia, C. (Febuary, 2018). "New species of shark discovered has ancestors older than dinosaurs" Retrieved, March 27th, 2018, from, http://www.foxnews.com/science/2018/02/26/new-species-shark-discovered-has-ancestors-older-than-dinosaurs.html

Centers for Disease Control and Prevention. (2014). Depression in the U.S. household population, 2009-2012. *Hyattsville, MD: National Center for Health Statistics*. Retrieved on December 21st, 2017 from: https://www.cdc.gov/nchs/data/databriefs/db172.pdf

Centers for Disease Control and Prevention. (2015). HIV Surveillance Report, 2013. *Atlanta: U.S. Department of Health and Human Services*. Retrieved on December 21st, 2017 from: https://www.cdc.gov/hiv/pdf/library/reports/surveillance/cdc-hiv-surveillance-report-2015-vol-27.pdf

Centers for Disease Control and Prevention. (2016). Sexually Transmitted Disease Surveillance. *Atlanta: U.S. Department of Health and Human Services*. Retrieved on December 21st, 2017 from: https://www.cdc.gov/std/stats16/CDC_2016_STDS_Report-for508WebSep21_2017_1644.pdf.

Condit, C. M., Parrott, R., & Harris, T. M. (2002). Lay understandings of the relationship between race and genetics: development of a collectivized knowledge through shared discourse. *Public Understanding of Science*, *11*(4), 373-387.

Condit, C. M., Parrott, R. L., Bates, B. R., Bevan, J., & Achter, P. J. (2004a). Exploration of the impact of messages about genes and race on lay attitudes. *Clinical genetics*, *66*(5), 402-408.

Condit, C. M., Parrott, R. L., Harris, T. M., Lynch, J., & Dubriwny, T. (2004b). The role of "genetics" in popular understandings of race in the United States. *Public Understanding of Science*, *13*(3), 249-272.

Cooper, R. S., Kaufman, J. S., & Ward, R. (2003). Race and genomics. *The New England Journal of Medicine*, *348*(12), 1166-1170.

Croft, W., & Cruse, D. A. (2004). *Cognitive linguistics*. New York, NY: Cambridge University Press.

Crump, M. J., McDonnell, J. V., & Gureckis, T. M. (2013). Evaluating Amazon's Mechanical Turk as a tool for experimental behavioral research. *PloS one*, *8*(3), e57410.

Dar-Nimrod, I., & Heine, S. J. (2011a). Genetic essentialism: on the deceptive determinism of DNA. *Psychological bulletin*, *137*(5), 800-818.

Dar-Nimrod, I., & Heine, S. J. (2011b). Some thoughts on essence placeholders, interactionism, and heritability: reply to Haslam (2011) and Turkheimer (2011). *Psychological Bulletin*, *137*(5), 829-833.

Donovan, J., Pasquetto, I., & Pierre, J. (2018, January). Cracking Open the Black Box of Genetic Ancestry Testing. In *Proceedings of the 51st Hawaii International Conference on System Sciences*.

Dotson, E., Bonam, C., & Jagers, J. (2017). Redefining race as a process: Implications for healthcare leadership. *Journal of Health Administration Education*, *34*(2), 295-318.

Dubriwny, T. N., Bates, B. R., & Bevan, J. L. (2004). Lay understandings of race: Cultural and genetic definitions. *Public Health Genomics*, *7*(4), 185-195.

Duster, T. (2006). The molecular reinscription of race: unanticipated issues in biotechnology and forensic science. *Patterns of Prejudice*, *40*(4-5), 427-441.

Edwards, H. (1972). The myth of the racially superior athlete. *Intellectual Digest*, 44, 32-38

Eligon, J. (2017, May). "Sergeant Says He Faced Taunts at Work After Learning He's Part Black." *The New York Times*. Retrieved, March 19th, 2018, from, https://www.nytimes.com/2017/05/12/us/cleon-brown-black-lawsuit.html

Entine, J. (2008). *Taboo: Why black athletes dominate sports and why we're afraid to talk about it*. Public Affairs.

Epstein, C. J. (2006). Medical genetics in the genomic medicine of the 21st century. *The American Journal of Human Genetics*, *79*(3), 434-438.

Excoffier, L. G. L., Laval, G., & Schneider, S. (2005). Arlequin (version 3.0): An integrated software package for population genetics data analysis. *Evolutionary Bioinformatics Online*, *1*, 47-50.

Feldman, M. W. (2010). The biology of ancestry: DNA, genomic variation, and race. *Doing race: 21 essays for the 21st century*, 136-59.

Feldman, M. W., Lewontin, R. C., & King, M. C. (2003). Race: a genetic melting pot. *Nature*, 424(6947), 374-374.

Fiscella, K., Franks, P., Gold, M. R., & Clancy, C. M. (2000). Inequality in quality: addressing socioeconomic, racial, and ethnic disparities in health care. *Journal of the American Medical Association*, *283*(19), 2579-2584.

Franco, M. G., & Holmes, O. L. (2017). Biracial Group Membership Scale. *Journal of Black Psychology*, *43*(5), 435-450.

Fujimura, J. H., & Rajagopalan, R. (2011). Different differences: The use of 'genetic ancestry' versus race in biomedical human genetic research. *Social Studies of Science*, *41*(1), 5-30.

Fullwiley, D. (2014). The "contemporary synthesis": When politically inclusive genomic science relies on biological notions of race. *Isis*, *105*(4), 803-814.

Gannett, L. (2014). Biogeographical ancestry and race. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, *47*, 173-184.

Genetics Working Group. (2005). The use of racial, ethnic, and ancestral categories in human genetics research. *The American Journal of Human Genetics*, *77*(4), 519-532.

Gerstein, M. B., Bruce, C., Rozowsky, J. S., Zheng, D., Du, J., Korbel, J. O., ... & Snyder, M. (2007). What is a gene, post-ENCODE? History and updated definition. *Genome research*, *17*(6), 669-681.

Gil-White, F. J. (2001). Are ethnic groups biological "species" to the human brain? Essentialism in our cognition of some social categories. *Current Anthropology*, 42, 515–554.

Gelman, S. A. (2003). *The essential child: Origins of essentialism in everyday thought*. Oxford Series in Cognitive Development.

Gelman, S. A. (2004). Psychological essentialism in children. *Trends in cognitive sciences*, *8*(9), 404-409.

Gelman, S. A., Frazier, B. N., Noles, N. S., Manczak, E. M., & Stilwell, S. M. (2015). How much are Harry Potter's glasses worth? Children's monetary evaluation of authentic objects. *Journal of Cognition and Development*, *16*(1), 97-117.

Gelman, S. A., & Wellman, H. M. (1991). Insides and essences: Early understandings of the non-obvious. *Cognition*, *38*(3), 213-244.

Good, J. J., Sanchez, D. T., & Chavez, G. F. (2013). White ancestry in perceptions of Black/White biracial individuals: Implications for affirmative-action contexts. *Journal of Applied Social Psychology*, *43*(S2), E276-E286.

Gould, S. J. (1996). *The mismeasure of man*. New York, NY: WW Norton & Company.

Graves Jr, J. L. (2015). Why the nonexistence of biological races does not mean the nonexistence of racism. *American Behavioral Scientist*, *59*(11), 1474-1495.

Greely, H. T. (2008). Genetic genealogy: Genetics meets the marketplace. In *Revisiting Race in a Genomic Age*. Edited by Barbara A. Koenig, Sandra Soo-Jin Lee and Sarah S. Richardson. New Brunswick: Rutgers University Press, pp. 215–34.

Guo, G., Fu, Y., Lee, H., Cai, T., Harris, K. M., & Li, Y. (2014). Genetic bio-ancestry and social construction of racial classification in social surveys in the contemporary United States. *Demography*, *51*(1), 141-172.

Haga, S. B., & Venter, J. C. (2003). FDA races in wrong direction. *Science, 301,* 466.

Halder, I., Shriver, M., Thomas, M., Fernandez, J. R., & Frudakis, T. (2008). A panel of ancestry informative markers for estimating individual biogeographical ancestry and admixture from four continents: utility and applications. *Human mutation*, *29*(5), 648-658.

Hamilton, M., & Rajaram, S. (2001). The concreteness effect in implicit and explicit memory tests. *Journal of Memory and Language*, *44*(1), 96-117.

Haslam, N. (2011). Genetic essentialism, neuroessentialism, and stigma: commentary on Dar-Nimrod and Heine (2011). *Psychological Bulletin*, *137*(5), 819-824.

Haslam, N., Rothschild, L., & Ernst, D. (2000). Essentialist beliefs about social categories. *British Journal of Social Psychology*, *39*(1), 113-127.

Haslam, N., Rothschild, L., & Ernst, D. (2002). Are essentialist beliefs associated with prejudice?. *British Journal of Social Psychology*, *41*(1), 87-100.

Haslam, N., & Whelan, J. (2008). Human natures: Psychological essentialism in thinking about differences between people. *Social and Personality Psychology Compass*, *2*(3), 1297-1312.

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation, moderation, and conditional process modeling.

Hayes, A. F. (2016). PROCESS: a versatile computational tool for observed variable mediation, moderation, and conditional process modeling. 2012. *Acesso em*, *2*.

Hayes, A. F. (2018). *Introduction to mediation, moderation, and conditional process analysis*. (2nd Ed.). New York, N.Y: The Guilford Press.

Heine, S. J. (2017). DNA is not destiny: The remarkable, completely misunderstood relationship between you and your genes. New York, NY: Norton.

Heine, S. J., Dar-Nimrod, I., Cheung, B. Y., & Proulx, T. (2017). Chapter Three-Essentially Biased: Why People Are Fatalistic About Genes. *Advances in Experimental Social Psychology*, *55*, 137-192.

Hendrick, H. W. (1979). Differences in group problem-solving behavior and effectiveness as a function of abstractness. *Journal of Applied Psychology*, *64*(5), 518-525.

Heyman, G. D., & Gelman, S. A. (2000). Beliefs about the origins of human psychological traits. Developmental Psychology, 36, 663–678.

Hill, B. J., Rosentel, K., Bak, T., Hebert, L. E., & Bouris, A. (2017). Race and HIV Risk: Exploring Race and HIV Risk Perceptions Among Young Men Who Have Sex With Men. *Journal of Adolescent Health*, *60*(2), S9-S10.

Hirschman, C., Alba, R., & Farley, R. (2000). The meaning and measurement of race in the US census: Glimpses into the future. *Demography*, *37*(3), 381-393.

Hirschman, E. C., & Panther-Yates, D. (2008). Peering inward for ethnic identity: Consumer interpretation of DNA test results. *Identity: An International Journal of Theory and Research*, *8*(1), 47-66.

Hoberman, J. M. (1997). *Darwin's athletes: How sport has damaged Black America and preserved the myth of race*. Houghton Mifflin Harcourt.

Hochschild, J. L., & Sen, M. (2011). Reification or Blurring: The Impact of Genomic Ancestry Testing on Americans' Racial Identity. *Unpublished paper, Harvard University, department of Government*.

Hochschild, J. L., Weaver, V. M., & Burch, T. R. (2012). *Creating a new racial order: How immigration, multiracialism, genomics, and the young can remake race in America*. Princeton, NJ: Princeton University Press.

Hoffman, K. M., Trawalter, S., Axt, J. R., & Oliver, M. N. (2016). Racial bias in pain assessment and treatment recommendations, and false beliefs about biological differences between blacks and whites. *Proceedings of the National Academy of Sciences*, *113*(16), 4296-4301.

Ho, A. K., Roberts, S. O., & Gelman, S. A. (2015). Essentialism and racial bias jointly contribute to the categorization of multiracial individuals. *Psychological science*, *26*(10), 1639-1645.

Horvath, M., & Ryan, A. M. (2003). Antecedents and potential moderators of the relationship between attitudes and hiring discrimination on the basis of sexual orientation. *Sex Roles*, *48*(3-4), 115-130.

Institute of Medicine. (2003). *Unequal treatment: Confronting racial and ethnic disparities in health care*. Washington, DC: National Academies Press.

James, D. (2017). Internalized racism and past-year major depressive disorder among African-Americans: The role of ethnic identity and self-esteem. *Journal of racial and ethnic health disparities*, *4*(4), 659-670.

Jayaratne, T. E., Ybarra, O., Sheldon, J. P., Brown, T. N., Feldbaum, M., Pfeffer, C. A., & Petty, E. M. (2006). White Americans' genetic lay theories of race differences and sexual

orientation: Their relationship with prejudice toward Blacks, and gay men and lesbians. *Group Processes & Intergroup Relations*, *9*(1), 77-94.

Jayaratne, T. E., Gelman, S. A., Feldbaum, M., Sheldon, J. P., Petty, E. M., & Kardia, S. L. (2009). The perennial debate: Nature, nurture, or choice? Black and White Americans' explanations for individual differences. *Review of General Psychology*, *13*(1), 24-33.

Johnson, M. D., & Kisielius, J. (1985). Concreteness-Abstract-Ness and the Feature-Dimension Distinction. In *NA - Advances in Consumer Research Volume 12, eds. Elizabeth C. Hirschman and Moris B. Holbrook, Provo, UT: Association for Consumer Research,* P. 325-*328.*

Kang, S. K., Plaks, J. E., & Remedios, J. D. (2015). Folk beliefs about genetic variation predict avoidance of biracial individuals. *Frontiers in psychology*, *6:357.*

Keil, F. C. (1989). Concepts, kinds, and cognitive development. Cambridge, MA: MIT Press.

Keller, J. (2005). In genes we trust: the biological component of psychological essentialism and its relationship to mechanisms of motivated social cognition. *Journal of personality and social psychology*, *88*(4), 686-702.

Kim, S. E., Pérez-Stable, E. J., Wong, S., Gregorich, S., Sawaya, G. F., Walsh, J. M., & Kaplan, C. P. (2008). Association between cancer risk perception and screening behavior among diverse women. *Archives of Internal Medicine*, *168*(7), 728-734.

Kimel, S. Y., Huesmann, R., Kunst, J. R., & Halperin, E. (2016). Living in a genetic world: How learning about interethnic genetic similarities and differences affects peace and conflict. *Personality and Social Psychology Bulletin*, *42*(5), 688-700.

Knofczynski, G. T., & Mundfrom, D. (2008). Sample sizes when using multiple linear regression for prediction. *Educational and Psychological Measurement*, *68*(3), 431-442.

Koenig, B. (2010). Which Differences Make a Difference? Race, DNA and Health. *Doing Race: 21 Essays for the 21st Century*, 160-84.

Kosoy, R., Nassir, R., Tian, C., White, P. A., Butler, L. M., Silva, G., ... & De La Vega, F. M. (2009). Ancestry informative marker sets for determining continental origin and admixture proportions in common populations in America. *Human mutation*, *30*(1), 69-78.

Kteily, N., Bruneau, E., Waytz, A., & Cotterill, S. (2015). The ascent of man: Theoretical and empirical evidence for blatant dehumanization. *Journal of personality and social psychology*, *109*(5), 901-931.

Jorde, L. B., & Wooding, S. P. (2004). Genetic variation, classification and'race'. *Nature genetics*, *36*, S28-S33.

Lawton, B., & Foeman, A. (2017). Shifting Winds: Using Ancestry DNA to Explore Multiracial Individuals' Patterns of Articulating Racial Identity. *Identity*, *17*(2), 69-83.

Lee, S. S. J., Mountain, J., Koenig, B., Altman, R., Brown, M., Camarillo, A., ... & Ford, R. (2008). The ethics of characterizing difference: guiding principles on using racial categories in human genetics. *Genome Biology*, *9*(7), 404.1-404.4.

Leyens, J., Rodriguez-Perez, A., Rodriguez-Torres, R., Gaunt, R., Pal- adino, M., Vaes, J., & Demoulin, S. (2001). Psychological essentialism and the differential attribution of uniquely human emotions to ingroups and outgroups. *European Journal of Social Psychology, 31,* 395–411.

Lewis, A. E. (2003). Everyday race-making: Navigating racial boundaries in schools. *American Behavioral Scientist*, *47*(3), 283-305.

Linnaeus, C. V. (1758). Systema naturae, 10th edn, vol. 1. *Stockholm: L. Salvii*.

Machunsky, M., & Meiser, T. (2009). Ingroup projection as a means to define the superordinate category efficiently: Response time evidence. *Social Cognition*, *27*(1), 57-75.

Mason, W., & Suri, S. (2012). Conducting behavioral research on Amazon's Mechanical Turk. *Behavior research methods*, *44*(1), 1-23.

Massey, Douglas S., and Jennifer A. Martin. 2003. The NIS Skin Color Scale.

Medin, D. L., & Ortony, A. (1989). Psychological essentialism. *Similarity and analogical reasoning*, *179*-195.

Mersha, T. B., & Abebe, T. (2015). Self-reported race/ethnicity in the age of genomic research: its potential impact on understanding health disparities. *Human Genomics*, *9*(1), 1-15.

Moran, M. D. (2003). Arguments for rejecting the sequential Bonferroni in ecological studies. *Oikos*, *100*(2), 403-405.

Morning, A. (2009). Toward a sociology of racial conceptualization for the 21st century. *Social Forces*, *87*(3), 1167-1192.

Morning, A. (2011). *The nature of race: How scientists think and teach about human difference*. University of California Press.

Morning, A. (2017). Kaleidoscope: contested identities and new forms of race membership. *Ethnic and Racial Studies*, 1-19.

Murji, K., & John, S. (2005). Introduction: Racialization in theory and practice. In *Racialization: Studies in Theory and Practice*. Edited by Karim Murji and John Solomos. New York, NY: Oxford University Press, pp. 1–27.

Murray, C., & Herrnstein, R. (1994). The bell curve. *Intelligence and Class Structure in American Life, New York*.

National Center for Health Statistics. (2016). Health, United States, 2015: With Special Feature on Racial and Ethnic Health Disparities. *Hyattsville, MD*. Retrieved on December 21st, 2017 from: https://www.cdc.gov/nchs/data/hus/hus15.pdf

National Institutes of Health. (2017). *Help Me Understand Genetics Genetic Testing*. Retrieved on December 7th, 2017 from: https://ghr.nlm.nih.gov/primer/testing/genetictesting

National Institutes of Health. (2018). *What is a gene?* Retrieved on April 13th, 2018 from: https://ghr.nlm.nih.gov/primer/basics/gene

Nelson, A. (2002). Unequal treatment: confronting racial and ethnic disparities in health care. *Journal of the National Medical Association*, *94*(8), 666-668.

Nelson, A. (2008). Bio science: Genetic genealogy testing and the pursuit of African ancestry. *Social Studies of Science*, *38*(5), 759-783.

Nelson, A. (2016). *The social life of DNA: Race, reparations, and reconciliation after the genome*. Boston, MA: Beacon Press.

Norenzayan, A., & Heine, S. J. (2005). Psychological universals: What are they and how can we know?. *Psychological bulletin*, *131*(5), 763-784.

Nugent, H. (2007). Race raw Nobel scientist James Watson scraps tour after being suspended. Retrieved on December 6th , 2007 from: http://archive.li/2vw82Omi, M., & Howard, W. (1994). *Racial Formation in the United States: From the 1960s to the 1990s*, 2nd ed. New York, NY: Routledge.

Ossorio, P., & Duster, T. (2005). Race and genetics: controversies in biomedical, behavioral, and forensic sciences. *American Psychologist*, *60*(1), 115-128.

Paivio, A. (1969). Mental imagery in associative learning and memory. *Psychological Review*, *76*(3), 241-263.

Panofsky, A., & Donovan, J. (2017). When genetics challenges a racist's identity: Genetic ancestry testing among white nationalists.

Pasta, D. J. (2009). Learning When to Be Discrete: Continuous vs. Categorical Predictors. SAS Global Forum 2009. ICON Clinical Research, San Francisco, CA.

Pauker, K., & Ambady, N. (2009). Multiracial faces: How categorization affects memory at the boundaries of race. *Journal of Social Issues*, *65*(1), 69-86.

Peery, D., & Bodenhausen, G. V. (2008). Black+ White= Black: Hypodescent in reflexive categorization of racially ambiguous faces. *Psychological Science*, *19*(10), 973-977.

Peralta, C. A., Ziv, E., Katz, R., Reiner, A., Burchard, E. G., Fried, L., ... & Shlipak, M. (2006). African ancestry, socioeconomic status, and kidney function in elderly African Americans: a genetic admixture analysis. *Journal of the American Society of Nephrology*, *17*(12), 3491-3496.

Phelan, J. C. (2005). Geneticization of deviant behavior and consequences for stigma: The case of mental illness. *Journal of Health and Social Behavior*, 46, 307–322.

Phelan, J. C., Cruz-Rojas, R., & Reiff, M. (2002). Genes and stigma: The connection between perceived genetic etiology and attitudes and beliefs about mental illness. *Psychiatric Rehabilitation Skills*, *6*(2), 159-185.

Phelan, J. C., Link, B. G., Zelner, S., & Yang, L. H. (2014). Direct-to-consumer racial admixture tests and beliefs about essential racial differences. *Social psychology quarterly*, *77*(3), 296-318.

Plaks, J. E., Malahy, L. W., Sedlins, M., & Shoda, Y. (2012). Folk beliefs about human genetic variation predict discrete versus continuous racial categorization and evaluative bias. Social Psychological and Personality Science, 3(1), 31-39.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2008). Behavioral Genetics (5th edition). New York: Worth Publishers.

Reiner, A. P., Carlson, C. S., Ziv, E., Iribarren, C., Jaquish, C. E., & Nickerson, D. A. (2007). Genetic ancestry, population sub-structure, and cardiovascular disease-related traits among African-American participants in the CARDIA Study. *Human Genetics*, *121*(5), 565-575.

Rhemtulla, M., Brosseau-Liard, P. É., & Savalei, V. (2012). When can categorical variables be treated as continuous? A comparison of robust continuous and categorical SEM estimation methods under suboptimal conditions. *Psychological methods*, *17*(3), 354-373.

Richeson, J. A., & Sommers, S. R. (2016). Toward a social psychology of race and race relations for the twenty-first century. *Annual review of psychology*, *67*, 439-463.

Risch, N., Burchard, E., Ziv, E., & Tang, H. (2002). Categorization of humans in biomedical research: genes, race and disease. *Genome biology*, *3*(7), comment2007-1.

Rips, L. I. (1989). Similarity, typicality, and categorization. In S. Vosniadou & A. Ortony (Eds.), Similarity and analogical reasoning (pp. 21–59). New York: Cambridge University Press.

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

Rosenberg, N. A., Pritchard, J. K., Weber, J. L., Cann, H. M., Kidd, K. K., Zhivotovsky, L. A., & Feldman, M. W. (2002). Genetic structure of human populations. *science*, *298*(5602), 2381-2385.

Roth, W. D., & Lyon, K. (2018). Genetic ancestry tests and race: Who takes them, why, and how

do they affect racial identities. In K. Suzuki & D. von Vacano (Eds.), *Reconsidering race: Cross-disciplinary and interdisciplinary approaches*. New York: Oxford University Press. *Forthcoming*

Rothbart, M., & Taylor, M. (1992). Category labels and social reality: Do we view social categories as natural kinds? In G. R. Semin & K. Fiedler (Eds.), *Language, interaction and social cognition* (pp. 11-36). Thousand Oaks, CA, US: Sage Publications, Inc.

Rotimi, C. N. (2004). Are medical and nonmedical uses of large-scale genomic markers conflating genetics and'race'?. *Nature genetics*, *36*, S43-S47.

Rowe, D. C., & Rodgers, J. E. (2005). Under the skin: On the impartial treatment of genetic and environmental hypotheses of racial differences. *American Psychologist*, *60*(1), 60-70.

Royal, C. D., Novembre, J., Fullerton, S. M., Goldstein, D. B., Long, J. C., Bamshad, M. J., & Clark, A. G. (2010). Inferring genetic ancestry: opportunities, challenges, and implications. *The American Journal of Human Genetics*, *86*(5), 661-673.

Rushton, J. P. (1990). Race and Crime: A Reply to Robers and Gabor. *Canadian J. Criminology*, *32*, 315-334.

Rushton, J. P., & Jensen, A. R. (2005). Thirty years of research on race differences in cognitive ability. *Psychology, public policy, and law*, *11*(2), 235-294.

Rushton, J. P., & Jensen, A. R. (2010). Race and IQ: A theory-based review of the research in Richard Nisbett's Intelligence and How to Get It. *The Open Psychology Journal*, *3*(1), 9-35.

Saha, S., Arbelaez, J. J., & Cooper, L. A. (2003). Patient–physician relationships and racial disparities in the quality of health care. *American Journal of Public Health*, *93*(10), 1713-1719.

Sanchez, D. T., & Bonam, C. M. (2009). To disclose or not to disclose biracial identity: The effect of biracial disclosure on perceiver evaluations and target responses. *Journal of Social Issues*, *65*(1), 129-149.

Sanchez, D. T., Good, J. J., & Chavez, G. (2011). Blood quantum and perceptions of Black-White biracial targets: The Black ancestry prototype model of affirmative action. *Personality and Social Psychology Bulletin*, *37*(1), 3-14.

Sankar, P., Cho, M. K., Condit, C. M., Hunt, L. M., Koenig, B., Marshall, P., ... & Spicer, P. (2004). Genetic research and health disparities. *JAMA*, *291*(24), 2985-2989.

Schmalor, A., Cheung, B. Y., & Heine, S. J. (2016). Unpublished data. The University of British Columbia.

Scodari, C. (2017). When Markers Meet Marketing: Ethnicity, Race, Hybridity, and Kinship in Genetic Genealogy Television Advertising. *Genealogy*, *1*(4), 22, 1-14.

Senior, V., Marteau, T. M., & Weinman, J. (2000). Impact of genetic testing on causal models of heart disease and arthritis: an analogue study. *Psychology & Health*, *14*(6), 1077-1088.

Shih, M., Bonam, C., Sanchez, D., & Peck, C. (2007). The social construction of race: Biracial identity and vulnerability to stereotypes. *Cultural Diversity and Ethnic Minority Psychology*, *13*(2), 125-133.

Shiloh, S., Rashuk-Rosenthal, D., & Benyamini, Y. (2002). Illness causal attributions: an exploratory study of their structure and associations with other illness cognitions and perceptions of control. *Journal of Behavioral Medicine*, *25*(4), 373-394.

Shriver, M. D., Parra, E. J., Dios, S., Bonilla, C., Norton, H., Jovel, C., ... & Baron, A. (2003). Skin pigmentation, biogeographical ancestry and admixture mapping. *Human genetics*, *112*(4), 387-399.

Simons, D. J., & Keil, F. C. (1995). An abstract to concrete shift in the development of biological thought: The insides story. *Cognition*, *56*(2), 129-163.

Smedley, A., & Smedley, B. D. (2012). *Race in North America: Origin and evolution of a worldview*. Westview Press.

Smith, C. D. (1981). Recognition memory for sentences as a function of concreteness/abstractness and affirmation/negation. *British Journal of Psychology*, *72*(1), 125-129.

Soylu Yalcinkaya, N., Estrada-Villalta, S., & Adams, G. (2017). The (Biological or Cultural) Essence of Essentialism: Implications for Policy Support among Dominant and Subordinated Groups. *Frontiers in psychology*, *8*, 900. doi: 10.3389/fpsyg.2017.00900

Stephan, W. G. (1977). Stereotyping: The role of ingroup-outgroup differences in causal attribution for behavior. *The Journal of Social Psychology*, *101*(2), 255-266.

Stephens, J. C., Schneider, J. A., Tanguay, D. A., Choi, J., Acharya, T., Stanley, S. E., ... & Duan, J. (2001). Haplotype variation and linkage disequilibrium in 313 human genes. *Science*, *293*(5529), 489-493.

Sternberg, R. J., Grigorenko, E. L., & Kidd, K. K. (2005). Intelligence, race, and genetics. *American Psychologist*, *60*(1), 46-59.

Strevens, M. (2003). Concept Acquisition without a Final Criterion.

Teachman, B. A., Gapinski, K. D., Brownell, K. D., Rawlins, M., & Jeyaram, S. (2003). Demonstrations of implicit anti-fat bias: the impact of providing causal information and evoking empathy. *Health Psychology*, *22*(1), 68-78.

Tishkoff, S. A., & Kidd, K. K. (2004). Implications of biogeography of human populations for'race'and medicine. *Nature genetics*, *36*, S21-S27.

U. S. Food and Drug Administration/Center for Drug Evaluation and Research. (2017, April). Retrieved, March 19th, 2018, from, https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm551185.htm

U. S. Food and Drug Administration/Center for Drug Evaluation and Research. (2018, March). Retrieved, March 19th, 2018, from, https://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm599560.htm

Verkuyten, M. (2003). Discourses about ethnic group (de-) essentialism: Oppressive and progressive aspects. *British Journal of Social Psychology*, *42*(3), 371-391.

Wang, V. O., & Sue, S. (2005). In the eye of the storm: race and genomics in research and practice. *American Psychologist*, *60*(1), 37-45.

Warren, J. W., & Twine, F. W. (1997). White Americans, the new minority?: Non-blacks and the ever-expanding boundaries of whiteness. *Journal of Black Studies*, *28*(2), 200-218.

Waytz, A., Hoffman, K. M., & Trawalter, S. (2015). A superhumanization bias in Whites' perceptions of Blacks. *Social Psychological and Personality Science*, *6*(3), 352-359.

Whitfield, K. E., & McClearn, G. (2005). Genes, environment, and race: Quantitative genetic approaches. *American Psychologist*, *60*(1), 104-114.

Wickens, D. D., & Engle, R. W. (1970). Imagery and abstractness in short-term memory. *Journal of Experimental Psychology*, *84*(2), 268-272.

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

Williams, R. (2006). Generalized ordered logit/partial proportional odds models for ordinal dependent variables. *Stata Journal*, *6*(1), 58-82.

Williams, M. J., & Eberhardt, J. L. (2008). Biological conceptions of race and the motivation to cross racial boundaries. *Journal of personality and social psychology*, *94*(6), 1033-1047.

Witherspoon, D. J., Wooding, S., Rogers, A. R., Marchani, E. E., Watkins, W. S., Batzer, M. A., & Jorde, L. B. (2007). Genetic similarities within and between human populations. *Genetics*, *176*(1), 351-359.

Wolinsky, H. (2006). Genetic genealogy goes global. Although useful in investigating ancestry, the application of genetics to traditional genealogy could be abused. *EMBO Reports*, *7*, 1072-1074.

Young, D. M., Sanchez, D. T., & Wilton, L. S. (2017). Biracial perception in black and white: How Black and White perceivers respond to phenotype and racial identity cues. *Cultural Diversity and Ethnic Minority Psychology*, *23*(1), 154-164.

Yzerbyt, V., Judd, C. M., & Corneille, O. (2004). Subjective Essentialism in Action. In *The psychology of group perception*(pp. 90-107). Psychology Press.

APPENDIX A

RACE CONCEPTION SCALE ITEMS

1. If a black American family traveled around the world, people they met would probably think of them as black, too.
2. The physical features of different racial groups haven't really changed much over the centuries.
3. The same racial categories have pretty much always existed.
4. It's impossible to determine how a person will be racially categorized by examining their DNA. (R)
5. No one can change his or her race - you are who you are.
6. If a white American family traveled around the world, people they met would probably think of them as white, too.
7. It's natural to notice the racial group to which people belong.
8. I believe physical features determine race.
9. Generally speaking, two black people will always look more similar to each other than a black person and a white person ever would.
10. How a person is defined racially depends on the social context. (R)
11. Siblings born to the same parents will always be of the same race as each other.
12. Young children probably learn about which people fall into which racial groups automatically, without much help from adults.
13. A person's race is fixed at birth.
14. The political climate can dictate whether someone is categorized as black or white. (R)
15. In 200 years, society will use basically the same racial categories.
16. There's agreement across cultures about which racial groups people fall into.
17. The average person is highly accurate at identifying people by race.
18. People who are of different races may look quite similar to each other. (R)
19. Racial categories haven't always existed in the world. (R)
20. It's easy to tell what race people are by looking at them.
21. Racial groups are primarily determined by biology.
22. It's possible to be a full member of more than one race. (R)

(R) = Reverse coded

Citation: Williams, M. J., & Eberhardt, J. L. (2008). Biological conceptions of race and the motivation to cross racial boundaries. *Journal of personality and social psychology*, *94*(6), 1033-1047.

APPENDIX B

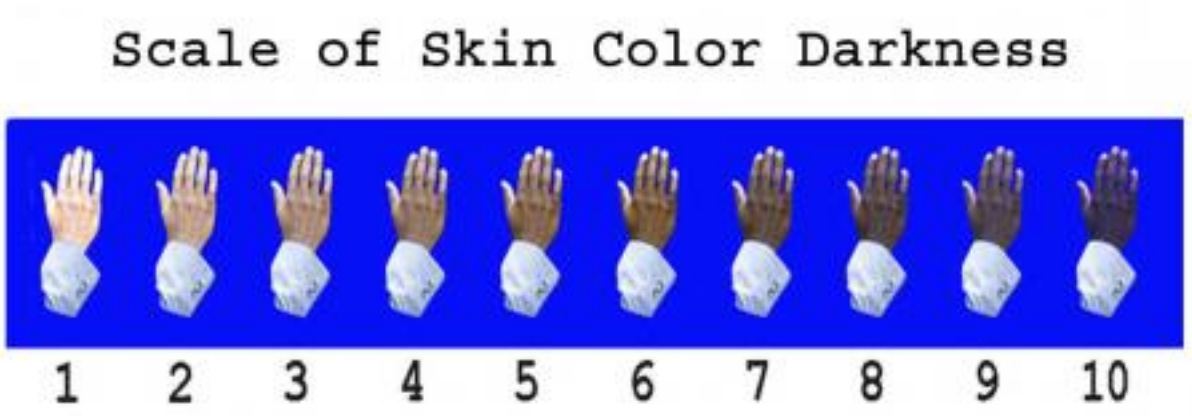
BIOLOGICAL DIFFERENCE (FALSE BELIEFS) SCALE ITEMS

1. This person will age more slowly than the average White person
2. This person's nerve-endings are less sensitive than the average a White person's nerve-endings
3. This person's skin is thicker than the average White's person skin
4. This person has a more sensitive sense of smell than a White person; they can differentiate odors and detect faint smells better than the average a White person
5. This person is significantly more fertile than the average White person
6. This person is better at detecting movement than the average White person
7. This person has a stronger immune system than the average White person and is less likely to contract colds

Items adapted from: Hoffman, K. M., Trawalter, S., Axt, J. R., & Oliver, M. N. (2016). Racial bias in pain assessment and treatment recommendations, and false beliefs about biological differences between blacks and whites. *Proceedings of the National Academy of Sciences*, *113*(16), 4296-4301.

APPENDIX C

MASSeY SKIN TONE SCALE



Citation: Massey, Douglas S., and Jennifer A. Martin. 2003. The NIS Skin Color Scale.

APPENDIX D

RACIAL STEREOTYPING SCALE ITEMS

1. Clean (R)
2. Dirty
3. Looks put-together (R)
4. Criminal
5. Articulate (R)
6. Unsuccessful
7. Wealthy (R)
8. Pleasant (R)
9. Dangerous
10. Poor
11. Good (R)
12. Bad
13. Nice (R)
14. Educated (R)
15. Threatening
16. Intelligent (R)
17. Friendly (R)
18. Uneducated

(R) = Reverse coded

Citation: Bonam, C., Yantis, C., & Taylor, V.J. (2017). Invisible middle class Black space: Asymmetrical person and space stereotyping at the race-class nexus. Manuscript under revision.

APPENDIX E

RACE CONCEPTION MANIPULATION PILOT

This pilot study examines the effectiveness of the race conception manipulations (articles) used in Study 3, along with participants' evaluations of, and reactions to, each manipulation (article). In particular, this study investigates the extent to which each manipulation shifts people into biological, racialist, and component racialist conceptions of race. Following results from Study 1 and 2, I expect that the more concrete the biological race conception, the more extreme participant's biological, racialist, and component racialist conceptions of race (See Figure 1).

Method

Participants

Following participant recruitment methods and sample size-determining procedures detailed in the Pilot Study, I recruited 296 (56.4% female; M_{age}= 39.55 years, SD_{age} = 13.49) White U.S. citizen adults from MTurk.

Materials and Procedure

After providing informed consent for the web-based study, participants were randomly assigned to read one of four articles discussing research on a "recent scientific discovery". Participants were told that the purpose of the study was to develop materials for future research with the goal of understanding how to more effectively present scientific discoveries to the general population. In reality, the four articles were used to manipulate participants' race conceptions (discussed below). After reading the assigned article, participants completed measures relating to the article, measures of race conceptions, along with some basic demographic questions.

Biological race conception. Participants were assigned to read one of four articles. In one article, which served as the *control*, participants read about the discovery of a new species of shark (taken directly from Ciaccia, 2018). This article was used as the control because of its length, and because it discussed a new scientific discovery, which fit the study's cover story. The other three articles discussed scientific discoveries relevant to race. These articles were adaptations of the "Scientists Reveal That Race Has No Genetic Basis, But Does Have a Biological One" article used in Williams & Eberhardt (2008) to shift people into a biological conception of race. However, because of the purpose of this study I adapted the article to fit three different racial conceptualizations. In particular, one article discussed the scientific discovery of a general *biological*, but not specifically genetic, basis of race; this represented the *biological essentialism* condition. Another article discussed the scientific discovery of a *genetic* basis of race; this represented the *racialism* condition. The third article discussed the discovery of racial genetic differences; this represented the *component racialism* condition (see Appendix B for full articles).

Article evaluations. After reading the assigned article, participants rated them along four dimensions on 7-point bipolar Likert scales: (1) Difficulty to read (1 = *extremely easy* to 7 = *extremely difficult*); (2) Difficulty to understand (1 = *extremely easy* to 7 = *extremely difficult*); (3) Engagement (1 = *extremely unengaging* to 7 = *extremely engaging*); (4) Interestingness (1 = *extremely uninteresting* to 7 = *extremely interesting*). Participants then evaluated the information presented in the article along three dimensions on 7-point bipolar Likert scales: (1) Complexity (1 = *extremely simple* to 7 = *extremely complex*); (2) Believability: 1 = *extremely unbelievable* to 7 = *extremely believable*); (3) Clarity (1 = *extremely unclear* to 7 = *extremely clear*).

Reading comprehension/Attention checks. Next, participants responded to six reading

comprehension questions that gauged their level of attention while reading the article. The reading comprehension questions, all multiple choice, included selecting: (1) the name of the interviewed scientist; (2) the interviewed scientist's university; (3) the scientific journal in which the research paper was published; (4) the researchers' other lines of work; (5) the species of shark discovered (asked only when assigned the control condition), or the name of the gene mentioned in the article (asked only for the component racialism, racialism, and biological essentialism conditions.

Conceptual manipulation checks. Last, to assess the extent to which the articles shifted racial conceptualizations, participants complete the Race Conception Scale (Williams & Eberhardt, 2008; $\alpha = .89$), to assess their endorsement of biological views of race; and the Racialism measure (Study 2; $\alpha = .74$), to assess their endorsement of genetic views of race. Participants also completed a newly constructed measure of component racialist ideology, the Component Racialism measure. The Component racialism measure captures beliefs that racial groups possess unique genetic compositions, which produce observed racial differences. For this measure, participants rated the extent to which they agree to two items on a 7-point Likert Scale (1= *strongly disagree* to 7= *strongly agree*): (1) Racial groups each possess a unique set of genes; (2) The chief reason why members of racial groups are so different in behavior and character is because they possess different genes. Both items were averaged with higher scores representing greater endorsement of component racialist ideology ($r = .51$, $p < 001$).

Results

I conducted several one-way ANOVAs to investigate evaluations of each article, and the extent to which they shifted race conceptions. I also conducted ANOVA analysis to examine participants' recollection of each article. Appendix Table 1 presents correlations among main

study variables, along with their means and standard deviations. Appendix Table 2 presents the means and standard deviations of dependent variables as a function of race conception.

Appendix Table 1. Correlations (*r*), Means (*M*), and Standard Deviations (*SD*) for main study variables

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
1. Diff. Reading	-									
2. Diff. Understanding	.76***	-								
3. Engagement	-.28***	-.31***	-							
4. Interestingness	-.28***	-.32***	.74***	-						
5. Complexity	.46***	.47***	-.13*	-.13*	-					
6. Believability	-.23***	-.30**	.35***	.33***	-.07	-				
7. Clarity	-.50***	-.56***	.48***	.44***	-.19**	.44***	-			
8. Bio.Essentialism	-.05	.01	.07	.05	.06	.05	.10	-		
9. Racialism	.08	.13*	-.01	.00	.15*	-.07	-.01	.53***	-	
10. Component Racialism	.08	.15**	.06	.07	.14*	-.07	.01	.55***	.75***	-
M(*SD*)	3.23 (1.60)	3.24 (1.58)	4.75 (4.75)	5.01 (1.47)	4.46 (1.40)	5.29 (1.44)	5.41 (1.32)	4.31 (.88)	3.84 (1.13)	3.98 (1.43)

Note. Diff. = Difficulty; Bio. = Biological;
* $p < .05$, ** $p < .01$, *** $p < .001$

Appendix Table 2 Means (*M*), and Standard Deviations (*SD*) of main depdent variables as a function of experiemental condition.

	Mean (Standard Deviation)			
Condition	**Component Racialism** n= 73	**Racialism** n= 75	**Biological Essentialism** n= 74	**Control** n= 74
Measure: Article				
Difficulty Reading	3.29 (1.65)	3.33 (1.59)	3.30 (1.58)	3.01 (1.61)
Difficulty Understanding	3.47 (1.64)	3.31 (1.50)	3.19 (1.51)	2.99 (1.64)
Engagement	4.64 (1.46)	4.77 (1.46)	4.78 (1.43)	4.78 (1.65)
Interestingness	5.04 (1.42)	4.96 (1.39)	4.99 (1.52)	5.05 (1.56)
Complexity	4.62 (1.43)	4.60 (1.34)	4.34 (1.39)	4.28 (1.44)
Believability	5.36 (1.27)	5.12 (1.46)	5.11 (1.49)	5.56 (1.50)
Clarity	5.34 (1.40)	5.38 (1.17)	5.41 (1.11)	5.50 (1.57)
Conceptual Check				
Biological Racial Essentialism	4.52 (.84)	4.32 (.78)	4.15 (.86)	4.27 (1.00)
Racialism	4.16 (1.08)	4.00 (1.00)	3.51 (1.11)	3.73 (1.23)
Component Racialism	4.52 (1.14)	4.09 (1.38)	3.45 (1.45)	3.86 (1.54)

Article evaluation. *Difficulty reading*. Results revealed no condition effect on the difficulty to read the article, $F(3, 291) = .62$, $p = .604$, $\eta_p^2 = 0.01$.

Difficulty understanding. Results revealed no condition effect on the difficulty to understand the article, $F(3, 291) = 1.21$ $p = .305$, $\eta_p^2 = 0.02$.

Engagement. Results revealed no condition effect on article engagement, $F(3, 291) = .15$, $p = .929$, $\eta_p^2 < 0.01$.

Interestingness. Results revealed no condition effect on article interestingness, $F(3, 291) = .07$, $p = .977$, $\eta_p^2 < 0.01$.

Complexity. Results revealed no condition effect on article complexity, $F(3, 291) = 1.12$ $p = .342$, $\eta_p^2 = 0.01$.

Believability. Results revealed no condition effect on article believability, $F(3, 288) = 1.66$, $p = .176$, $\eta_p^2 = 0.02$.

Clarity. Results revealed no condition effect on article clarity, $F(3, 291) = .19$, $p = .902$, $\eta_p^2 < 0.01$.

Conceptual manipulation checks. *Biological essentialism*. Results revealed no effect of condition on biological racial essentialism, $F(3, 290) = 2.35$, $p = .073$, $\eta_p^2 = 0.02$.

Racialism. Results revealed a condition effect on genetic racial determinism, $F(3, 290) = 5.79$, $p = .003$, $\eta_p^2 = 0.05$. Planned contrast revealed that participants endorsed genetic determinist views of race to a greater extent in the Component Racialism condition relative to the Biological essentialism and Control conditions, $p < .001$ and $p = .020$, respectively. Planned contrast also revealed that participants endorsed genetic determinist views of race to a greater extent in the Racialism condition relative to the Biological essentialism condition, $p = .015$.

Component racialism. Results revealed a condition effect on component racialist beliefs, $F(3, 289) = 7.58$, $p < .001$, $\eta_p^2 = 0.07$. Planned contrast revealed that participants endorsed component racialist ideologies to a greater extent in the Component Racialism condition relative to the Racialism, Biological essentialism, and Control conditions, $p = .065$, $p < .001$, and $p = .004$, respectively. Planned contrast also revealed that participants endorsed component racialist ideologies to a greater extent in the Racialism condition relative to the Biological essentialism condition, $p = .005$.

Reading comprehension/Attention checks. I summed (total = 5) the number of correct responses to the reading comprehension questions and performed a one-way ANOVA to

investigate differences in information recollection. Results revealed no condition effect on the number of correct reading comprehension questions, $F(3, 292) = 1.11$, $p = .347$, $\eta_p^2 = 0.01$, (Component Racialism: $M = 3.93$.; $SD = 1.19$; Racialism: $M = 3.83$; $SD = 1.38$; Biological Essentialism: $M = 4.12$; $SD = .99$; Control: $M = 4.12$; $SD = 1.20$).

Discussion and Conclusion

Against predictions, manipulating the concreteness of racial conceptualization (i.e., component racialism, racialism, biological essentialism, control), did not shift participants' endorsement of biological essentialist views of race. The equal endorsement of biological essentialist views of race across the conditions supports work showing that lay people often think about race in a biological way (Smedley & Smedley, 2005; Sternberg, Grigorenko, & Kidd, 2005; Wang & Sue, 2005). In addition, in hindsight, as a genetic cause is de facto a "biological cause", it is not surprising that shifting people into a racialist or component racialist conceptions of race did not lead them to view race as *more* biologically determined than people in the biological essentialism condition.

Further, confirming predictions, manipulating the concreteness of racial conceptualization shifted participants' endorsement of racialist ideologies. Specifically, participants endorsed genetic determinist views of race to a greater extent in the Component Racialism condition relative to the Biological essentialism and Control conditions, and in the Racialism condition relative to the Biological essentialism condition. Similarly, confirming predictions, manipulating the concreteness of racial conceptualization shifted endorsement of component racialist ideologies. Specifically, participants endorsed component racialist ideologies to a greater extent in the Component Racialism condition relative to the Racialism, Biological essentialism, and Control conditions, and in in the Racialism condition relative to the Biological

essentialism condition.

There were no differences between the (1) Racialism and Control conditions, and the (2) Biological essentialism and Control conditions on the Component racialism and Racialism measures. For these results, the similarities in rating across the two measures may be because even though lay people often think about race in biological ways, this "biological" was of thinking is, more often than not, genetic (Condit et al., 2004). Thus, perhaps when uninhibited (i.e., in the control condition), people think about race in both biological (broadly) and genetic (more specifically) ways. Consequently, when forced to think about race in either a biological way or a genetic way, people's conceptualization of race shifts to fit the position; which might explain the similarities between the control condition and biological essentialism and racialism conditions in this study. In addition, there was no difference in the endorsement of genetic determinist views of race between the Component Racialism and Racialism conditions. This is likely because both positions argue that genes determine race (although for different reasons).

Overall, results revealed no differences in how participants evaluated the articles, and their content, along all dimensions (difficulty to read, difficulty to understand, engagement, complexity, clarity, interestingness, and believability). Results also showed no difference in participants' recollection of information presented in each article; thus suggesting that they paid equal attention (measured by performance on reading comprehension questions) to the content of each article. These results strengthen claims that changes in participants' race conceptualizations that follow reading the article are mostly driven by content of each article, and not by other factors such as level of understanding, clarity, and believability. In addition, this study provides more empirical support component racialism; especially how it differs from other forms of race conceptions. Reported correlations show that the component racialism measure was correlated

moderated with both the measure of biological racial essentialism (r = .55) and the measure of racialism (r = .75). Thus, suggesting that although the component racialism may be related to other race conceptions, it remains a unique conception of race.

APPENDIX F

Component Racialism

"Scientists Pinpoint Genetic Underpinnings of Race: Different Races have Different Sets of Genes"

CHARLOTTESVILLE—Scientists working on mapping the origins of life through the Human Genome Project have uncovered some genetic codes that they believe can be used as indicators of racial background.

"Up till now, [we] weren't able to determine a person's race based just on DNA," said Robert Kaminsky, a University of Virginia scientist and lead author of the study, which was just released in the prestigious journal *Gene*. "But now we're able to use some of the genetic cues to skin color and other physical features to guess at what a person may look like, based on a very small genetic sample."

Dr. Kaminsky and a graduate student, Lisa Faridany, along with colleague Anthony Schmidt of the Georgetown Medical Center, have been working for several years on mapping the genotypic expressions involved in skin color and other phenotypic physical features. Their research is based on the idea that after humans migrated out of Africa, populations developed distinct genetic markers. According to the researchers, these isolated populations represent what we call racial groups.

Results from the study showed that racial groups each possess a unique set of genes. For example, they found that racial groups each have varying types of the melanocortin 1 receptor (MCR1) gene, which is implicated most powerfully in skin color.

The researchers used skin, blood, and other tissue samples from hospital patients whose race was indicated in their charts, but was kept hidden from lab members until the genetic analyses were complete.

"We found that once we had a good idea of where the genetic components to some of these key physical features were located, we were able to correctly guess the patients' racial backgrounds 87% of the time, which is well above chance rate," Dr. Kaminsky said. "And with Black and White patients in particular, our success rates were even higher."

Their results add to the growing body of evidence that so much of who we are as people can be traced to our genetic origins— including race.

"This doesn't mean that there aren't other biological influences on race, just like everything else," Dr. Kaminsky cautioned. "But in the end, we obtain our genetic material from our parents, so we generally inherit their race along with everything else."

The researchers maintain that race provides a "suitable proxy" for genetic differences among human populations and can be used to understand genetic differences that predispose people to illness and disease. Dr. Kaminsky says that "a large fraction of genomic variation is responsible for differences among racial groups."

He pointed to evolutionary theories as to why humans might have evolved to have different physical appearances. For example, the melanin that produces a dark skin color among people of African heritage may have served as a life-saving protection against strong sun exposure, he said. And among people living in what is now Northern Europe, their relatively lesser access to sunlight was aided by fairer skin, which allows for greater absorption of Vitamin D.

Dr. Kaminsky and his colleagues are continuing their contribution to the Human Genome Project with current work on the genetic underpinnings of depression and other mood disorders.

Racialism

"Scientists Pinpoint Genetic Underpinnings of Race"

CHARLOTTESVILLE—Scientists working on mapping the origins of life through the Human Genome Project have uncovered some genetic codes that they believe can be used as indicators of racial background.

"Up till now, [we] weren't able to determine a person's race based just on DNA," said Robert Kaminsky, a University of Virginia scientist and lead author of the study, which was just released in the prestigious journal *Gene*. "But now we're able to use some of the genetic cues to skin color and other physical features to guess at what a person may look like, based on a very small genetic sample."

Dr. Kaminsky and a graduate student, Lisa Faridany, along with colleague Anthony Schmidt of the Georgetown Medical Center, have been working for several years on mapping the genotypic expressions involved in skin color and other phenotypic physical features. They have focused particularly on the melanocortin 1 receptor (MCR1) gene, which is implicated most powerfully in skin color. The present study explores the link between this gene and the phenylalanine hydroxylase protein, which is involved in melanin production, in varying amounts for different racial groups.

The researchers used skin, blood, and other tissue samples from hospital patients whose race was indicated in their charts, but was kept hidden from lab members until the genetic analyses were complete.

"We found that once we had a good idea of where the genetic components to some of these key physical features were located, we were able to correctly guess the patients' racial backgrounds 69% of the time, which is well above chance rate," Dr. Kaminsky said. "And with Black and White patients in particular, our success rates were even higher."

Their results add to the growing body of evidence that so much of who we are as people can be traced to our genetic origins— including race.

"This doesn't mean that there aren't other biological influences on race, just like everything else," Dr. Kaminsky cautioned. "But in the end, we obtain our genetic material from our parents, so we generally inherit their race along with everything else."

He pointed to evolutionary theories as to why humans might have evolved to have different physical appearances. For example, the melanin that produces a dark skin color among people of African heritage may have served as a life-saving protection against strong sun exposure, he said. And among people living in what is now Northern Europe, their relatively lesser access to sunlight was aided by fairer skin, which allows for greater absorption of Vitamin D.

Dr. Kaminsky and his colleagues are continuing their contribution to the Human Genome Project with current work on the genetic underpinnings of depression and other mood disorders.

Biological Essentialism

"Scientists Reveal That Race Has No Genetic Basis, But Does Have a Biological One"

CHARLOTTESVILLE—Scientists working on mapping the origins of life through the Human Genome Project have definitively demonstrated that no genetic codes can be tied to racial background.

"Up till now, there was a big question [in the scientific community] about whether we could determine a person's race based just on DNA," says Robert Kaminsky, a University of Virginia scientist and lead author of the study, which was just released in the prestigious journal *Gene*. "But now we know the answer— there are no genetic markers that indicate what racial group a person belongs to."

Dr. Kaminsky and a graduate student, Lisa Faridany, along with colleague Anthony Schmidt of the Georgetown Medical Center, have been working for several years on mapping the genotypic expressions involved in skin color and other phenotypic physical features. Their study explored the link between various genes (for example the melanocortin 1 receptor (MCR1) gene that determines skin color) and race.

The researchers used skin, blood, and other tissue samples from hospital patients whose race was indicated in their charts, but was kept hidden from lab members until the genetic analyses were complete.

"We found that even when we had a good idea of where the genetic components to some of these key physical features were located, we were able to correctly guess the patients' racial backgrounds only 14% of the time, which is really no better than chance rate," Dr. Kaminsky said. "There's just no one cue or set of cues that indicates, say, whether someone is Black or White."

Their results add to the growing body of evidence that although genes do play an important role in who we are, other biological factors may in many circumstances be even more powerful in determining race.

"This doesn't mean that there aren't other biological components to race," Dr. Kaminsky cautioned. "We do inherit our physical appearance from our parents, so although we did not find evidence that genes determine race, there can still be other biological explanations for race that we have yet to discover."

Dr. Kaminsky and his colleagues are continuing their contribution to the Human Genome Project with current work on the genetic underpinnings of depression and other mood disorders.

Control

"New species of shark discovered has ancestors older than dinosaurs"

Maybe they just needed a bigger boat.
After decades of looking for it, scientists have discovered a new species of shark, one whose ancestors lived 250 million years ago.
Appropriately dubbed Atlantic sixgill shark, the Hexanchus vitulus belongs to the sixgill family of sharks and lives in the Atlantic Ocean. Atlantic sixgill sharks are different from the sixgill sharks that live in the Indian and Pacific Oceans only on a molecular level — it is not something that could be spotted with the naked eye.
"We showed that the sixgills in the Atlantic are actually very different from the ones in the Indian and Pacific Oceans on a molecular level, to the point where it is obvious that they're a different species even though they look very similar to the naked eye," said Florida Tech assistant professor Toby Daly-Engel in a statement. Engel is also the lead researcher of the study.
The sixgill shark is mostly found in "tropical and temperate waters" according to the study's abstract, which cited areas of the Atlantic Ocean as Belize, Gulf of Mexico and Bahamas.
The scientists looked at 1,310 base pairs of two mitochondrial genes and found that there were enough differences to determine that the Atlantic version of the sixgill shark is different enough from those living in the Pacific and Indian oceans to be considered a different species.
The study, which has been published in the journal *Marine Biodiversity*, was a collaboration of work from different institutions. The Florida Institute of Technology, MarAlliance, Florida State University Coastal and Marine Laboratory and the National Marine Fisheries Service, Southeast Fisheries Science Center were all involved.
Sixgill sharks have been on the planet for millions of years, with ancestors having swam in the oceans for 250 million years. By comparison, dinosaurs first appeared approximately 230 million years ago, according to the USGS.
Because sixgill sharks swim thousands of feet below the ocean surface, identifying and studying them has been difficult. Unlike their relatives in the Indian and Pacific Oceans, they do not grow very large, reaching approximately six feet in length by adulthood. They also have saw-like teeth on the lower jaw and six gill slits.
Sixgill sharks in the Indian and Pacific Oceans can reach over 15 feet in length when fully grown.
"Because we now know there are two unique species, we have a sense of the overall variation in populations of sixgills. We understand that if we overfish one of them, they will not replenish from elsewhere in the world," Daly-Engel added in the statement. He also said that the study sheds more light on shark diversity, "particularly diversity in the deep ocean, which we don't know much about."

Citation: Ciaccia, C. (Febuary, 2018). "New species of shark discovered has ancestors older than dinosaurs" Retrieved, March 27th, 2018, from, http://www.foxnews.com/science/2018/02/26/new-species-shark-discovered-has-ancestors-older-than-dinosaurs.html

APPENDIX G

RESULTS WITH CONTROLS

Pilot Study

Preliminary analyses. Controlling for Biological Racial Essentialism

Racial categorization. A one-way analysis of variance (ANOVA) revealed a main effect of racial ancestry on racial categorization, $F(2, 237) = 117.80$, $p < .001$, $\eta_p^2 = 0.50$

Black/sub-Saharan Biogeographical ancestry. A one-way ANOVA revealed a main effect of racial ancestry on biogeographical ancestry, $F(2, 236) = 204.43$, $p < .001$, $\eta_p^2 = 0.63$.

Mediation analyses: Biogeographical ancestry mediates the relationship between racial ancestry and racial categorization. Biogeographical ancestry mediated the relationship between racial ancestry and racial categorization, omnibus: $b = .03$, $SE = .01$, CI [.018, .041]. Participants ascribed more Black/sub-Saharan biogeographical ancestry to the 2B/2W target compared to the 1B/3W target, which in turn led them to categorize the 2B/2W target as more Black, $b = -.86$, $SE = .15$, CI [-1.205, -.600]. Participants ascribed more Black/sub-Saharan biogeographical ancestry to the 3B/1W target compared to the 2B/2W target, which in turn led them to categorize the 3B/1W target as more Black, $b = .86$, $SE = .15$, CI [.594, 1.193].

Study 1

Primary analyses: Biogeographical ancestry effects. Controlling for Biological Racial Essentialism

Complex racial categorization. *Black.* Increasing sub-Saharan biogeographical ancestry lead to greater Black racial categorization, $b = .21$, $SE = .03$, $p < .001$, CI [.158, .262]. *White*. Increasing sub-Saharan biogeographical ancestry lead to decreased White racial categorization, $b = -.20$, $SE = .03$, $p < .001$, CI [-.251, -.151].

Black cultural practices. Increasing sub-Saharan biogeographical ancestry increased beliefs that the target engaged in Black-associated cultural practices, $b = .20$, $SE = .04$, $p < .001$, CI [.122, .273]

Black racial stereotyping. Increasing sub-Saharan biogeographical ancestry was not associated with academic orientation, $b = -.05$, $SE = .04$, $p = .189$, 95%CI [-.131, .026], athletic ability, $b = .02$, $SE = .04$, $p = .645$, CI [-.052, .084], or criminal inclination, $b = .06$, $SE = .04$, $p = .093$, CI [-.011, .138].

Biological difference. Increasing sub-Saharan biogeographical ancestry increased beliefs that the target is biologically different from White people, $b = .11$, $SE = .05$, $p = .033$, CI [.009, .216].

Mediation analyses: Racial categorization as a mediator.

Black racial categorization as a mediator

Black cultural practices. Black racial categorization mediated the relationship between biogeographical ancestry and Black cultural practices, $b = .12$, $SE = .02$, CI [.075, .173].

Black racial stereotyping. Black racial categorization mediated the relationship between biogeographical ancestry and athleticism, $b = .05$, $SE = .02$, CI [.020, .089]. Black racial categorization did not mediate the relationship between biogeographical ancestry and academic orientation, $b = .00$, $SE = .02$, CI [-0.34, .030] or and criminal inclination, $b = -.01$, $SE = .02$, CI [-.045, .024].

Biological difference. Black racial categorization did not mediate the relationship between biogeographical ancestry and biological difference, $b = .04$, $SE = .02$, CI [-.007, .088].

White racial categorization as a mediator

Black cultural practices. Black racial categorization mediated the relationship between biogeographical ancestry and Black cultural practices, b = .05, SE = .02, CI [.007, .095].

Black racial stereotyping. Black racial categorization mediated the relationship between biogeographical ancestry and athleticism, b = -.04, SE = .02, CI [-.070, -.004]. Black racial categorization did not mediate the relationship between biogeographical ancestry and academic orientation, b = -.03, SE = .02, CI [-.057, .003] or and criminal inclination, b = .02, SE = .02, CI [-.011, .059].

Biological difference. Black racial categorization did not mediate the relationship between biogeographical ancestry and biological difference, b = -.04, SE = .03, CI [-.088, .012].

Study 2

Racialism as the control

Primary analyses: Biogeographic and racial ancestry effects

Black genetic overlap. Results revealed a main effect racial ancestry, $F(2, 637) = 3.06$, $p = .048$, $\eta_p^2 = 0.01$. Results also revealed a main effect of biogeographical ancestry effect, $F(2, 637) = 55.17$, $p < .001$, $\eta_p^2 = 0.15$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 637) = 3.39$, $p = .009$, $\eta_p^2 = 0.02$.

Complex Racial Categorization. *Black racial categorization*. Results revealed a main effect of biogeographical ancestry, $F(2, 637) = 37.63$, $p < .001$, $\eta_p^2 = 0.11$. Results also revealed a a main effect of racial ancestry, $F(2, 637) = 19.19$, $p < .001$, $\eta_p^2 = 0.06$. Last, results revealed no biogeographical by racial ancestry interaction, $F(4, 637) = 2.11$, $p = .078$, $\eta_p^2 = 0.01$.

White racial categorization. Results revealed a main effect of racial ancestry, $F(2, 636) = 11.77$, $p < .001$, $\eta_p^2 = 0.04$. Results also revealed a main effect of biogeographical ancestry,

$F(2, 636) = 42.92$, $p < .001$, $\eta_p^2 = 0.12$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 636) = 4.96$, $p = .001$, $\eta_p^2 = 0.03$.

Cultural Practices. Results revealed a main effect of racial ancestry, $F(2, 637) = 8.97$, $p < .001$, $\eta_p^2 = 0.03$. Results also revealed a main effect of biogeographical ancestry, $F(2, 637) = 14.81$, $p < .001$, $\eta_p^2 = 0.04$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 637) = 2.94$, $p = .020$, $\eta_p^2 = 0.02$.

Skin tone. Results revealed a main effect of racial ancestry, $F(2, 637) = 8.27$, $p < .001$, $\eta_p^2 = 0.03$. Results also revealed a main effect of biogeographical ancestry, $F(2, 637) = 34.92$, $p < .001$, $\eta_p^2 = 0.10$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 637) = 3.08$, $p = .016$, $\eta_p^2 = 0.02$.

Racial Stereotyping. Results revealed no main effect of biogeographical ancestry, $F(2, 635) = 0.20$, $p = .817$, $\eta_p^2 < 0.01$, no main effect of racial ancestry, $F(2, 635) = 2.83$, $p = .060$, $\eta_p^2 = 0.01$, or their two-way interaction, $F(4, 635) = 0.46$, $p = .767$ $\eta_p^2 < 0.01$.

Superhumanization. *Physicality.* Results revealed no main effect of racial ancestry, $F(2, 637) = 2.40$, $p = .092$, $\eta_p^2 = 0.01$, no main effect of biogeographical ancestry, $F(2, 637) = 2.10$, $p = .123$, $\eta_p^2 = 0.01$, or a racial ancestry by biogeographical ancestry interaction, $F(4, 637) = .54$, $p = .707$, $\eta_p^2 < 0.01$.

Pain tolerance. Results revealed no main effect of racial ancestry, $F(2, 637) = 0.91$, $p = .925$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 637) = 0.96$, $p = .384$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 637) = 0.48$, $p = .750$, $\eta_p^2 < 0.01$.

Illness/disease perceptions. *Physical illness*. Results revealed a main effect of racial ancestry, $F(2, 636) = 4.61$, $p = .010$, $\eta_p^2 = 0.01$. Results revealed no main effect of

biogeographical ancestry, $F(2, 636) = 1.82$, $p = .163$, $\eta_p^2 = 0.01$, or a biogeographical ancestry by racial ancestry interaction, $F(4, 636) = 1.93$, $p = .105$ $\eta_p^2 = 0.01$.

Mental illness. Results revealed no main effect of racial ancestry, $F(2, 636) = 1.78$, $p = .170$, $\eta_p^2 = 0.01$, no main effect of biogeographical ancestry, $F(2, 636) = 0.80$, $p = .450$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 636) = 1.30$, $p = .270$, $\eta_p^2 = 0.01$.

STD/I. Results revealed no main effect of racial ancestry, $F(2, 636) = 1.30$, $p = .274$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 636) = 0.61$, $p = .544$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 636) = .92$, $p = .453$, $\eta_p^2 = 0.01$.

Biological Difference. Results revealed no main effect of racial ancestry, $F(2, 637) = 0.39$, $p = .962$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 637) = 2.27$, $p = .104$, $\eta_p^2 = 0.01$, or their two-way interaction, $F(4, 637) = 0.23$, $p = .921$, $\eta_p^2 < 0.01$.

Moderated-mediations: The indirect effect of biogeographical ancestry X racial ancestry interaction through Black genetic overlap and Racial Categorization

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and Black racial categorization

Cultural Practices. Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and Black racial categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.050, -.009]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.07, *SE* = .01, 95%CI [-.970, -.040]. This indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.060, -.019], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.040, .019].

Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.049, -.007]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.06, *SE* = .02, 95%CI [-.093, -.026]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.03, *SE* = .01, 95%CI [-.055, -.013], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.037, .017].

Skin tone. Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and Black racial categorization in serial, *index* = -.04, *SE* = .01, 95%CI [-.068, -.012]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.09, *SE* = .02, 95%CI [-.130, -.052]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.05, *SE* = .01, 95%CI [-.077, -.025], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .02, 95%CI [-.050, .025].

Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap, *index* = -.05, *SE* = .02, 95%CI [-.097, -.016]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.12, *SE* = .03, 95%CI [-.191, -.066]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.07, *SE* = .02, 95%CI [-.116, -.033], and absent when the target had 1B/3W racial ancestry, *index* = -.02, *SE* = .03, 95%CI [-.074, .036].

Racial Stereotyping. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.003, .007]; or the indirect effect of

biogeographical ancestry on racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.025, .004].

Superhumanization. *Physicality.* Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.001, .013]; or the indirect effect of biogeographical ancestry on physicality through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.042, .002].

Pain tolerance. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.009, .003]; or the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.025, .009].

Illness/disease perceptions. *Physical illness*. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on perceptions of physical illness susceptibility through Black genetic overlap, *index* = .00, *SE* = .01, 95%CI [-.025, .001].

Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.013, .000].

Mental illness. Racial ancestry moderated the indirect effect of biogeographical ancestry on perceptions of mental illness susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.035, -.001]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .02, 95%CI [-.071, -.003]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -

.02, *SE* = .01, 95%CI [-.042, -.002], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.025, .011].

Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.006, .006]

STD/I. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.013, .002]; or the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.029, .010].

Biological Difference. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.011, .003]; or the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.005, .034].

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and White racial categorization

Cultural Practices. Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and White racial categorization in serial, *index* = -.02, *SE* = .01, 95%CI [-.030, -.004]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .01, 95%CI [-.058, -.019]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI

[-.034, -.010], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.021, .011].

Skin tone. Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and White racial categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.052, -.008]. Consistent with predictions, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.06, *SE* = .02, 95%CI [-.100, -.036]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.060, -.018], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.038, .020].

Racial Stereotyping. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.008, .001].

Superhumanization. *Physicality***.** Racial ancestry moderated the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.003, .024]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = .03, *SE* = .01, 95%CI [.013, .048]. However, inconsistent with predictions, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = .02, *SE* = .01, 95%CI [.006, .029], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.008, .018].

Pain tolerance. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.002, .011].

Illness/disease perceptions. *Physical illness*. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .007].

Mental illness. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.0001, .011].

STD/I. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .008].

Biological Difference. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .008].

Mediation: The indirect effect of biogeographical ancestry through genetic overlap and racial categorization.

Indirect effect of biogeographical ancestry through Black genetic overlap and Black racial categorization

Racial Stereotyping. The indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.032, .005], or

through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .008].

Superhumanization. *Physicality.* The indirect effect of biogeographical ancestry was not carried to physicality through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.055, .002], or through Black genetic overlap and Black racial categorization in serial, *index* = .01, *SE* = .00, 95%CI [-.001, .016].

Pain tolerance. The indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.032, .011], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.010, .004].

Illness/disease perceptions. *Physical illness*. The indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.014, .001].

Mental illness. The indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.006, .008].

STD/I. The indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.035, .013], or through Black genetic overlap and Black racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.014, .003].

Biological Difference. The indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .02, *SE* = .01, 95%CI [-.006, .042],

or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.013, .004].

Indirect effect of biogeographical ancestry through Black genetic overlap and White racial categorization

Racial Stereotyping. The indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.025, .011], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.010, .002].

Superhumanization. *Physicality***.** The indirect effect of biogeographical ancestry was carried to physicality through Black genetic overlap, *index* = -.03, *SE* = .02, 95%CI [-.066, -.006]. Consistent with predictions, this indirect effect was present when comparing the 50A/50E and 25A/75E targets, *index* = .06, *SE* = .03, 95%CI [.006, .131], and when comparing the 50A/50E and 75A/25E targets, *index* = -.06, *SE* = .03, 95%CI [-.137, -.006].

Pain tolerance. The indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.042, .005], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.002, .014].

Illness/disease perceptions. *Mental illness*. The indirect effect of biogeographical ancestry was not carried to perceptions mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.000, .014].

STD/I. The indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.045, .006],

or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.005, .011].

Biological Difference. The indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.014, .034], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.005, .010].

Biological Racial Essentialism

Black genetic overlap. Results revealed a main effect racial ancestry, $F(2, 636) = 3.36$, $p = .035$, $\eta_p^2 = 0.01$. Results also revealed a main effect of biogeographical ancestry effect, $F(2, 636) = 54.45$, $p < .001$, $\eta_p^2 = 0.15$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 636) = 3.12$, $p = .015$, $\eta_p^2 = 0.02$.

Complex Racial Categorization. *Black racial categorization*. Results revealed a main effect of biogeographical ancestry, $F(2, 636) = 37.22$, $p < .001$, $\eta_p^2 = 0.11$. Results also revealed a a main effect of racial ancestry, $F(2, 636) = 18.72$, $p < .001$, $\eta_p^2 = 0.06$. Last, results revealed a biogeographical by racial ancestry interaction, $F(4, 636) = 2.68$, $p = .031$, $\eta_p^2 = 0.02$.

Specifically, when the target's racial ancestry was unspecified, participants racially categorized the 50A/50E target as more Black than the 25A/75E target, but less Black than the 75A/25E target, $p < .001$ and $p = .002$, respectively: $F(2, 636) = 28.90$, $p < .001$. Planned contrasts also revealed that when the target had 1B/3W racial ancestry, participants racially categorized the 50A/50E target as more Black than the 25A/75E target, but less Black than the 75A/25E target, *ps* = .044, $F(2, 636) = 8.14$, $p < .001$. Last, planned contrasts revealed that when the target had 3B/1W racial ancestry, participants racially categorized the 50A/50E target as

Black no differently from the 25A/75E target, $p = .130$, but less Black than the 75A/25E target, $p = .019$: $F(2, 636) = 7.59$, $p = .001$.

In addition, when the target had 25A/75E biogeographical ancestry, participants racially categorized the unspecified target as less Black than both the1B/3W and 3B/1W targets, $ps < .001$: $F(2, 636) = 17.94$, $p < .001$. When the target had 50A/50E biogeographical ancestry, participants racially categorized the unspecified target as Black no differently from the 1B/3W target, $p = .164$, but less Black than the 3B/1W target, $p = .001$: $F(2, 636) = 5.55$, $p = .004$. Last, when the target had 75A/25E biogeographical ancestry, there were no differences in Black racial categorization: $F(2, 636) = 2.65$, $p = .073$.

White racial categorization. Results revealed a main effect of racial ancestry, $F(2, 635) = 11.22$, $p < .001$, $\eta_p^2 = 0.03$. Results also revealed a main effect of biogeographical ancestry, $F(2, 635) = 41.67$, $p < .001$, $\eta_p^2 = 0.12$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 635) = 5.46$, $p < .001$, $\eta_p^2 = 0.03$.

Cultural Practices. Results revealed a main effect of racial ancestry, $F(2, 636) = 8.71$, $p < .001$, $\eta_p^2 = 0.03$. Results also revealed a main effect of biogeographical ancestry, $F(2, 636) = 14.71$, $p < .001$, $\eta_p^2 = 0.04$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 636) = 3.79$, $p = .005$, $\eta_p^2 = 0.02$.

Skin tone. Results revealed a main effect of racial ancestry, $F(2, 636) = 8.30$, $p < .001$, $\eta_p^2 = 0.03$. Results also revealed a main effect of biogeographical ancestry, $F(2, 636) = 34.42$, $p < .001$, $\eta_p^2 = 0.10$. Last, results also revealed a biogeographical ancestry by racial ancestry interaction, $F(4, 636) = 3.30$, $p = .011$, $\eta_p^2 = 0.02$.

Racial Stereotyping. Results revealed no main effect of biogeographical ancestry, $F(2, 635) = 0.25$, $p = .779$, $\eta_p^2 < 0.01$, no main effect of racial ancestry, $F(2, 635) = 2.94$, $p = .054$, $\eta_p^2 = 0.01$, or their two-way interaction, $F(4, 635) = 0.50$, $p = .737$ $\eta_p^2 < 0.01$.

Superhumanization. *Physicality.* Results revealed a main effect of racial ancestry, $F(2, 636) = 3.53$, $p = .030$, $\eta_p^2 = 0.01$. Results results revealed no main effect of biogeographical ancestry, $F(2, 636) = 1.55$, $p = .212$, $\eta_p^2 < 0.01$, or a racial ancestry by biogeographical ancestry interaction, $F(4, 636) = 1.10$, $p = .356$, $\eta_p^2 < 0.01$.

Pain tolerance. Results revealed no main effect of racial ancestry, $F(2, 636) = 0.22$, $p = .804$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 636) = 1.06$, $p = .248$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 636) = 0.42$, $p = .798$, $\eta_p^2 < 0.01$.

Illness/disease perceptions. *Physical illness*. Results revealed a main effect of racial ancestry, $F(2, 635) = 4.05$, $p = .018$, $\eta_p^2 = 0.01$. Results revealed no main effect of biogeographical ancestry, $F(2, 635) = 1.95$, $p = .143$, $\eta_p^2 = 0.01$, or a biogeographical ancestry by racial ancestry interaction, $F(4, 635) = 1.61$, $p = .170$ $\eta_p^2 = 0.01$.

Mental illness. Results revealed no main effect of racial ancestry, $F(2, 635) = 1.78$, $p = .170$, $\eta_p^2 = 0.01$, no main effect of biogeographical ancestry, $F(2, 635) = 0.64$, $p = .528$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 635) = 1.19$, $p = .314$, $\eta_p^2 = 0.01$.

STD/I. Results revealed no main effect of racial ancestry, $F(2, 635) = 1.18$, $p = .307$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 635) = 0.54$, $p = .585$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 635) = .80$, $p = .524$, $\eta_p^2 = 0.01$.

Biological Difference. Results revealed no main effect of racial ancestry, $F(2, 636) = 0.23$, $p = .792$, $\eta_p^2 < 0.01$, no main effect of biogeographical ancestry, $F(2, 636) = 1.96$, $p = .141$, $\eta_p^2 < 0.01$, or their two-way interaction, $F(4, 636) = 0.31$, $p = .85$, $\eta_p^2 < 0.01$.

Moderated-mediations: The indirect effect of biogeographical ancestry X racial ancestry interaction through Black genetic overlap and Racial Categorization

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and Black racial categorization

Cultural Practices. Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and Black racial categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.050, -.009]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.07, *SE* = .02, 95%CI [-.097, -.040]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.060, -.019], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.040, .018].

Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap, *index* = -.03, *SE* = .01, 95%CI [-.049, -.007]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.06, *SE* = .02, 95%CI [-.096, -.028]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.03, *SE* = .01, 95%CI [-.058, -.015], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.036, .017].

Skin tone. Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and Black racial categorization in serial, *index* = -.04, *SE* = .01, 95%CI [-.068, -.012]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.09, *SE* = .02, 95%CI [-.133, -.053]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.05, *SE* = .01,

95%CI [-.081, -.027], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .02, 95%CI [-.053, .026].

Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap, *index* = -.05, *SE* = .02, 95%CI [-.097, -.015]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.12, *SE* = .03, 95%CI [-.192, -.066]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.07, *SE* = .02, 95%CI [-.118, -.033], and absent when the target had 1B/3W racial ancestry, *index* = -.02, *SE* = .03, 95%CI [-.076, .035].

Racial Stereotyping. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.003, .007]; or the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.025, .004].

Superhumanization. *Physicality***.** Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.005, .009]; or the indirect effect of biogeographical ancestry on physicality through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.036, .008].

Pain tolerance. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.007, .005]; or the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.028, .006].

Illness/disease perceptions. *Physical illness*. Racial ancestry moderated the indirect effect of biogeographical ancestry on perceptions of physical illness susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.040, -.003]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .02, 95%CI [-.080, -.009]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.048, -.005], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.029, .012].

Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.011, .001].

Mental illness. Racial ancestry moderated the indirect effect of biogeographical ancestry on perceptions of mental illness susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.038, -.002]. Specifically, this indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.04, *SE* = .02, 95%CI [-.076, -.008]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.046, -.005], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.027, .012].

Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.005, .006].

STD/I. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and Black racial categorization in serial,

index = .00, *SE* = .00, 95%CI [-.012, .003]; or the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.031, .007].

Biological Difference. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.010, .003]; or the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.008, .032].

Indirect effect of biogeographical ancestry x racial ancestry through Black genetic overlap and White racial categorization

Cultural Practices. Racial ancestry moderated the indirect effect of biogeographical ancestry on cultural practice through Black genetic overlap and White racial categorization in serial, *index* = -.01, *SE* = .01, 95%CI [-.028, -.004]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.03, *SE* = .01, 95%CI [-.056, -.017]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.02, *SE* = .01, 95%CI [-.033, -.010], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.021, .010].

Skin tone. Racial ancestry moderated the indirect effect of biogeographical ancestry on skin tone through Black genetic overlap and White racial categorization in serial, *index* = -.03, *SE* = .01, 95%CI [-.053, -.008]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = -.06, *SE* = .02, 95%CI [-.101, -.036]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = -.04, *SE* = .01, 95%CI [-.060, -.018], and absent when the target had 1B/3W racial ancestry, *index* = -.01, *SE* = .01, 95%CI [-.039, .020].

Racial Stereotyping. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on racial stereotyping through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.008, .002].

Superhumanization. *Physicality.* Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physicality through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.003, .023]. This indirect effect was strongest when the target's racial ancestry was not specified, *index* = .03, *SE* = .01, 95%CI [.010, .048]. However, this indirect effect was present, but relatively weaker, when the target had 3B/1W racial ancestry, *index* = .02, *SE* = .01, 95%CI [.005, .029], and absent when the target had 1B/3W racial ancestry, *index* = .00, *SE* = .01, 95%CI [-.008, .018].

Pain tolerance. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on pain tolerance through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.0004, .012].

Illness/disease perceptions. *Physical illness*. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on physical illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.003, .008].

Mental illness. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [.000, .011].

STD/I. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on STD/I susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.003, .010].

Biological Difference. Racial ancestry did not moderate the indirect effect of biogeographical ancestry on biological difference through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.003, .010].

Mediation: The indirect effect of biogeographical ancestry through genetic overlap and racial categorization.

Indirect effect of biogeographical ancestry through Black genetic overlap and Black racial categorization

Racial Stereotyping. The indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.032, .005], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.004, .009].

Superhumanization. *Physicality***.** The indirect effect of biogeographical ancestry was not carried to physicality through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.050, .010], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.006, .011].

Pain tolerance. The indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.036, .007], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.009, .006].

Illness/disease perceptions. *Physical illness*. The indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.013, .002].

Mental illness. The indirect effect of biogeographical ancestry was not carried to perceptions of mental illness susceptibility through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.006, .009].

STD/I. The indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.040, .010], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.014, .004].

Biological Difference. The indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.001, .040], or through Black genetic overlap and Black racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.013, .004].

Indirect effect of biogeographical ancestry through Black genetic overlap and White racial categorization

Racial Stereotyping. The indirect effect of biogeographical ancestry was not carried to racial stereotyping through Black genetic overlap, *index* = -.01, *SE* = .01, 95%CI [-.026, .011], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [-.010, .002].

Superhumanization. *Physicality***.** The indirect effect of biogeographical ancestry was carried to physicality through Black genetic overlap, *index* = -.03, *SE* = .02, 95%CI [-.066, -.001]. Consistent with predictions, this indirect effect was present when comparing the 50A/50E and 25A/75E targets, *index* = .03, *SE* = .01, 95%CI [.009, .048], and when comparing the 50A/50E and 75A/25E targets, *index* = -.03, *SE* = .01, 95%CI [-.053, -.011].

Pain tolerance. The indirect effect of biogeographical ancestry was not carried to pain tolerance through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.047, .000], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .00, 95%CI [.000, .016].

Illness/disease perceptions. *Mental illness*. The indirect effect of biogeographical ancestry was not carried to perceptions mental illness susceptibility through Black genetic overlap and White racial categorization in serial, *index* = .01, *SE* = .01, 95%CI [.000, .015].

STD/I. The indirect effect of biogeographical ancestry was not carried to perceptions of STD/I susceptibility through Black genetic overlap, *index* = -.02, *SE* = .01, 95%CI [-.051, .002], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.004, .013].

Biological Difference. The indirect effect of biogeographical ancestry was not carried to biological difference through Black genetic overlap, *index* = .01, *SE* = .01, 95%CI [-.019, .032], or through Black genetic overlap and White racial categorization in serial, *index* = .00, *SE* = .01, 95%CI [-.004, .013].

IRB FORM

Approval Notice
Continuing Review

October 5, 2017

Courtney Bonam, PhD
Psychology
Psychology
1007 W Harrison, M/C 285
Chicago, IL 60607
Phone: (312) 355-0808

RE: Protocol # 2012-0591
"Race and Space"

Dear Dr. Bonam:

Please note that this research did not have Institutional Review Board (IRB) approval beginning at 12:01 a.m. on 14 September 2017 and until IRB approval was again granted on 3 October 2017.

Please note that stamped .pdfs of all approved recruitment and consent documents have been uploaded to OPRSLive, and can be accessed under "Approved Documents" tab. Please remember to use only those approved documents to recruit and enroll subjects into this research project. OPRS/IRB no longer issues paper letters or stamped/approved documents.

Your Continuing Review was reviewed and approved by the Expedited review process on October 3, 2017. You may now continue your research.

Please note the following information about your approved research protocol:

Protocol Approval Period: October 3, 2017 - October 3, 2018
Approved Subject Enrollment #: 10000 (Currently 2095 subjects enrolled).
Additional Determinations for Research Involving Minors: The Board determined that this research satisfies 45CFR46.404, research not involving greater than minimal risk. Therefore, in accordance with 45CFR46.408, the IRB determined that only one parent's/legal guardian's permission/signature is needed. Wards of the State may not be enrolled unless the IRB grants specific approval and assures inclusion of additional protections in the research required under 45CFR46.409. If you wish to enroll Wards of the State contact OPRS and refer to the tip sheet.
Performance Sites: UIC
Sponsor: RTOG, Society for Psychological Study of Social Issues
PAF#: Not available,Not available

Grant/Contract No: Not available,Not available
Grant/Contract Title: Not available,Not available
Research Protocol(s):

a) Race & Space Research Protocol; Version 9; 05/18/2017

Recruitment Material(s):

a) Professional Org Recruitment Email; Version 3; 07/11/2012
b) UIC Recruitment Email; Version 3; 07/11/2012
c) Online Study Description; Version 3; 07/11/2012
d) Your Opinions Psychology Mass Testing Survey (for use in mass testing only); Version 1; 11/05/2012
e) Your Opinions Psychology Subject Pool Mass Testing Survey 2 (for use in mass testing only); Version 1; 01/17/2014
f) Your Opinions Psychology Subject Pool Mass Testing Survey 3 (for use in mass testing only); Version 1; 01/17/2014
g) Your Opinions Psychology Subject Pool Mass Testing Survey (for use in mass testing only); Version 1; 08/17/2015
h) Flyer; Version 4; 09/27/2016
i) Raffle Flyer; Version 1; 05/18/2017
j) Raffle Recruitment Email; Version 1; 05/18/2017
k) Volunteer Recruitment Message; Version 2; 06/27/2017
l) Volunteer Flyer; Version 2; 06/27/2017

Informed Consent(s):

a) Debrief; Version 1; 07/11/2012
b) Lab Consent; Version 4; 08/31/2015
c) Online Consent; Version 8; 06/27/2017
d) Waiver of Signed Consent Document granted under 45 CFR 46.117 for online consent

Parental Permission(s):

a) A Waiver of Parental Permission has been granted under 45 CFR 46.116(d) and 45 CFR 46.408(c); however, as per UIC Psychology Subject Pool policy, at least one parent must sign the Blanket Parental Permission document prior to the minor subject's participation in the UIC Psychology Subject Pool.

Your research meets the criteria for expedited review as defined in 45 CFR 46.110(b)(1) under the following specific category(ies):

(6) Collection of data from voice, video, digital, or image recordings made for research purposes., (7) Research on individual or group characteristics or behavior (including but not limited to research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices and social behavior) or research employing survey, interview, oral

history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies.

Please note the Review History of this submission:

Receipt Date	Submission Type	Review Process	Review Date	Review Action
09/14/2017	Continuing Review	Expedited	10/03/2017	Approved

Please remember to:

→ Use your **research protocol number** (2012-0591) on any documents or correspondence with the IRB concerning your research protocol.

→ Review and comply with all requirements on the guidance,
"UIC Investigator Responsibilities, Protection of Human Research Subjects"
(http://research.uic.edu/irb/investigators-research-staff/investigator-responsibilities).

Please note that the UIC IRB has the prerogative and authority to ask further questions, seek additional information, require further modifications, or monitor the conduct of your research and the consent process.

Please be aware that if the scope of work in the grant/project changes, the protocol must be amended and approved by the UIC IRB before the initiation of the change.

We wish you the best as you conduct your research. If you have any questions or need further help, please contact OPRS at (312) 996-1711 or me at (312) 413-1518. Please send any correspondence about this protocol to OPRS at 203 AOB, M/C 672.

Sincerely,

Alma Milat, BS
IRB Coordinator, IRB # 2
Office for the Protection of Research Subjects

Enclosure(s): Following approved recruitment and consent documents have been uploaded under "approved documents" tab in OPRSLive:

1. **Informed Consent Document(s):**
   a) Debrief; Version 1; 07/11/2012
   b) Lab Consent; Version 4; 08/31/2015
   c) Online Consent; Version 8; 06/27/2017

2. Recruiting Material(s):

a) Professional Org Recruitment Email; Version 3; 07/11/2012
b) UIC Recruitment Email; Version 3; 07/11/2012
c) Online Study Description; Version 3; 07/11/2012
d) Your Opinions Psychology Mass Testing Survey (for use in mass testing only); Version 1; 11/05/2012
e) Your Opinions Psycholgy Subject Pool Mass Testing Survey 2 (for use in mass testing only); Version 1; 01/17/2014
f) Your Opinions Psychology Subject Pool Mass Testing Survey 3 (for use in mass testing only); Version 1; 01/17/2014
g) Your Opinions Psychology Subject Pool Mass Testing Survey (for use in mass testing only); Version 1; 08/17/2015
h) Flyer; Version 4; 09/27/2016
i) Raffle Flyer; Version 1; 05/18/2017
j) Raffle Recruitment Email; Version 1; 05/18/2017
k) Volunteer Recruitment Message; Version 2; 06/27/2017
l) Volunteer Flyer; Version 2; 06/27/2017

cc: Michael E. Ragozzino, Psychology, M/C 285
OVCR Administration, M/C 672

VITA

NAME:	Drexler Denzie James
EDUCATION:	B.Sc. Psychology, Illinois Institute of Technology, Chicago, IL, 2013 M.A., Social and Personality Psychology, University of Illinois at Chicago, Chicago, IL, 2016
TEACHING:	Department of Psychology, University of Illinois at Chicago, Chicago, Illinois, 2013 – present
PUBLICATIONS:	James, D. (2017). Internalized Racism and Past-Year Major Depressive Disorder among African Americans: The Role of Ethnic Identity and Self-esteem. *Journal of Racial and Ethnic Health Disparities.* 4, 659-4670. DOI: 10.1007/s40615-016-0269-1 Molina, K.M., & James, D. (2016). Discrimination, Internalized Racism, and Depression: A Comparative Study of African American and Afro-Caribbean Adults in the U.S. *Group Processes & Intergroup Relations* Kosyluk, K. A., Corrigan, P. W., Jones, N., James, D., Abelson S., & Malcolm A. (2016). Campus Solidarity Campaign (CSC): Developing a Campaign to Promote an Environment of Solidarity and Support on College Campuses for Students with Mental Illness. *Rehabilitation Education*. Jones, N., Corrigan, P. W., James, D., Parker, J., & Larson, N. (2013). Peer support, self-determination, and treatment engagement: A qualitative investigation. *Psychiatric Rehabilitation Journal*, *36*(3), 209.
HONORS/ AWARDS:	Participant, Summer Institute in Social Psychology (SISPP), SPSP & NSF Chancellor's Graduate Research Fellowship College of Liberal Arts and Sciences Travel Award Clara Mayo Grant Summer Research Award Presidential Scholars Undergraduate Research Award
PROFESSIONAL MEMBERSHIP:	Association for Psychological Science Psi Chi International Honor Society Society for Personality and Social Psychology Society for the Psychological Study of Social Issues
SERVICE:	Committee on Graduate Studies Student representative Diversity Advancement Committee