Sociocultural Patterning of Biological Processes: An Examination with Biological Health and Brain Functioning

by

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Dedication

I dedicate this dissertation to my mom and dad, who taught me to approach my work and other things in life with both a sense of urgency and ease.

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Abstract

This dissertation investigates the relationship between the biological system and the sociocultural environment. For a long time, the "nature" (biological processes that are considered as part of the genetic inheritance) and "nurture" (external environment) are thought of as two separate sources of influence on behaviors. However, recent work demonstrate that the biological system is inherently embedded in and shaped by the sociocultural environment. My dissertation builds on this emerging line of work and extends the current literature by examining three questions. In Chapter 1, I provide the theoretical background of my research questions. In Chapter 2, I present a study examining how human culture may shape the *structural* properties of the brain. I showed that there exists systematic cultural difference in the GM volume of several brain regions. Importantly, this cultural difference is more pronounced among individuals carrying the "plasticity allele" which predisposes them to be more sensitive to the cultural context. In Chapter 3, I focus on the aspect of the sociocultural environment that is characterized by inequality, and I investigate its consequences on biological health. In the first study, I showed that racial residential segregation and income inequality jointly exacerbated the COVID-19 fatalities in large U.S. cities during the pandemic. In the second study, I showed that while the experience of discrimination can be stressful, recognizing negative events as discrimination and reporting the discrimination could reflect a resilient psyche, which in turn predicts better long-term health. In Chapter 4, I demonstrate how biological health and brain functioning are inherently related to each other. I found that poor biological health is associated with a less optimal functional organization of the whole-brain network, over and above of the effect of

aging and other factors that can compromise brain functioning. Finally, in Chapter 5, I summarize the findings and identify future directions for this research.

Chapter 1 Introduction

Inquiries about nature versus nurture have always been at the heart of the investigation of the human mind. This debate starts with philosophers at early times. For instance, John Locke famously coined the term "*tabula rasa*", suggesting that the human mind is born as a "blank slate" and all mental contents are acquired from environments (Locke, 1836). Other philosophers, such as Plato and Descartes, proposed innatism, arguing that the mind is pre-wired with innate knowledge and ideas. This inquiry about the influence of innate inheritance and external environment on mind and behavior permeates the past century of work in psychology. On the one hand, the investigation of how environment, or "situations" are powerful determinants of one's behavior has been the main thrust of social psychology, starting with the prominent theories by Kurt Lewin (Lewin, 1936) and lasting to the past three decades of research on the influence of human culture (Markus & Kitayama, 1991), among others. On the other hand, the advances of genetic and heritability studies have also found firm evidence that intelligence, personality traits, and various psychological disorders such as schizophrenia and autism spectrum disorders are in certain degrees genetically inherited (e.g., Davies et al., 2011; Vukasović & Bratko, 2015). Hence, most modern scientists would take the perspective that both biological processes ("nature") and environmental contexts ("nurture") contribute substantially to the development of mind and behaviors (Belsky & Pluess, 2009b; Bleidorn et al., 2010; W. A. Collins et al., 2000).

However, recent years of research has begun to show that it is not always easy to tease apart the influence of nature versus nurture, and in the many cases the influence of the two are

not additive but interactive. For instance, previous studies examining *Gene-Environment Interaction* have found that the effect of genetic inheritance (e.g., genetic predisposition to depression) depends on whether one's environment is stable and resourceful, or unpredictable and stressful (Caspi et al., 2003). Similar evidence emerges from work adopting the framework of gene-culture coevolution, showing that cultural practices can alter, and in some cases completely mask genetic heritability (Uchiyama et al., 2021).

Moreover, it is increasingly recognized that a clear dichotomy of nature versus nurture is actually not feasible, as nature and nurture can shape one another. For instance, epigenetic research shows that the environment can modulate the functioning of genes by changing how a particular gene is being expressed (Cole, 2014; Holliday et al., 1990). This pattern of nurture shaping nature was initially demonstrated in animal models, but later generalized to human beings as well (Heijmans et al., 2008; Meaney, 2001). Hence, emerging evidence suggests that biological processes, including genetic processes, can be modulated by the environment. The sociocultural environment may thus have a powerful influence on various physiological and neural processes – the processes typically considered as part of the biological inheritance by earlier research (Kitayama & Salvador, 2017; Kitayama & Uskul, 2011; Kitayama & Yu, 2020).

The present dissertation is inspired by this emerging line of work and will investigate three questions along this general theme. In chapter two, I examine how culture, a set of shared values, norms, and practices may shape the structural properties of the human brain. In chapter three, I focus on systemic racism, another critical element of sociocultural environment that many marginalized racial/ethnic groups in the U.S. are living with. I test how systemic racism is linked to biological health across two studies, one on the individual-level, and the other on the level of public health. In chapter four, I examine the interconnection between brain and

biological health. The aging literature has provided ample evidence on how the brain, both its structure and functioning, changes through aging. However, this literature has largely focused on aging per se and ignored the physiological effects of stressors and adversities that can accumulate in one's body throughout the life course. In the current work, I test how physiological health, a viable reflection of accumulated stress and adversities from the environment, is associated with the functional organization of the brain. In the remaining of this chapter, I briefly introduce the theoretical background for each question.

Cultural Shaping of Brain Structures

The past three decades of research in cultural psychology has revealed systematic cultural differences in various psychological processes, such as the self, emotion, cognition, and motivation (Heine et al., 1999; Markus & Kitayama, 1991; Nisbett et al., 2001; Tsai et al., 2006). While the literature heavily focuses on the comparison of East (individuals of East Asian descent, such as Chinese, Japanese, and Korean) versus West (individuals of West European descent, such as European Americans and Canadians), recent studies testing individuals from other continents show consistent evidence that culture exerts profound influences on mind and behaviors (Krys et al., 2022; San Martin et al., 2018).

Prior work has mapped cultural differences in behaviors onto several fundamental cultural dimensions along which such differences can be better organized and understood. One such dimension is the independent versus interdependent self-construal (Markus & Kitayama, 1991). Westerners value the independent of the self from others, and they tend to construe the self as defined by a set of internal attributes such as goals, desires, traits, and the like (Markus & Kitayama, 1991; Savani et al., 2008). In contrast, Easterners value the interdependence of the self with others, and they tend to prioritize social roles, obligations, and duties over personal

preferences and goals (Heine, 2001; Markus & Kitayama, 1991; Triandis, 1989). This difference in the construal of the self is thought to be the root of many other cultural differences in behaviors, such as cognitive style, emotion expression, and attribution.

In recent years, the adoption of neuroscience methods to cultural psychology, or *cultural neuroscience*, has further identified the neural basis of cultural differences in self-construal and related behaviors. For instance, one classical work shows that culture modulates the neural basis of self- and other-representation (Zhu et al., 2007). It is well-known that the medial prefrontal cortex (mPFC) is selectively recruited when people think and reflect on oneself. Zhu et al. compared Westerners and Chinese and found that while both groups use mPFC to process information about the self, Chinese use mPFC to process information about their closed others (e.g., mother) as well. This finding suggests that cultural differences in behaviors are not descriptive but fundamentally ingrained into one's brain functioning. Hence, cultural neuroscience research has begun to reveal that human brain is socioculturally patterned (Kitayama & Salvador, 2017; Kitayama & Yu, 2020).

So far, although evidence is mounting that culture can shape the functional aspect of the brain, it remains largely unknown if cultural influence can go deeper and shape the *structural* aspect of the brain, such as gray matter (GM) volume and structural connectivity. In my work I focus on the GM volume, a well-validated index of the capacity of a brain region in exerting its functions. This possibility that culture may shape the GM volume of certain brain regions is inspired by (1) the principle of neuroplasticity, which suggests that a brain region may expand in its GM volume if it is repetitively recruited in carrying out certain task (Maguire et al., 2000; Rosenzweig et al., 1962); and (2) framework conceptualizing engagement with culture as carrying out various "cultural tasks" that are in line with the values and norms of the culture

(Kitayama et al., 2009a). Therefore, because different cultures sanction different set of "cultural tasks" that are repetitively carried out by the individuals, it is plausible that there exists systematic cross-cultural variation in the GM volume of brain regions involved in carrying out those tasks. In chapter two, I test this possibility using structural brain images of a group of European American and East Asian undergraduates.

Systemic Racism and Biological Health

The cultural psychology research so far has predominantly adopted a cross-cultural approach, comparing individuals from different cultural regions. The field is still in its initial stage in showing the dynamic of within-culture variations. Some recent cultural psychology work has suggested that one important aspect of within-culture variations lies in the social status hierarchy such as social class (Kraus et al., 2009; Miyamoto, 2017; Park et al., 2013). In the U.S., for instance, individuals of middle-class background are more likely to show the typical psychological profiles of European Americans previously identified by cultural psychology literature. They are more independent, and they value and pursue freedom, choice, and personal goals. Individuals of working-class background in comparison tend to be more interdependent. They are more likely to adjust to the needs of others and the demands of the social context. This difference in psychological tendencies is thought to be afforded by the differential access to economic capital, power, and status across social classes (Stephens et al., 2014).

There have been some efforts trying to link this line of work to the large body of literature documenting the wide disparities in a variety of outcomes such as educational and health across social classes (Stephens et al., 2012, 2014). One socially constructed variable that is highly conflated with social class in the U.S. is race. Racial/ethnic minority groups are more likely to occupy the lower social class and be deprived of various socioeconomic resources, as a

result of the system of racism (Pew Research Center, 2011, 2016a). The deprivation of resources, as well as the systemic racism ingrained in the discriminatory practices by the institutions of employment, education, housing, and criminal justice serve as a powerful social context underlying biological processes for racial/ethnic minorities (Williams et al., 2019; Williams & Collins, 1995; Williams & Mohammed, 2009), and they are likely to be the root cause of the well-documented racial health disparity.

Although racial disparity in health has been well studied, the present work examines two questions that are left unanswered by prior work. First, the COVID-19 pandemic hit the U.S. in March 2020. Since the very early stage of the pandemic, the racial health disparity was laid bare as marginalized racial/ethnic groups, such as Black and Hispanic Americans, accounted for a disproportionate number of cases and deaths (Centers for Disease Control and Prevention, 2020). The disproportionate sufferings of racial minorities are likely to be due to the greater pre-existing health conditions among these groups, as a result of the system of racism in American society that has deprived them of various social resources. However, it is unclear what structural factors underlying systemic racism are responsible for the human toll during the pandemic. In the first study of chapter three, I built on prior literature in sociology and demography and examined how racial residential segregation and income inequality jointly exacerbated the COVID-19 fatalities in large U.S. cities.

Second, although it is well-established that structural inequality compromises health among members of marginalized racial/ethnic groups (Kawachi et al., 1997; Williams & Collins, 2001), evidence is mixed regarding how the subjective perception of inequality and discrimination is related to biological health. Because the experience of discrimination is threatening and stressful, many studies have linked perceived discrimination to poor health

outcomes (Pascoe & Richman, 2009; Williams et al., 2003). However, some studies in the literature have found null effect, and sometimes a positive effect of perceived discrimination on health (Fuller-Rowell et al., 2012; Krieger & Sidney, 1996; LaVeist et al., 2001). Researchers have thus argued that it is also important to consider the extent to which individuals *report* experiences of discrimination reflects how they are interpreting their social reality (Crocker & Major, 1989; Fuller-Rowell et al., 2012; Major et al., 2002). Hence, although the experience of discrimination is stressful as it occurs, the acknowledgement and reporting of discrimination could reflect a well-adjusted and resilience psyche among racial minorities, which could entail long-term health benefits (Fuller-Rowell et al., 2012; LaVeist et al., 2001; Sellers et al., 1998). In the second study of chapter three, I use a large community sample of Black and White Americans that has been longitudinally followed up over 15 years and test how perceived discrimination predicts mortality risk.

Biological Health and Functional Organization of the Brain

The foregoing discussion suggests that the sociocultural context, no matter one's cultural environment or the systemic racism/inequality inscribed to daily social interactions and institutions, has profound influences on both biological health in the body and neural processes in the brain. One implication of this conclusion is that since both the body and the brain are intricately shaped by the environment, it is possible that there is a tight link between biological health and brain functioning as well. Indeed, it is commonly known that the brain functions better when the person is healthy. For instance, poverty and other adversities in life often lead to worse physical health as well as impaired cognitive performances (Evans & Schamberg, 2009; LeClere & Soobader, 2000; Mani et al., 2013). Similarly, aging is linked to deteriorating

physical health and cognitive decline (Blatter et al., 1995; Hedden & Gabrieli, 2004). Physical exercise, on the other hand, often improves cognition (Åberg et al., 2009; Hillman et al., 2008).

So far, the literature has linked biological health to both changes in structural (e.g., cortical volume) and functional (e.g., functional activation) features of various brain regions (Åberg et al., 2009; Kraynak et al., 2018; Sapolsky, 1994). However, the optimal functioning of the brain requires not only well-operated brain regions, but also the efficient organization and connections between different regions. Recent advances in network neuroscience have indeed demonstrated that the brain operates like a network (Newman, 2006; Power et al., 2011; Rubinov & Sporns, 2010), and certain features of this network are tightly linked to optimal cognitive functioning (J. R. Cohen & D'Esposito, 2016; E. S. Duncan & Small, 2016; Hilger et al., 2017). Therefore, in chapter four, I focus on one critical feature of the functional organization of the brain – modularity. I test whether biomarkers of bodily health are associated with modularity of the functional brain network, and whether modularity in turn predicts cognitive performance.

Chapter 2 Cultural Shaping of the Brain Structures[1]

One core principle in the study of psychology and neuroscience is that the brain shows plasticity in its function and structure in response to environmental input (Rosenzweig, 1996). In human work, neuroplasticity has been demonstrated by work showing plastic increase of gray matter (GM) volume through extensive training in various tasks, such as spatial navigation and playing musical instruments (Gaser & Schlaug, 2003; Maguire et al., 2000). So far, it is unclear whether neuroplasticity is implicated in cross-cultural variations in regionally specific GM volume. Different cultures sanction varying sets of "cultural tasks" (Kitayama et al., 2009b). Hence, depending on the specific tasks that are positively sanctioned, individuals engaged in different cultures may recruit different regions of the brain to varying extents. The field of cultural neuroscience has grown rapidly over the last decade (Kitayama et al., 2019). However, it has not systematically examined cultural variation in GM volume in the brain, and whether this variation is due to experience in engaging culturally scripted behaviors. The present work will seek to fill this gap.

Cultural Variations in Psychological Processes

The cultural psychology literature has documented profound differences in cognition, emotion, and motivation between Westerners (primarily North Americans of European descent) and Easterners (primarily those of Chinese, Japanese, and Korean descent). Prior theorization has argued that these differences are rooted in the fundamental way of how the self is construed and

[1] Chapter 2 is based on Yu et al. (2019) published in *Cerebral Cortex*, and Kitayama, Yu et al. (2020) published in *Social Cognitive and Affective Neuroscience*.

understood (Markus & Kitayama, 1991). Westerners are more likely to view the self as independent and perceive the self as defined by a set of internal attributes such as goals, desires, traits, and the like (Markus & Kitayama, 1991; Savani et al., 2008). In comparison, Easterners are more likely to view the self as interdependent. They perceive the self as embedded in relationships and thus defined by various relational characteristics such as roles, memberships, obligations, and duties (Heine, 2001; Markus & Kitayama, 1991; Triandis, 1989).

The independent and interdependent self-construal are reflected and reinforced through the culturally scripted behaviors that people perform in their daily life. Prior work has documented that independently orientated individuals, such as European Americans, tend to hold a strong valuation of their personal choices and are more likely to express their personal preferences (H. Kim & Markus, 1999; Savani et al., 2010). Because of their strong personal orientation, they prioritize self-interest and personal goals compared to social norms and expectations (Heine et al., 1999; Kitayama & Park, 2014; Varnum et al., 2014). Interdependently orientated individuals, such as East Asians, in contrast, tend to abide by social norms and expectations. They value social harmony and interpersonal obligations, and they readily attune to the needs and desires of closed others (Heine et al., 1999; Weisz et al., 1984). They are more likely to take others' perspective, and are more accurate in doing so (D. Cohen & Gunz, 2002; Wu & Keysar, 2007). We may then wonder, to the extent that individuals with different self-construal engage in a divergent set of behaviors that require the functioning of different brain regions, will there be a systematic difference in the structures of these brain regions as a function of independence versus interdependence?

Neuroplasticity and Cultural Variation in Cortical Gray Matter Volume

This hypothesis is inspired by the principle of neuroplasticity (Rosenzweig et al., 1962). Numerous human neuroimaging research have demonstrated that a given brain region may expand in its GM volume if it is repetitively recruited to carry out certain tasks. For instance, Maguire et al. (2000) tested a group of cab drivers in London and found that their posterior hippocampus, the brain region critical for spatial navigation, showed increased GM volume as a function of years of experience in the profession. Similar evidence exists for professional musicians (keyboard players) and brain regions governing motor control and auditory processing (Gaser & Schlaug, 2003), and individuals who are undergoing extensive training in performing classic three-ball cascade juggling and brain regions responsible for visuospatial processing (Draganski et al., 2004). Since different cultures sanction different set of "cultural tasks", and being enculturated entails repetitive engagement and eventually mastery of those tasks, it is plausible that there exists systematic cross-cultural variation in the GM volume of certain brain regions involved in carrying out those tasks.

As mentioned above, independent individuals value and express their personal choices and preferences, and they prioritize their own goals and interests. These behaviors are aligned with what neuroscientists have denoted as the "prefrontal functions". Certain prefrontal regions, especially the orbitofrontal cortex (OFC), has been linked to value-based judgment ("What do I like?") (O'Doherty, 2011) and the ability to sustain transitivity in personal preferences (Fellows 2011). It is thus found to be critical for goal pursuit behaviors based on one's own interests and preferences (O'Doherty, 2011; Rolls & Grabenhorst, 2008). Hence, independent individuals such as European Americans may exhibit greater GM volume in the OFC. Conversely, interdependent individuals pay greater attention to the needs and desires of others while downregulating personal goal pursuit. They are thus more likely to take the perspectives of others. Although

perspective taking, or "theory of mind", recruits several cortical regions, one region that is consistently involved is the temporoparietal junction (TPJ) (Samson et al., 2004; Saxe & Kanwisher, 2003; Schurz et al., 2014). Moreover, damages in TPJ result in a selective impairment of the ability in perspective taking (Apperly et al., 2004; Young et al., 2010). Therefore, we may expect that interdependent individuals such as East Asians may exhibit greater GM volume in the TPJ.

Prior work has provided emerging evidence for this hypothesis. For instance, Chee et al. (2010) compared young Singaporean Chinese and European American adults and found that Americans showed greater GM volume in the prefrontal regions, including the OFC. This pattern was replicated in later studies, which also found that Chinese showed greater GM volume in several temporal regions, including the TPJ (Huang et al., 2019; Tang et al., 2018). Moreover, Kitayama et al. (2017) tested Japanese and found that GM volume at the OFC was negatively correlated with interdependent self-construal, suggesting that variation in this region is linked to how one construes the self and the associated behavioral tendencies.

The Dopamine D4 Receptor Gene (*DRD4*)

One limitation of the work discussed so far is that all evidence is correlational in nature, so it does not inform causality. While it is possible that cultural difference in the GM volume results from repeated engagement in culturally sanctioned tasks, an alternative possibility is that the pre-existing brain differences cause the cultural difference in self-construal and behaviors. To address this thorny issue, I draw on the *Gene-Environment Interaction* literature. This literature has revealed that certain genetic variants, so-called "plasticity alleles", predispose people to be more susceptible to environmental influences, including cultural influences (Belsky & Pluess, 2009a; Kitayama et al., 2014; Sheese et al., 2007). Hence, if the cultural difference in GM

volume is found to be more pronounced among individuals carrying the "plasticity alleles" (i.e., those who are relatively more enculturated), this would add further support to the hypothesis that cultural difference in GM volume is due to cultural experience.

While several genetic variations have been found to modulate environmental influences (Set et al., 2014), one gene, the dopamine D4 receptor gene (*DRD4*), appears to be particularly important for cultural influences (Kitayama et al., 2014, 2016; Sasaki et al., 2013; Silveira et al., 2016; Tompson et al., 2018). Compared to the more common allele of *DRD4* (the 4-repeat allele), two evolutionarily more recent alleles emerged (the 7-repeat and 2-repeat allele, called 7/2-R hereafter) over the last 50,000 years (E. Wang et al., 2004). This time period coincides with the emergence of human culture, suggesting that these alleles may have coevolved with culture that unfolded during the period. Moreover, the 7/2-R are associated with increased dopamine signaling capacity in vivo (Asghari et al., 1995; E. Wang et al., 2004), and neuroimaging work has found that they upregulate neural markers of reward processing (Glazer et al., 2020), suggesting that they may increase the efficiency of learning reward contingencies in the environment. Since reinforcement learning is a critical mechanism for cultural acquisition (Kitayama et al., 2016), it stands to reason that the 7/2-R may enhance one's ability to learn cultural rules and norm, and individuals carrying the 7/2-R may be more likely to exhibit culturally typical patterns of psychological processes.

Indeed, it has been found that the well-documented cultural difference in social orientation (East Asians being more interdependent and European Americans being more independent) is very pronounced among those who carry the 7/2-R of *DRD4*, but it becomes negligible among those who carry neither (Kitayama et al. 2014). This pattern extends to cultural difference in emotional experience as well. Prior work shows that European Americans prefer to

experience positive emotions in lieu of negative emotions, whereas East Asians prefer the harmonious balance between positive and negative emotions (Kitayama et al., 2000). A recent study finds that this pattern was observed among carriers of the 7/2-R, but not among non-carriers (Tompson et al. 2018). Given the emerging evidence, I thus hypothesize that *DRD4* status may moderate the cultural differences in GM volume of cortical regions noted above, such that cultural differences are enlarged among carriers of the 7/2-R.

The Present Study

In the present study, I first aim to replicate previous findings showing that European Americans have greater GM volume in prefrontal regions such as the OFC, and East Asians have greater GM volume in TPJ. I also test whether GM volume in these regions is associated with independent and interdependent self-construal. More importantly, I test whether cultural differences in GM volume are moderated by *DRD4*. I predict that the potential cultural differences would be significantly more pronounced among carriers of the 7/2-R compared to non-carriers.

Method

Participants

One hundred and thirty-two healthy right-handed young adults at the University of Michigan were recruited for the study. Sixty-six of them (45 females and 21 males) were European Americans born and raised in the United States. The average age was 20.2 years, with a range of 18 and 23 years. The remaining 66 (40 females and 26 males) were East-Asian born Asians. At the time of testing, they had been in the United States for less than 10 years[2]. The

[2] The East Asian participants had been in the United States for different amounts of time. In a supplementary analysis presented in the discussion section, I test whether the GM volume of the brain regions examined varies as a function of time they had been in the United States.

average age was 21.2 years, with a range of 18 and 27 years. All the participants were from a larger participant pool (N = 635) dedicated to cultural psychology and genetic research. Participants were selected for the present study based on their *DRD4* genotype. They were recruited in a way such that approximately half of the participants were carriers of the 7-R or 2-R allele of *DRD4*, and the other half did not carry these two alleles. Six participants (3 from each cultural group) were excluded from further structural brain image analysis due to poor quality of their brain scan. The current study was approved by the Internal Review Board of the University of Michigan, and all the participants provided informed consent and were paid for their participation.

Genotyping

The details of the genotyping procedure have been described in Kitayama et al. (2014). Genomic DNA was extracted from saliva samples collected from the participants. The DNA was extracted using a high-capacity membrane-based column (QuickGene810, AutoGen, Inc., Holliston, MA) and was quantitated using an A260/A280 ratio with a NanoDrop spectrophotometer (ThermoScientific, Inc., Wilmington, DE) and agarose gel electrophoresis. The *DRD4* VNTR polymorphism was amplified with *DRD4* forward primer 5'-GCGACTACGTGGTCTACTCG and *DRD4* reverse primer 5'-AGGACCCTCATGGCCTTG (Lichter et al. 1993), using the Roche GC-Rich PCR System amplification buffer (Roche Applied Science, Inc., Mannheim, Germany). The samples were heated in a Stratagene thermocycler (Life Technologies, Inc., Grand Island, NY) at 95°C for 3 min, then cycled 40 times at 95°C for 20 s, 57°C for 20 s, and 72°C for 1 min, followed by 72°C for 3 min. Polymerase chain reaction products were separated and visualized on a 2% agarose gel (type 1-A, Sigma, St. Louis, MO) stained with ethidium bromide.

Among the 126 participants that were included in the structural imaging analysis, frequencies of the *DRD4* alleles were: for European American participants, 10.3% 2R, 4.0% 3R, 65.9% 4R, 0.8% 5R, 16.7% 7R, and 2.4% 8R; for East Asian participants, 22.2% 2R, 0.8% 3R, 76.2% 4R, and 0.8% 5R. As in previous work (Reist et al. 2007; Sasaki et al. 2013; Kitayama et al. 2014), carriers of 7R and 2R alleles (32 European Americans and 26 East Asians)[3] were compared with non-carriers of these alleles (mostly 4R/4R, together with more infrequent variants including the 3R, 5R, and 8R alleles; 31 European Americans and 37 East Asians).

Questionnaire: Self-Construal Scale

The Singelis self-construal (SC) Scale (Singelis, 1994) was used to assess SC. Fifteen items in the scale measured independent SC. Sample items included "I do my own thing, regardless of what others think" and "My personal identity, independent of others, is very important to me". The remaining 15 items measured interdependent SC. Sample items included "I often have the feeling that my relationships with others are more important than my own accomplishments" and "I should take into consideration my parents' advice when making education/career plans." Participants rated each item on a 7-point Likert scale (1 – strongly disagree, 7 – strongly agree). Internal consistency of independent and interdependent SC was adequate for both American participants ($\alpha = 0.65$ and $\alpha = 0.62$, respectively) and Asian participants ($\alpha = 0.68$ and $\alpha = 0.72$, respectively). Participants completed the SC scale approximately two weeks before they underwent the scanning session.

Image Acquisition

[3] An individual is categorized as carrier if either one of the two alleles of *DRD4* is 2R or 7R. Hence, if someone's genotype is 7R/4R, for instance, this person is a carrier. This explains why the number of carriers appears to be greater than the allelic frequency of 2R or 7R mentioned above.

Scanning was performed using a Phillips 3 Tesla MRI scanner (Phillips Medical Systems, Andover, MA). A high-resolution T1-weighted structural image was acquired from all participants (echo time = 4.6 ms, repetition time = 9.8 ms, 256 × 200 matrix, flip angle = eight degrees, field of view = 256 × 256 × 180 (mm), 180 contiguous 1mm sagittal slices per volume).

Image Processing and Analysis

Pre-processing and measurement. Structural brain images of the present study were processed and analyzed using voxel-based morphometry (VBM) (Ashburner & Friston, 2000), implemented in Statistical Parametric Mapping (SPM) software (SPM8; Wellcome Department of Cognitive Neurology, London, UK). VBM has been commonly used in studies examining the relationship between culture or culturally-related psychological variables and the structural aspects of the brain (Kitayama et al., 2017; F. Wang et al., 2017). Each participant's structural image was first examined for its orientation and origin point, and it was adjusted if necessary to better match the template. The images were then segmented into different tissue classes including GM, white matter (WM), and cerebrospinal fluid (CSF) by using prior probability templates. Next, a study-specific template of GM was created using the "Diffeomorphic Anatomical Registration through Exponentiated Lie" (DARTEL) algorithm (Ashburner, 2007), which was then affine-registered to Montreal Neurological Institute (MNI) space. All segmented GM images of the participants were then nonlinearly warped to match the space of the DARTEL template, and a modulation step was performed as well by multiplying the warped tissue probability maps by the Jacobian determinant of the warp to preserve GM volume for later analysis. Finally, the modulated images were smoothed with a 10-mm full-width half-maximum Gaussian kernel. The global volume of GM, WM, and CSF for all participants was calculated by

multiplying the total number of voxels of each tissue type by the voxel size. Total intracranial volume (TIV) was then calculated by summing the global volume of GM, WM and CSF.

Region of interest definition. Given our interest in the OFC and TPJ in the present study, I created an a priori anatomical regions of interest (ROIs) using the WFU PickAtlas toolbox (Maldjian et al., 2003) to bound our analysis (Fig. 2.1). For OFC, I used bilateral middle OFC defined by Automated Anatomical Labeling atlas, following prior work (Kitayama et al., 2017). For TPJ, because it is functionally defined, rather than anatomically defined, I took the two peak voxel coordinates of bilateral TPJ identified in Saxe and Kanwisher (2003), a foundational study on TPJ and perspective taking. I created a 10-mm sphere around the peak coordinates in MNI space.

Statistical analysis. I carried out voxel-level analysis in the framework of general linear model with the pre-processed GM images to test the predictions. All voxels with a GM value of < 0.2 were excluded to retain only the relatively homogenous voxels, given the potential edge effect at the border of GM and WM. Nonstationary cluster extent correction was also applied to correct for nonisotropic smoothness of VBM data (Hayasaka & Nichols, 2004). To test for the predicted cultural difference as well as the culture x *DRD4* interaction on the GM volume, I used a full factorial design. For OFC, because it was predicted that European Americans would show greater GM volume, the two following contrasts were calculated: (a) (American carriers group + American non-carriers group) - (Asian carriers group + Asian non-carriers group) for cultural difference, and (b) (American carriers group > Asian carriers group) - (American non-carriers group > Asian non-carriers group) for culture x *DRD4* status interaction. For TPJ, because it was predicted that East Asians would show greater GM volume, the two following contrasts were calculated: (a) (Asian carriers group + Asian non-carriers group) - (American carriers group +

American non-carriers group) for cultural difference, and (b) (Asian carriers group > American carriers group) - (Asian non-carriers group > American non-carriers group) for culture x *DRD4* status interaction. The threshold of significance was set at $p < .05$ (FWE-corrected at the voxel level), after small-volume correction using the ROIs created as noted above. We also carried out additional exploratory whole-brain analysis to confirm our predictions, and the threshold of significance was set at $p < .05$ (FWE-corrected at the voxel-level), unless notified otherwise. Lastly, I tested the correlation between GM volume and the SC. I used a multiple regression design and included independent SC or interdependent SC as regressor. The threshold of significance was set at $p < .05$ (FWE-corrected at the voxel level), after small-volume correction. Age, sex, and TIV were included as covariates in all the analyses.

Results

Demographics and Questionnaire Data

As expected, East Asians were more interdependent in their SC than European Americans ($t(124) = -2.565$, $p = .012$); however, the two groups did not differ on the independent SC ($t(124) = 0.380$, $p = .705$). Within each cultural group, East Asians were more interdependent than independent in their SC ($t(62) = 2.490$, $p = .015$), whereas European Americans showed comparable level of interdependent and independent SC ($t(62) = -.160$, $p = .873$). See Table 2.1 for demographics, self-construal mean scores, and *DRD4* genotype information for both East Asians and European Americans.

ROI Analysis

First, there was a significant cultural difference in the GM volume in the OFC. After controlling for age, sex, and TIV, European Americans showed greater GM volume in two clusters at bilateral OFC than East Asians (Cluster 1: peak voxel MNI coordinates -46, 50, -2; *Z*

value > 10; cluster size = 137; peak-level $p < .001$, FWE-corrected; Cluster 2: peak voxel MNI coordinates 48, 50, -2; *Z* value > 10; cluster size = 193; peak-level $p < .001$, FWE-corrected) after the small volume correction.

In addition, there was a significant Culture x *DRD4* status interaction on GM volume in the right OFC after controlling for the same set of covariates and after the small volume correction (peak voxel MNI coordinates 27, 60, -9; *Z* value = 3.21; cluster size = 19; peak-level $p = .033$, FWE-corrected). In particular, among carriers of the 7/2-R allele of *DRD4*, European Americans showed greater GM volume at the right OFC than East Asians. This cultural difference was substantially diminished among non-carriers (Fig. 2.2).

Second, there was also a significant cultural difference in the GM volume in the TPJ. After controlling for age, sex, and TIV, there were two clusters at the right TPJ that showed greater GM volume for East Asians than for European Americans (Cluster 1: peak voxel MNI coordinates 58, -46, 28; *Z* value = 4.09; cluster size = 35; peak-level $p = .002$, FWE-corrected; Cluster 2: peak voxel MNI coordinates 52, -43, 28; *Z* value = 3.61; cluster size = 2; peak-level $p = .009$, FWE-corrected) after the small volume correction.

In addition, there was a significant Culture x *DRD4* status interaction on GM volume in the right TPJ after controlling for the same set of covariates and after the small volume correction (peak voxel MNI coordinates 52, -46, 22; *Z* value = 3.73; cluster size = 56; peak-level $p = .006$, FWE-corrected). In particular, among carriers of the 7/2-R allele of *DRD4*, East Asians showed greater GM volume at the right TPJ than European Americans. This cultural difference was absent among non-carriers (Fig. 2.3).

Whole Brain Analysis

The findings from the ROI analysis above were also evident from the whole brain analysis. First, a large area of the prefrontal cortex was greater in GM volume for European Americans than for East Asians (Table 2.2 and Fig. 2.4). This area was centered at the right anterior PFC and extended to both the bilateral OFC and the medial prefrontal cortex. In addition, the left visual association area showed a similar cultural difference. This cultural difference (Americans > Asians) did not yield significant Culture x *DRD4* interaction with the most stringent threshold of significance ($p < .05$ FWE-correction). However, with a more liberal voxel-level threshold of $p < .001$ (uncorrected), a few voxels centered at right anterior OFC and left anterior cingulate cortex emerged as significant (see Fig. 2.5).

Second, a large area centering at the middle temporal gyrus bilaterally showed significant greater GM volume among East Asians than among European Americans (see Table 2.3 and Fig. 2.4). The bilateral supramarginal gyrus (where the TPJ rests on) and the somatosensory cortex showed a similar pattern. This cultural difference (Asians > Americans) did not yield a significant Culture x *DRD4* interaction with the most stringent threshold of significance ($p < .05$ FWE-correction). However, at a more liberal threshold of $p < .001$ (uncorrected), a few voxels near the bilateral TPJ, the right superior parietal lobe, and the right temporal pole showed a significant interaction between culture and *DRD4* (see Fig. 2.6).

Correlation with Self-Construal

First, there was a significant correlation between the GM volume of the right OFC and interdependent SC. After controlling for age, sex, and TIV, the OFC GM volume was negatively correlated with interdependent SC (Peak voxel MNI coordinates 40, 59, -6; Z value = 3.37; cluster size = 115; peak-level $p = .039$, FWE-corrected) after small volume correction using the bilateral OFC ROI (Fig. 2.7-A). Fig. 2.7-B shows the scatterplot. The negative relationship can

be found among both European Americans and East Asians. There was no significant correlation between independent SC and the OFC GM volume.

Second, there was a significant correlation between the GM volume of the right TPJ and independent SC. After controlling for age, sex, and TIV, the TPJ GM volume was negatively correlated with independent SC (Peak voxel MNI coordinates 52, -48, 33; *Z* value = 3.85; cluster size = 120; peak-level $p = .004$, FWE-corrected) after small volume correction using the bilateral TPJ ROI (Fig. 2.8-A). Fig. 2.8-B shows the scatterplot. The negative relationship can be found among both European Americans and East Asians. There was no significant correlation between interdependent SC and the TPJ GM volume.

Discussion

The central contribution of the present work is to show that *DRD4* genotype modulates cultural differences in the GM volume of OFC and TPJ. I first replicated earlier findings and showed that the GM volume in OFC was greater among European Americans, and the GM volume in TPJ was greater among East Asians. Moreover, these cultural differences were more pronounced for carriers of the 7/2-R than for non-carriers. Because 7/2-R has been shown to promote the effectiveness of reinforcement-based learning (including the learning of culture), the current result provides initial evidence that cultural difference in cortical GM volume is likely due to cultural experience in carrying out culturally scripted behaviors.

The present work supports recent theorizing that when certain psychological processes, such as personal goal pursuit or perspective taking are positively reinforced by cultural values and norms, the neural circuitries in relevant brain regions may also be consolidated and strengthened (Kitayama & Salvador, 2017). These changes are reflected macroscopically as an increase in GM thickness, gyrification, and volume of the regions. It is plausible that these

structural changes reinforced through cultural reward contingencies are mediated or stabilized by epigenetic pathways that are gradually established through socialization and enculturation (Meaney, 2001). In this way, culture is thought to be "embrained".

Another finding consistent with this proposition comes from the East Asian participants of the current sample. The East Asian participants had all been born in Asia, had subsequently moved to the U.S., and had stayed there for a duration ranging from a few months to a bit less than 10 years. Hence, it is possible to test whether their brain structures may show certain changes due to the exposure to the new cultural values or norms. Indeed, I observed that the GM volume of OFC was positively correlated with the duration they had been in the U.S., but only among carriers of the 7/2-R, those who supposedly are more sensitive to cultural influences. This finding adds further evidence that cultural experience can shape the structural properties of the brain. In this case, immigrating to a more independent culture where personal preference and personal goal pursuit is more positively sanctioned may increase the engagement of these psychological processes among East Asians (especially carriers of the 7/2-R), thus recruiting the OFC more extensively. Caution is needed in interpreting this finding, however, since the data were still cross-sectional in nature. Future work using longitudinal analysis to examine how acculturation shapes the brain structures will be extremely informative.

In the current data, cultural difference in GM volume is either largely diminished or completely absent among non-carriers of 7/2-R of *DRD4*, in line with prior work showing that cultural difference in independent and interdependent social orientation is negligible among non-carriers (Kitayama et al., 2014). One may wonder what this pattern implies for the larger literature on cultural differences in psychological and neural processes, given that almost half of European Americans (and East Asians) are indeed non-carriers of 7/2-R. It is worth noting that

DRD4 is only one among the many genes that have been shown to modulate cultural differences in behaviors (H. S. Kim & Sasaki, 2014). Moreover, only a small number of outcome variables, such as explicit belief-based measure of social orientation and GM volume in the brain, have been examined in relation to the "plasticity alleles" so far. It is possible that non-carriers of 7/2-R of *DRD4* might still show culturally normative psychological tendencies and neural patterns when assessed with different outcome variables, and a different set of "plasticity alleles" may moderate these cultural differences.

It is noteworthy that the present study replicated the inverse association between interdependent SC and GM volume of OFC (Kitayama et al. 2017). Moreover, the present study also found that independent SC was inversely associated with the GM volume of TPJ. It is interesting that we did not find the positive correlation between GM volume of a brain region (e.g., OFC) and the self-construal congruent with this brain region (e.g., independence). Rather, the negative correlation between the brain region and the psychological process incongruent with it was identified. It might be the case that psychological tendencies of, say, personal goal pursuit is promoted by inhibition of competing psychological tendencies of interdependence. Conversely, perspective taking may be promoted by inhibition of competing tendencies of independence. Future work should zero in on how the self-construal and the associated psychological processes promote or inhibit the functioning of different brain regions, and what consequences it may lead to on the structure of these regions.

The effects observed in the present study are mostly lateralized to the right hemisphere of the brain. In the case of TPJ, this finding is not unexpected because prior work suggests that the right TPJ tends to be more important for perspective taking. For example, one transcranial magnetic stimulation study finds that disruption of the right (rather than left) TPJ selectively

impairs perspective taking (Young, Camprodon, Hauser, Pascual-Leone, & Saxe, 2010). This is consistent with the finding of a recent meta-analysis (Krall et al., 2015). The difference in the role of left versus right OFC, however, is less known (Rolls & Grabenhorst, 2008). Future work should replicate the lateralized finding and elucidate whether such pattern has any implications for the role of left versus right OFC and TPJ.

There were some brain regions other than the OFC and TPJ in which the GM volume also showed significant cultural difference. One notable region where European Americans had greater GM volume than East Asians was the visual association area. This region maps onto the lateral occipital cortex (LOC), which cognitive neuroscientists has shown to be critical for object processing (Grill-Spector et al., 2001). This pattern is consistent with the finding in cultural psychology literature that European Americans tend to be more analytic in their cognitive style and allocate greater attention on the central object in a visual scene during visual processing (Masuda & Nisbett, 2001; Nisbett et al., 2001). In parallel with this pattern, I found that East Asians showed greater GM volume in the retrosplenial cortex, together with the posterior parahippocampal gyrus. These regions are part of the cortical network responsible for processing contextual scene and background (Epstein, 2008). This pattern is also consistent with previous findings that East Asians are more holistic in their cognitive style and are more attentive to the contextual surrounding and the relationship between the focal objects and the context (Goto et al., 2013; Na & Kitayama, 2011; Nisbett et al., 2001).

Some limitations of the current work must be acknowledged. First, the present analysis is limited by the availability to only the structural brain imaging data, but not functional brain imaging data. Hence, while I have argued that cultural differences in the GM volume of the OFC and TPJ are likely due to different cultural tendency to engaging in goal-pursuit behavior or

perspective taking, I did not show that these two regions are indeed activated if the participants were to actually perform these behaviors. Although this limitation is remedied by the fact that I chose a priori ROIs that prior work has shown to be critical for the behaviors under discussion (e.g., personal goal pursuit, perspective taking), future work should simultaneously collect structural and functional data to better elucidate the mechanism of cultural differences in brain structures. Second, the present study tested only one gene, *DRD4*. There are a few other genes which have also been found to modulate environmental influences (Caspi et al., 2003; Set et al., 2014). The choice of focusing on *DRD4* was motivated by prior empirical and theoretical evidence showing that it is particularly important in modulating cultural differences in psychological tendencies and neural processes (Glazer et al., 2020; Kitayama et al., 2014). However, future studies should extend this line of work by testing other plasticity alleles. Third, the present study only tested GM volume. Future work should examine whether culture may also shape the structural connectivity of networks governing various culturally sanctioned behaviors.

Despite these limitations, the present study offers the first evidence that culture may indeed be a critical agent in shaping not only the functioning of the brain, but also the structure features of the brain. Although the data are correlational, and further longitudinal or causal analysis is needed to draw a firm conclusion on this point, the present finding is consistent with the thesis that the biological system is influenced by and embedded within the larger ecological, social, and cultural contexts.

Table 2-1 Descriptive statistics of demographics, self-construal scores, and DRD4 status information

	European Americans	East Asians	Significance
n	63	63	
Mean age (SD)	20.2 (1.61)	21.2 (1.65)	t = -3.498, p = .001
DRD4 (7/2-R vs. other alleles)	32/31	26/37	χ^2 = 1.150, p = .284
Sex (male vs. female)	20/43	24/39	χ^2 = 0.559, p = .455
Self-construal			
Independent: mean (SD)	4.87 (0.56)	4.83 (0.59)	t = 0.380, p = .705
Interdependent: mean (SD)	4.85 (0.53)	5.10 (0.56)	t = -2.565, p = .012

Table 2-2 Regions where European Americans had greater GM volume compared to East Asians (cluster size > 10)

Region (Brodmann's area)	MNI coordinates			Z value	Cluster size	Cluster-level *P* value (FWE-corrected)
	x	y	z			
Right anterior prefrontal cortex (10)	2	68	1	>8	19386	< 0.001
Left visual association area (18)	-15	-84	-17	7.14	13618	< 0.001
Right caudate	10	17	3	4.79	230	0.003
Right thalamus	4	-7	4	5.04	148	0.006
Left visual association area (19)	34	-87	22	4.66	35	0.022
Right somatosensory cortex (5)	3	-42	60	4.64	14	0.032
Left insula	-40	15	-9	4.57	11	0.034

Table 2-3 Regions where East Asians had greater GM volume compared to European Americans (cluster size > 10)

Region (Brodmann's area)	MNI coordinates			Z value	Cluster size	Cluster-level *P* value (FWE-corrected)
	x	y	z			
Right middle temporal gyrus (21)	51	-10	-29	7.10	3179	< 0.001
Left middle temporal gyrus (21)	-56	-9	-29	6.82	1724	< 0.001
Right supramarginal gyrus (40)	68	-25	22	6.45	2277	< 0.001
Left supramarginal gyrus (40)	-63	-30	43	5.40	286	0.002
Right temporal pole (38)	52	20	-26	4.89	49	0.018
Left somatosensory cortex (1)	-66	-19	22	4.56	15	0.031
Right retrosplenial area (30)	20	-40	0	4.60	15	0.031
Right somatosensory cortex (1)	52	-25	49	4.48	14	0.032

Right premotor cortex (6)	16	-7	70	4.48	13	0.033

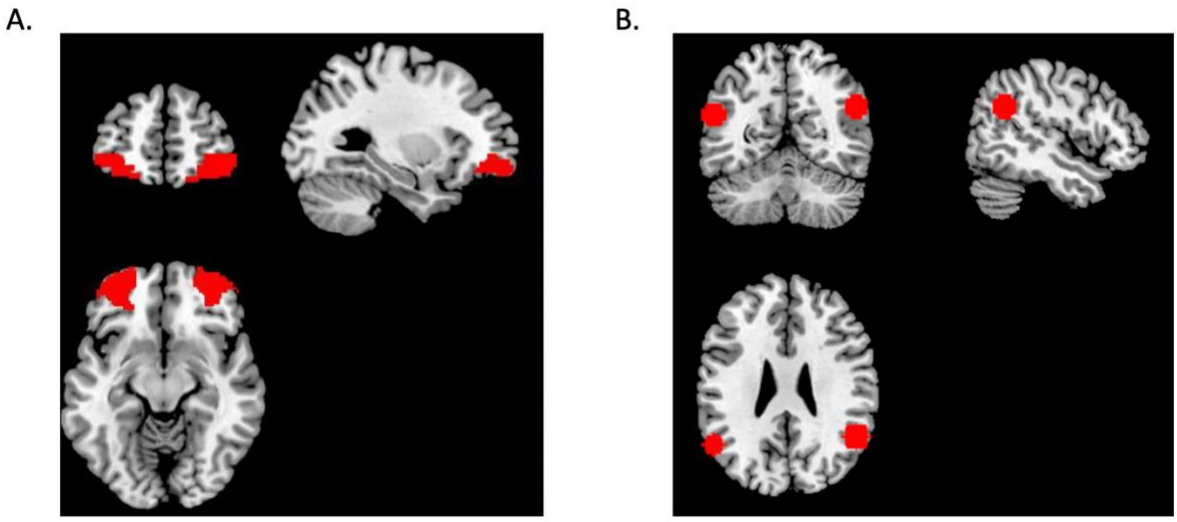

Figure 2-1 The anatomical region of interest (ROI) for the present analysis from a coronal (top left), sagittal (top right), and axial (bottom left) view.

A. The orbitofrontal cortex (OFC) ROI. B. The temporoparietal junction (TPJ) ROI.

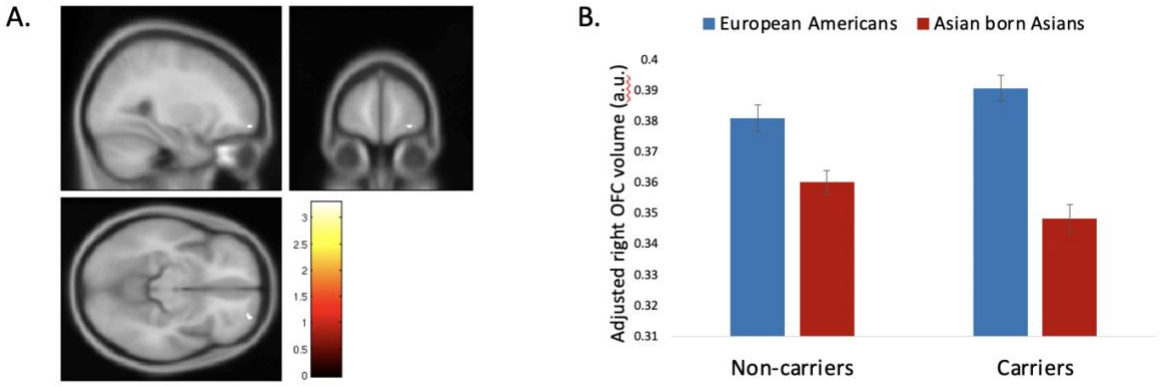

Figure 2-2 ROI analysis on Culture x *DRD4* interaction at OFC using small-volume correction.

A. Cluster that shows significant interaction between culture and *DRD4* status on gray matter (GM) volume within the OFC ROI (thresholded at P value of < 0.05 family-wise error (FWE) corrected). B. Value of the peak voxel as a function of Culture and *DRD4* status. Among carriers, European Americans showed significantly greater right OFC GM volume than East Asians. This effect was significantly diminished among non-carriers.

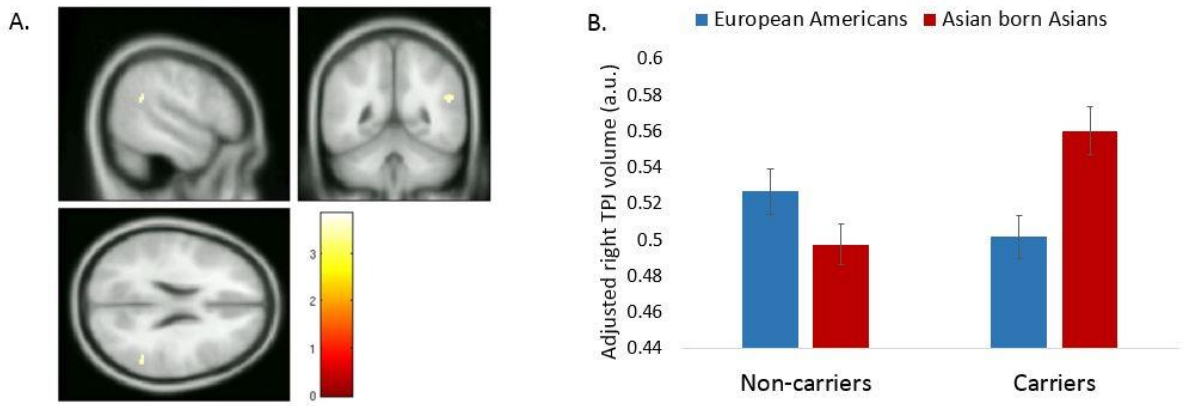

Figure 2-3 ROI analysis on Culture x *DRD4* interaction on TPJ using small-volume correction.

A. Cluster that shows significant interaction between culture and *DRD4* status on GM volume within the TPJ ROI (thresholded at *P* value of < 0.05 family-wise error (FWE) corrected). B. Value of the peak voxel as a function of Culture and *DRD4* status. Among carriers, East Asians showed significantly greater right TPJ GM volume than European Americans. The pattern tended to reverse among non-carriers.

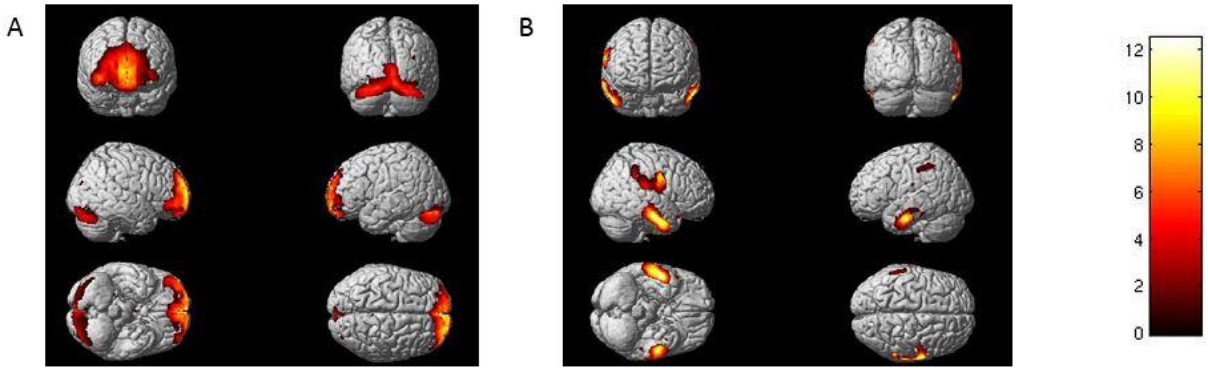

Figure 2-4 Regions where European Americans and East Asians exhibited greater GM volume, shown in SPM render-style brain.

A. Regions where European Americans had significantly greater GM volume than East Asians. B. Regions where East Asians had significantly greater GM volume than European Americans. The color bar shows the corresponding Z-scores. Threshold was set at T = 4.56 (corresponding to a *P* value of < 0.05 FWE-corrected).

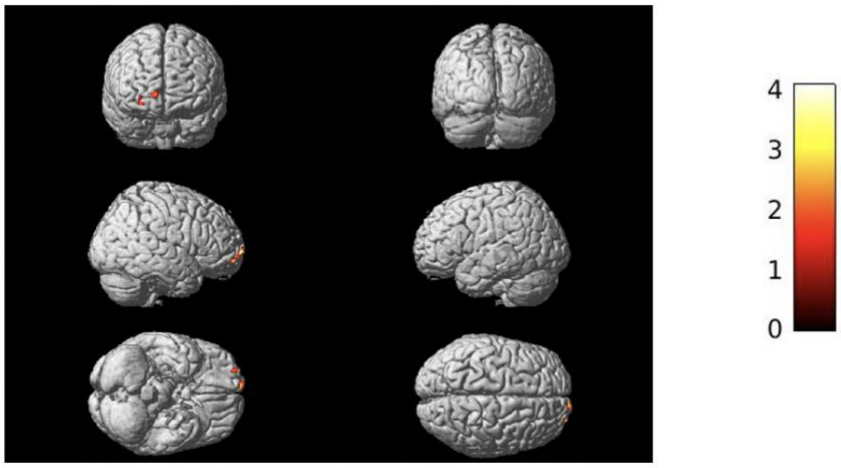

Figure 2-5 Regions in which the GM volume showed Culture x *DRD4* interaction, such that cultural difference (European Americans > East Asians) was more pronounced among carriers than non-carriers.

The color bar shows the corresponding Z-scores. Threshold was set at 3.16 for display purpose (corresponding to a P value of < 0.001 uncorrected).

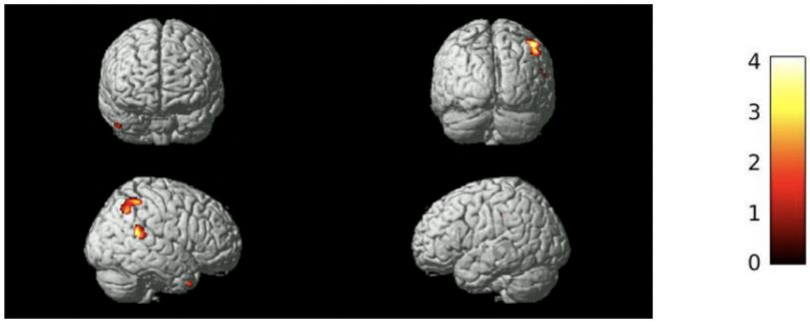

Figure 2-6 Regions in which the GM volume showed Culture x *DRD4* interaction, such that cultural difference (East Asians > European Americans) was more pronounced among carriers than non-carriers.

The color bar shows the corresponding Z-scores. Threshold was set at 3.16 for display purpose (corresponding to a P value of < 0.001 uncorrected).

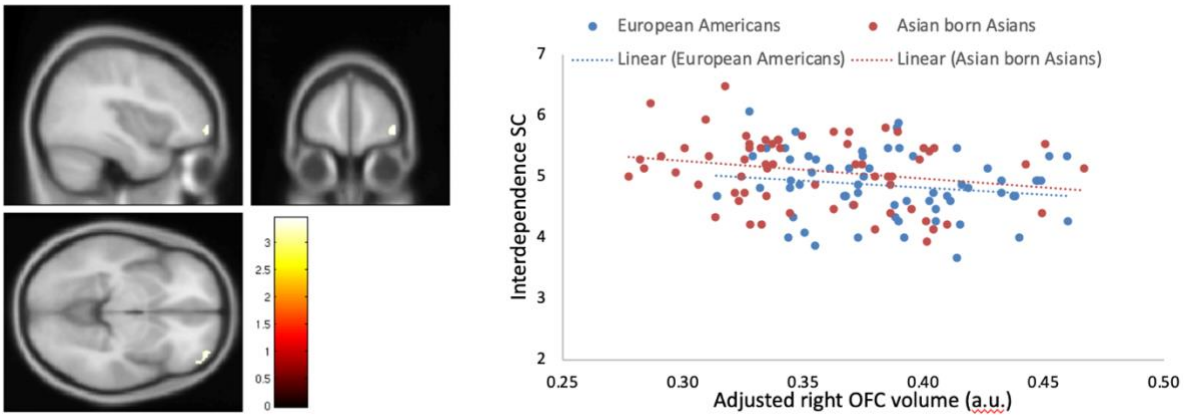

Figure 2-7 ROI analysis on the correlation between OFC volume and interdependence self-construal (SC) using small-volume correction.

A. Cluster that shows significant negative correlations between interdependent SC and GM volume within the OFC ROI (thresholded at *P* value of < 0.05 family-wise error (FWE) corrected). B. Scatterplot on right OFC GM volume (peak voxel) and interdependent SC as a function of culture.

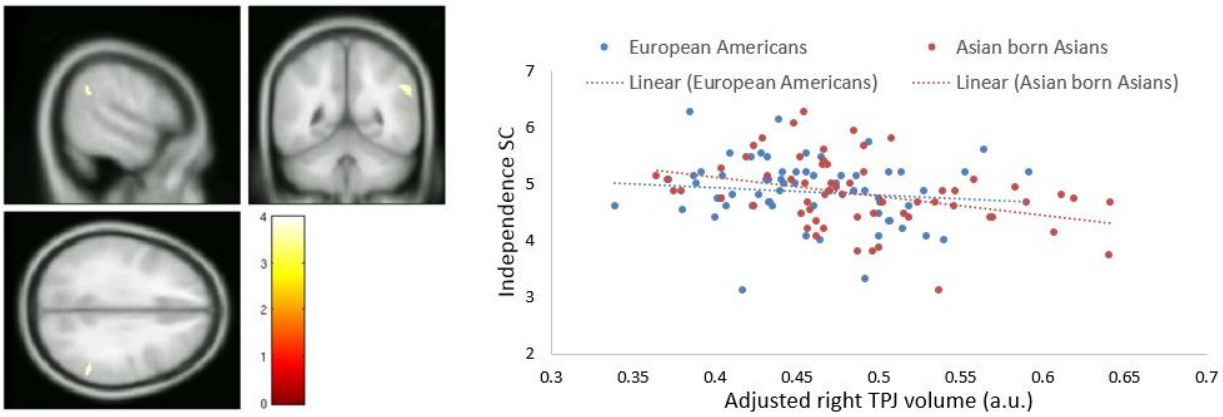

Figure 2-8 ROI analysis on the correlation between TPJ volume and independence SC using small-volume correction.

A. Cluster that shows significant negative correlations between independent SC and GM volume within the TPJ ROI (thresholded at *P* value of < 0.05 family-wise error (FWE) corrected). B. Scatterplot on right TPJ GM volume (peak voxel) and independent SC as a function of culture.

Chapter 3 Systemic Racism and Biological Health

In Chapter 2, I showed that culture can shape the structure of the human brain. While culture is a critical component of the sociocultural context, most current conceptualization of culture does not capture the aspect of the sociocultural environment that is characterized by inequality (in terms of resource, power, and status), which also has profound influences on one's biological system. In the context of the U.S., another socially constructed variable, race, is highly relevant. Many marginalized racial or ethnic groups in the U.S. are deprived of various socioeconomic resources and relegated to lower social status, as a result of the system of racism (Pew Research Center, 2011, 2016a). This lack of wealth, resources, power, and status among marginalized groups has far-reaching consequences, one of which is the wide racial health disparity that still persists in American society (C. A. Collins & Williams, 1999a; Williams & Mohammed, 2009). In this chapter, I zoom in to the racial disparity in health and investigate how systemic racism impacts health through two studies. In Study 1, I focus on two important social structural factors underlying systemic racism, racial residential segregation and income disparity. I examine how these factors contribute to disproportionate growth of cases and deaths in large U.S. cities during the COVID-19 pandemic. In Study 2, I focus on individual's perception and response to racial discrimination, and I test how this psychological process prospectively predicts all-cause mortality during a 15-year period.

Study 1[4]

It has become increasingly clear that the rate of fatalities during the current COVID-19 pandemic is exceptionally high for racial and ethnic minorities in the U.S., especially Black and Hispanic Americans[5]. For instance, Blacks and Hispanics are almost three times more likely to be infected by the coronavirus than Whites (Centers for Disease Control and Prevention, 2020). The disproportionate suffering of minority groups is likely due to many social structural factors. These factors include unequal distribution of wealth and other societal resources, such as adequate housing and access to health care and other social services (Diez-Roux et al., 2000; Massey, 1996; Wilson, 2012). Further, racial biases reportedly exist in medical treatment at hospitals and clinics[6]. Many of these factors implicate both systemic racism and social class disparities, and therefore, they highlight the structural inequality in many metropolitan areas of the U.S.

Here, I focused on two central aspects of this inequality, one pertaining primarily to systemic racism (racial residential segregation) and the other related primarily to social class disparity (income inequality). These two factors are inherently related. For example, when racial and ethnic minorities are residentially segregated, income inequality often results in the area (Williams & Collins, 2001). Nevertheless, greater conceptual clarity may be achieved by combining a spatial variable (racial residential segregation) with a non-spatial, economic variable (income inequality). In combination, these two structural factors may illustrate both the reality of deepening poverty in the U.S. and its lethal effects during the pandemic.

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[4] Study 1 of Chapter 3 is based on Yu et al. (2021) published in *Annals of the New York Academy of Sciences*.
[5] To be most consistent with the U.S. census categories and the existing demography literature (e.g., Frey & Myers, 2005), we use White, Black, Hispanic, and Asian Americans, to refer to people of European, African, Latin American, and Asian descent, respectively.
[6] https://www.nytimes.com/2020/05/10/us/coronavirus-african-americans-bias.html

In the current work, I explored the hypothesis that metropolitan areas become more vulnerable to COVID-19 if wealth is unevenly distributed in each area, and as a consequence, poverty is concentrated in certain communities. When poverty is concentrated, it results in a deprivation of many social, medical, and community-related resources in the segregated communities (Intrator et al., 2016; Massey & Fischer, 2000). These communities may not have adequate access to medical and social services. They may also suffer from congested housing and compromised hygienic conditions (Gaskin et al., 2012; Massey, 1996). Moreover, healthy foods may be harder to come by (Goodman et al., 2018; Powell et al., 2007; Zenk et al., 2005). Over time, residents in these communities may develop medical conditions that compromise immunity, including obesity, diabetes, and cardiovascular problems (Kershaw & Pender, 2016; Simons et al., 2018; Usher et al., 2018). In addition, the residents may be more likely to work in essential service jobs. It is therefore not an option for them to work at home. They are thus exposed to the virus while working (U.S. Bureau of Labor Statistics, 2019). They may also have difficulty accessing adequate COVID-19 testing and medical treatment. All of these conditions would make the residents more vulnerable to infectious diseases such as COVID-19.

Poverty may become concentrated through two processes. The first process involves racial residential segregation (called "segregation" hereafter). Segregation refers to the extent to which households of two racial or ethnic groups, typically, the White majority and a minority group (e.g., Black, Hispanic, or Asian) are clustered into racial enclaves above and beyond the level expected by chance alone (O. D. Duncan & Duncan, 1955; Massey & Denton, 1988, 1989). Metropolitan areas high in segregation thus contain more and larger enclaves of both Whites and the minorities at issue. A large body of literature in sociology and demography (Massey, 1990; Massey & Denton, 1993; Massey & Eggers, 1990; Massey & Fischer, 2000; Quillian, 2012)

shows that when Blacks are segregated, poverty is concentrated in the segregated Black enclaves, reflecting a massive wealth disparity (more than seven-fold) between Whites and Blacks (Pew Research Center, 2011). In their classical contribution to this literature, "American Apartheid: Segregation and the making of the underclass," Massey and Denton (1993) observed, "Because of racial segregation, a significant share of black America is condemned to experience a social environment where poverty and joblessness are the norm (Page 2)." The economic consequence of segregation may also apply to Hispanics because of an equally stark wealth disparity between Whites and Hispanics. The first prediction of the present study then, is that the spread of COVID-19 should be faster in metropolitan areas that segregate the two minority groups (Blacks and Hispanics).

The second process by which poverty is concentrated in certain segments of metropolitan areas involves income inequality. When income inequality of a given area is high, it can affect all racial groups in the area. First, this factor may exacerbate the poverty of the segregated enclaves of the minority groups since these groups are also disadvantaged in income, relative to Whites (Pew Research Center, 2016b). Second, it may also lend itself to White enclaves that are as poor (Case & Deaton, 2015). Although residential segregation based on income is lower among Whites than in Blacks (Bischoff & Reardon, 2013; Coulton et al., 1996; Reardon & Bischoff, 2011), there may still exist poor White enclaves due to the dramatic loss of economic standings among Whites without college degrees over the last few decades (Case & Deaton, 2021; Murray, 2012). Hence, the poor White enclaves together with the poor minorities' enclaves, because of the synergic effect of segregation and income inequality, will form larger areas suffering from poverty and the relative deprivation of social, medical, and community-related resources.

Numerous prior studies investigated segregation and income inequality as correlates of health outcomes. A growing body of research shows that segregation is linked to chronic illnesses (e.g., hypertension, diabetes, and systemic inflammation) and greater mortality, particularly among racial minorities (C. A. Collins & Williams, 1999b; Kershaw & Pender, 2016; Simons et al., 2018; Usher et al., 2018). Likewise, the evidence shows that the unequal distribution of income is associated with poor health outcomes, such as obesity and cardiovascular diseases (Diez-Roux et al., 2000; Pickett et al., 2005). It has also been linked to reduced well-being and higher all-cause mortality (Kawachi et al., 1997; Oishi et al., 2011).

However, one important shortcoming of the current literature is that it largely ignores the two factors' possible joint, or interactive effects. One important exception comes from Nuru-Jeter and LaVeist (2011), who showed that Black-White segregation attenuates the effect of income inequality in predicting greater all-cause mortality among Blacks. They interpreted the pattern as reflecting higher social cohesion in segregated Black communities, which may serve as a protective factor against economic disparity (see Geronimus et al., 2015 for a similar argument). However, it is unclear whether such a protective effect extends to COVID-19 related outcomes. Indeed, social cohesion could conceivably contribute to the spread of infectious disease by increasing social contact with a wider range of individuals in the community (Salvador et al., 2020). More generally, as argued above, when metropolitan areas are both racially segregated and high in income inequality, more and larger enclaves will be plagued with poverty. These enclaves will be especially vulnerable to the disease. I thus tested whether the combination of the two facets of structural inequality (i.e., segregation and income inequality) would exacerbate the negative impact of COVID-19.

The 100 largest American metropolitan statistical areas (MSAs) (Frey & Myers, 2005) were examined during the pandemic. MSA refers to a single contiguous geographic region consisting of a city (or cities) and surrounding communities that are connected by social and economic factors (U.S. Census Bureau, 2020). This area typically encompasses multiple counties. The analysis unit of the current study was each of the 577 counties subsumed under the 100 largest MSAs. All measures, including a measure of income inequality (Gini), daily counts of COVID-19 cases or deaths, as well as all control variables, were assessed at the county level, except for segregation, which was assessed at the MSA level. Segregation typically occurs across city and county boundaries within a larger MSA, which would make counties or cities too granular to characterize the dispersal of different racial and ethnic groups within a single interconnected region for social and economic activity (U.S. Census Bureau, 2002).

A critical challenge in cross-area comparisons, including the current one, stems from the fact that the counties can vary on a variety of factors, including those directly influencing the reported numbers of cases and deaths. Counties may vary in the availability of COVID-19 diagnostic tests, as well as the diagnostic criteria in classifying symptoms and deaths as COVID-19-related or not. To address these potential biases, I followed our earlier work (Berg et al., 2020; Salvador et al., 2020) and tested the *growth rate* of both confirmed COVID-19 cases and deaths in the first 30 days of county-wise COVID-19 outbreak in our main analysis, which was supplemented by a robustness check testing an even shorter period of 15 days. The confounding variables are unlikely to vary systematically within such short periods and thus unlikely to influence the growth rate of cases and deaths (Berg et al., 2020; Salvador et al., 2020). Population size, population density, median income, percent of population over 65 years of age,

and the proportion of Blacks, Hispanics, and Asians in each county were controlled for in the current analysis.

Altogether, this study examined whether the MSA-level segregation and the county-level income inequality would jointly predict the growth rate of both COVID-19 cases and deaths in the first 30-day period of the county-wise outbreak. I expected that the progression of the disease is faster in counties located in racially segregated MSAs. Second, I also predicted that this effect of segregation should be augmented for counties higher in income inequality. In combination, it is expected that there would be a confluence of the two social structural variables: The spread of COVID-19 would be the fastest when high segregation is combined with high income inequality.

Method

Sample and Data

Daily reports of COVID-19 confirmed cases and deaths were retrieved from a public repository updated daily by the Johns Hopkins University Center for Systems Science and Engineering[7]. The results here are based on data from January 22, 2020 through June 20, 2020, before the second nation-wide outbreak began. The cumulative daily counts of confirmed cases and deaths were available for each of the 577 counties nested under the 100 MSAs we examined. Some MSAs were composed of only one county (for example, Bakersfield, CA, was composed of Kern county), but others included multiple counties (for example, Pittsburgh, PA, includes 7 counties: Allegheny, Fayette, Washington, Westmoreland, Butler, Beaver, and Armstrong). The Office of Management and Budget Bulletin was used to determine which counties belong to each

[7] https://github.com/CSSEGISandData/COVID-19/tree/master/csse_covid_19_data/csse_covid_19_time_series

of the MSAs[8]. Following prior work (Berg et al., 2020; Salvador et al., 2020), the data of the first 30 days of the outbreak of each county was analyzed, with Day 1 defined as the day when at least 20 confirmed cases or at least 1 death were reported in the county for the analyses on cases and deaths, respectively. The exact number of cases used as a cutoff, 20, is arbitrary. Berg et al. (2020) used 100, whereas Salvador et al. (2020) used both 100 and 20. Unlike in these two studies (which compared countries), the current study focused on a cross-county variation. To maximize the number of the counties included in the analysis, the smaller criterion of the two was used for the main analysis, followed by a robustness check that used the 100-case cutoff. In order to ensure that the estimate of the growth rate is robust, counties were excluded from the analysis if less than 15 days of data were available. This resulted in a total of 535 and 495 counties for the analyses of cases and deaths, respectively.

I used a dissimilarity index of segregation. It quantifies segregation as the degree of deviation from a random residential distribution of two social groups in within a given geographic area (O. D. Duncan & Duncan, 1955). This index is available for Black-White segregation, Hispanic-White segregation, and Asian-White segregation based on the 2005-2009 Census data, and it was made available by the Institute for Social Research at the University of Michigan (Frey & Myers, 2005)[9]. The index takes values from 0 to 100. It reflects the percentage of one group that would have to be relocated to attain the same spatial dispersion as the second group. Values of 60 or above are considered to show "high" degree of segregation (Williams & Collins, 2001). On average, the Black-White segregation was higher (M = 58, SD = 10.1) than either the Hispanic-White segregation (M = 46, SD = 8.2) or the Asian-White segregation (M =

[8] https://www.whitehouse.gov/omb/information-for-agencies/bulletins/
[9] https://www.psc.isr.umich.edu/dis/census/segregation.html

45, *SD* = 6.7) across the 100 MSAs, *p*s < .001. There was no significant difference between the latter two indices of segregation, $p = .313$.

To quantify income inequality, the Gini coefficient was obtained for each county. The Gini coefficient is a measure of income inequality based on dispersion of household income across the entire income distribution within a given geographic area. The Gini coefficient was estimated from the American Community Survey, a large-scale survey conducted by the U.S. Census Bureau[10].

Several covariates were included in the analysis. First, the proportions of Blacks, Hispanics, and Asians in each county (called Black, Hispanic, and Asian shares, respectively) were included. These minority share variables help us assess whether the growth of the number of cases and deaths might depend on the proportion of each minority group in the area. In addition, population size and population density were included since both of these variables could increase the speed of the spread of the virus. Proportion of elderly adults (over 65 years old) was included because mortality could go up as a function of age. Median household income was also controlled for to adjust for the overall economic status of each county. These data were also taken from the U.S. Census Bureau Website[11]. The correlations among the covariates at the county-level and MSA-level are included in Appendix A.

In addition, different states instituted lockdowns at different times after the outbreak. Hence, in a set of supplementary analyses, I additionally controlled for the number of days

[10]https://data.census.gov/cedsci/table?q=B19083%3A%20GINI%20INDEX%20OF%20INCOME%20INEQUALITY&g=0100000US.04000.001,.050000&hidePreview=true&tid=ACSDT5Y2018.B19083&moe=false

[11] https://www.census.gov/

during the 30-day period that were after the state-wide lockdown for each county. Dates for state-imposed stay at home orders were obtained from the Wall Street Journal[12].

Statistical Analysis

A three-level linear mixed model analysis was implemented with the lme4 package in R (Bates et al., 2015) for analyses on both confirmed cases and deaths. Infectious disease trajectories are approximately exponential in their initial phases (Anderson, 1982). Thus, the number of both confirmed cases and deaths were natural-log-transformed first and then subjected to linear mixed models with restricted maximum likelihood estimation. At level-1, the natural log of the cumulative number of either cases or deaths of the counties on each day was regressed on day (varying from 1 through 30), which was first centered. The main effect of day is necessarily positive and shows the rate of growth of the cases or deaths. Counties are nested under relevant MSAs. The resulting level of county constitutes the level-2. There are several predictors at level-2, i.e., Gini, population size, population density, median income, percentage of older adults, and Black, Hispanic, and Asian share. Finally, at level-3, the MSAs varied in the degree of segregation. Three measures of segregation were tested in separate analyses, i.e., Black-White segregation, Hispanic-White segregation, and Asian-White segregation.

I analyzed whether the growth rate of cases or deaths across the days in each county (the effect of day) would vary in magnitude as a function of the segregation of the MSA (the day x segregation interaction), and the multiplicative effect of income inequality and segregation (the day x Gini x segregation interaction). The hypothesis that the growth was particularly fast when high level of income inequality is combined with high level of segregation would be supported if this 3-way interaction proved significant. Further, I tested whether the growth rate of cases or

[12] https://www.wsj.com/articles/a-state-by-state-guide-to-coronavirus-lockdowns-11584749351

deaths varied as a function of the Black share, Hispanic share, and Asian share. This analysis sheds light on whether certain minority groups were impacted disproportionately, as suggested by public health data. This analysis enabled us to examine the racial disparity of the current pandemic in the U.S., even though data for the daily cumulative counts of cases and deaths separated by race is currently unavailable.

Each model estimated a random intercept, and a random slope across days for the MSAs and for the counties nested under the MSAs, to allow for heterogeneity in growth curves across counties and MSAs. Since our maximal model did not converge, we dropped the intercept-slope covariance (Bates et al., 2018)[13]. The day variable was centered, so the main effects can be interpreted as the effects at the mean day of the growth curve. Total population was natural-log-transformed to reduce skewness. All predictors in the model (except day) were z-scored. Post-hoc comparisons of the estimate of slopes across different conditions (high or low segregation, and high or low Gini) were carried out using the emmeans function in R, with *p* value adjusted for multiple comparisons using the Tukey's method.

Results

Confirmed Cases

The effect of Black-White segregation on the growth rate of confirmed cases was first tested. As presented in Table 3.1, the day x segregation interaction was significant, $b = .009$, $p < .001$. Counties located in MSAs with high Black-White segregation showed faster growth of confirmed cases. Although the day x Gini interaction was not significant, $b = .001$, $p = .511$, the 3-way interaction involving day, segregation, and Gini proved significant, $b = .003$, $p = .013$.

[13] We dropped intercept-slope covariance first because if either the random slope or random intercept is dropped, the covariance between the two will be automatically dropped. This way we can ensure that our models retain the most complete random effects structure the data allowed for.

The growth rate was higher for counties with high Gini (+1 SD) located in MSAs with high segregation (+1 SD), as compared to the remaining three conditions (high in Gini/low in segregation, low in Gini/high in segregation, and low in Gini/low in segregation). This difference was statistically significant for the high Gini/low segregation and low Gini/low segregation conditions, slope difference = 0.023, $z = 4.767$, $p < .001$; slope difference = 0.020, $z = 3.811$, $p < .001$, respectively. It was marginal for the low Gini/high segregation condition, slope difference = 0.007, $z = 2.359$, $p = .085$. The pattern is illustrated in the left panel of Fig. 3-1-A.

A comparable analysis was conducted with Hispanic-White segregation (see Table 3.1). The day x segregation interaction was significant, $b = .010$, $p < .001$. Counties located in MSAs with high Hispanic-White segregation showed faster growth of confirmed cases. Although the day x Gini interaction was not significant, $b = .0008$, $p = .552$, the 3-way interaction involving day, segregation, and Gini was significant, $b = .003$, $p = .013$. The growth rate was higher for counties with high Gini (+1 SD) located in MSAs with high segregation (+1 SD), as compared to the remaining three conditions. This difference was statistically significant for the high Gini/low segregation and low Gini/low segregation conditions, slope difference = 0.026, $z = 5.721$, $p < .001$; slope difference = 0.022, $z = 4.528$, $p < .001$, respectively. It was marginal for the low Gini/high segregation condition, slope difference = 0.007, $z = 2.311$, $p = .095$. Among the latter three conditions, counties with high segregation/low Gini also had larger growth rate than the remaining two conditions, ps < .05. The center panel of Fig. 3-1-A illustrates the pattern.

An identical model with Asian-White segregation was tested (see Table 3.1). Unlike in the first two models, there was no significant effect of Asian-White segregation on the growth of cases, $b = .003$, $p = .268$ (the right panel of Fig. 3-1-A). As in the prior analyses the interaction

between day and Gini was not significant, b = .002, p = .176. There was no 3-way interaction between day, segregation, and Gini, b = -.001, p = .617.

Did the covariates have any effects? First, across the three segregation models, there was a significant interaction between day and Black share, b = .007, p < .001 (Black-White segregation model), b = .007, p < .001 (Hispanic-White segregation model), b = .007, p < .001 (Asian-White segregation model). A similar trend is evident for Hispanics. The Day x Hispanic share interaction was significant in all the three models, b = .005, p = .001 (Black-White segregation model), b = .003, p = .022 (Hispanic-White segregation model), b = .005, p = .005 (Asian-White segregation model). The Asian share had no effect. Also, across the three models, both population size and population density of the counties predicted both a faster increase and a larger number of confirmed cases, ps < .05.

Deaths

I performed the same set of three-level mixed effects linear regressions predicting the growth rate of deaths of the counties nested under the MSAs. As shown in Table 3.2, the Black-White segregation model showed a significant day x segregation interaction, b = .012, p < .001. Counties located in MSAs with high Black-White segregation showed faster growth of deaths. The interaction between day and Gini was not significant, b = .003, p = .180. As in the analysis of confirmed cases, the 3-way interaction involving day, segregation, and Gini proved significant, b = .007, p < .001. The growth rate was significantly higher for counties with high Gini (+1 SD) located in MSAs with high segregation (+1 SD), as compared to the remaining three conditions (low in Gini/high in segregation, high in Gini/low in segregation, and low in both), slope difference = 0.020, z = 4.110, p < .001; slope difference = 0.038, z = 5.847, p < .001;

slope difference = 0.030, $z = 4.198$, $p < .001$, respectively. The latter three conditions did not differ from each other, ps > .10. This is illustrated in the left panel of Fig. 3-1-B.

The Hispanic-White segregation model showed a parallel pattern (see Table 3.2). The day x segregation interaction was significant, $b = .009$, $p = .003$, indicating greater growth rate of COVID-19 deaths for segregated MSAs (the center panel of Fig. 3-1-B). The day x Gini interaction was marginally significant, $b = .004$, $p = .085$. The 3-way interaction involving day, segregation, and Gini was significant, $b = .006$, $p = .002$. The growth rate was significantly higher for counties with high Gini (+1 SD) located in MSAs with high segregation (+1 SD), as compared to the remaining three conditions (low in Gini/high in segregation, high in Gini/low in segregation, and low in both), slope difference = 0.019, $z = 3.691$, $p = .001$; slope difference = 0.030, $z = 4.486$, $p < .001$; slope difference = 0.026, $z = 3.533$, $p = .002$, respectively. The latter three conditions did not vary significantly, ps > .20.

In the third model, the Asian-White segregation was tested (see Table 3.2). The effect of segregation on the rate of increase in deaths is only marginal, $b = .006$, $p = .064$. However, the day x Gini interaction was significant, $b = .005$, $p = .021$. Counties with more income inequality had a steeper increase in deaths due to COVID-19. The 3-way interaction involving day, segregation and Gini was negligible, $b = .001$, $p = .600$. (see the right panel of Fig. 3-1-B).

Did the covariates have any effects on the growth rate of deaths? First, across the three models, there was a clear evidence that counties with higher Black share reported a greater rate of increase in deaths, $b = .007$, $p = .003$ (Black-White segregation model), $b = .007$, $p = .002$ (Hispanic-White segregation model), $b = .007$, $p = .006$ (Asian-White segregation model). There was virtually no evidence that the share of either Hispanics or Asians had any effects. As in the analysis of confirmed cases, population size also predicted a greater rate of increase in deaths

and a larger total number of deaths, *p*s < .001. The effect of population density was also similar, but compared to the analysis of confirmed cases, it was much weaker.

Robustness Checks and Additional Analyses

To check the robustness of the findings above, several variations of the analysis were carried out. These supplementary analyses, along with the results, can be found in Appendix A.

Discussion

In the present work, it was found that the growth rate of COVID-19 cases and deaths was higher for MSAs that exhibit greater Black-White segregation and Hispanic-White segregation. Further, this effect of residential racial segregation was exacerbated by income inequality in the area. The effect demonstrated here is not trivial. For example, imagine if the Detroit metro area had been racially less segregated on the Black-White axis, so that it was at the same level as Albuquerque, NM. Simultaneously, imagine that the counties included in this metro area were economically more equal, perhaps at the level of Ionia county of Grand Rapids-Wyoming MSA, MI. Our model shows that under such conditions, the Detroit metro area would have suffered only 45.4% of the deaths reported by the end of the 30-day study period (744 predicted deaths, as compared to 1639 actual deaths). Together, the findings illustrate the lethal nature of structural inequalities.

Both segregation and income inequality have received substantial research attention in sociology, demography, and public health (Akee et al., 2019; Cooper et al., 2001; Hunter & Robinson, 2016; McCall & Percheski, 2010). However, rarely has this literature studied the two factors together. The current work is the first to demonstrate that combining the two factors has a synergistic effect on infections and deaths during a pandemic. Given the massive racial disparity in net wealth (Pew Research Center, 2011), segregated Black and Hispanic communities are

more likely deprived of a wide range of social, medical, and other related resources. When income inequality is also high, it exacerbates the poverty of segregated minority enclaves while also resulting in impoverished White enclaves (Case & Deaton, 2015; Massey & Fischer, 2000; Quillian, 2012). The combination of segregation and income inequality thus yields larger areas plagued with poverty and deprived of social, medical, and community-level resources within the metropolitan area. Such communities will then be more vulnerable to an infectious disease such as COVID-19. Conversely, reduced income disparity may buffer the malignant effect of segregation.

The current work implies that the joint effects of segregation and income inequality may have particularly dire consequences on racial or ethnic minorities, especially Blacks and Hispanics. Our data, however, are the counts of confirmed cases and deaths from the entire county, since race-stratified daily counts of cases or deaths at the county-level are not available at present. Therefore, it is currently impossible to be specific about any race-specific patterns. Nevertheless, we found that the spread of the disease was faster in counties with a higher share of Blacks. A comparable effect was much weaker for Hispanics in the analysis of deaths. Over many decades, the segregation of Black communities has been enforced and instituted particularly strictly in many large American cities (Massey & Denton, 1993; Williams & Collins, 2001), consistent with a higher mean of the dissimilarity index of segregation for Blacks than for either Hispanics or Asians. Thus, the segregated enclaves of Blacks may be mired with particularly insidious conditions. This finding is in line with the suggestion that the "hyper-segregation" (Massey & Denton, 1989) in American cities is a fundamental root cause of racial disparity in educational attainment, socio-economic status, and health (Massey & Denton, 1989; Williams & Collins, 2001).

It is unknown exactly how much impact segregation and income inequality had on Whites, particularly, Whites not plagued with dire poverty. At present, approximately 50% of all cases and deaths in the U.S. come from the White population[14]. Further, in the current data, the share of at least one of the two marginalized groups (Hispanics) did not predict increased growth of death in the area at large. This finding suggests that the lethal consequences of structural inequality may be widely shared across the entire area, not strictly limited to the minority groups alone.

What about Asians? There was weak evidence for the day x segregation interaction for deaths, but not for cases. Unlike in the case of Blacks and Hispanics, this effect was not moderated by income inequality (see Table 3.1.1 and 3.1.2). Despite the prejudice and stereotypes Asians and Asian Americans must contend with (Devos & Banaji, 2005; Suzuki, 1977), the median household wealth is no different between Asians and Whites in the U.S. (Pew Research Center, 2016b). Hence, the White-Asian segregation may be less likely to result in the concentration of poverty in Asian enclaves.

Recently, Kraus and colleagues (Kraus et al., 2019) found that representative samples of Americans estimated the current average wealth of Blacks to be 90% of the current average wealth of Whites in 2016. The respondents also estimated the average wealth of Blacks to be 50% of the average wealth of Whites approximately a half-century earlier, in the early 1960s. The true percentages, however, are mere 10% and 5%, respectively. Thus, most Americans fail to register the massive wealth disparity between Whites and minority groups such as Blacks and Hispanics. Moreover, they seem to believe in what would appear to be a remarkable progress toward racial equality. The authors suggest that the optimistic mythology of racial progress is

[14] https://covid.cdc.gov/covid-data-tracker/#demographics

false in economic domains and self-deceiving. It may perpetuate racism by blinding them to the racism that still pervades. In the present case, the false optimism could make it all the more difficult for Americans to realize the role of segregation in producing the devasting human toll during the pandemic.

Some limitations of the current work must be noted. First, as noted above, the county-wide statistics did not stratify the daily count of either infections or deaths by race. Future work must test the growth rate of both confirmed cases and deaths separately for different ethnic groups. Second, the current analysis did not include all racial minority groups that had suffered disproportionately (e.g., Native Americans). This is a major omission that must be rectified in future work. In such an effort, sufficient care must be taken to differentiate the varying historical reasons for segregation of different minority groups. Third, the current work is limited to the United States. It is unknown if a similar dynamic might exist in other countries that are also severely affected by COVID-19. Fourth, the current work focused on the effect of social structural inequality at a macro-level. It remains to be tested how this effect may interact with individual-level dynamics such as the frequency and diversity of social interactions (Salvador et al., 2020). Finally, as important as pandemics are, there is more to social life than virus infection. Future work must test whether the adverse effect of segregation and income inequality might generalize to other domains, such as life satisfaction and the community's well-being (Oishi et al., 2011). It will also be informative to test how certain social advantages (e.g., group cohesion) linked to segregation (Nuru-Jeter & LaVeist, 2011), especially among racial minority groups, might interact with the negative effects of structural inequality in these psychological domains.

Despite these limitations, the present study provides the first evidence that racial residential segregation and income inequality yield a synergetic effect of producing a “lethal

spiral" that leads to a greater number of fatalities during a pandemic. In closing, it should be noted that the current work does not imply that segregated Black/Hispanic enclaves should be blamed for spreading the virus. These enclaves are plagued with an assortment of adverse health conditions because of the existing structural inequality which, in turn, renders them particularly vulnerable to infectious diseases. Thus, more effort is warranted to eliminate discriminatory institutional practices that reinforces segregation and economic disparity. This effort would be indispensable for making our cities both virus-resistant and virus-resilient in the forthcoming era of infectious disease (Delaney & Reed, 2015).

Table 3-1 Regression coefficients for confirmed COVID-19 cases for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models. The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	4.514	100.120	<.001	4.523	100.608	<.001	4.523	93.686	<.001
Variables of Interest									
Day	0.070	31.768	<.001	0.070	33.761	<.001	0.071	29.460	<.001
Segregation	0.166	3.604	<.001	0.167	3.683	<.001	0.061	1.209	0.229
Day x Segregation	0.009	3.936	<.001	0.010	4.806	<.001	0.003	1.114	0.268
GINI	0.019	0.770	0.441	0.023	0.949	0.343	0.032	1.339	0.181
Day x GINI	0.001	0.658	0.511	0.001	0.595	0.552	0.002	1.356	0.176
Segregation x GINI	0.031	1.507	0.132	0.006	0.293	0.769	-0.029	-1.278	0.202
Day x Segregation x GINI	0.003	2.497	0.013	0.003	2.483	0.013	-0.001	-0.501	0.617
Minority Share									
Blacks Share	0.132	4.634	<.001	0.131	4.605	<.001	0.134	4.647	<.001
Day x Blacks Share	0.007	4.809	<.001	0.007	5.043	<.001	0.007	4.686	<.001
Hispanics Share	0.069	2.233	0.026	0.047	1.542	0.124	0.062	1.933	0.054
Day x Hispanics Share	0.005	3.279	0.001	0.003	2.304	0.022	0.005	2.857	0.005
Asians Share	-0.032	-1.226	0.221	-0.036	-1.373	0.170	-0.040	-1.502	0.134
Day x Asians Share	0.002	1.338	0.182	0.002	1.205	0.229	0.001	0.950	0.342
Covariates									
Natural Log of Population Size	0.789	27.565	<.001	0.798	28.028	<.001	0.800	27.912	<.001
Day x Natural Log of Population Size	0.034	22.506	<.001	0.035	23.179	<.001	0.035	22.795	<.001
Median Income	0.025	0.864	0.388	0.017	0.601	0.548	0.031	1.045	0.296
Day x Income	0.001	0.659	0.510	0.001	0.329	0.742	0.001	0.838	0.403
Population Density	0.067	2.941	0.003	0.080	3.461	0.001	0.087	4.078	<.001
Day x Population Density	0.003	2.438	0.015	0.003	2.521	0.012	0.004	3.803	<.001
Proportion Elderly	0.007	0.247	0.805	0.006	0.216	0.829	0.013	0.470	0.638

Day x Proportion Elderly	0.000	0.288	0.773	0.000	0.235	0.814	0.001	0.525	0.600

Table 3-2 Regression coefficients for COVID-19 deaths for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models. The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	1.311	23.987	<.001	1.325	23.761	<.001	1.320	22.474	<.001
Variables of Interest									
Day	0.067	23.759	<.001	0.068	22.860	<.001	0.068	21.787	<.001
Segregation	0.210	3.724	<.001	0.185	3.196	0.002	0.125	1.962	0.052
Day x Segregation	0.012	4.099	<.001	0.009	2.993	0.003	0.006	1.871	0.064
GINI	0.091	2.039	0.042	0.090	2.040	0.042	0.111	2.512	0.012
Day x GINI	0.003	1.343	0.180	0.004	1.728	0.085	0.005	2.321	0.021
Segregation x GINI	0.049	1.360	0.175	0.063	1.661	0.097	-0.017	-0.423	0.672
Day x Segregation x GINI	0.007	4.124	<.001	0.006	3.140	0.002	0.001	0.525	0.600
Minority Share									
Blacks Share	0.145	3.179	0.002	0.142	3.102	0.002	0.142	3.029	0.003
Day x Blacks Share	0.007	2.977	0.003	0.007	3.070	0.002	0.007	2.777	0.006
Hispanics Share	0.027	0.583	0.560	-0.018	-0.403	0.687	0.021	0.429	0.668
Day x Hispanics Share	0.001	0.239	0.812	-0.002	-0.819	0.413	0.000	0.166	0.868
Asians Share	-0.069	-1.602	0.110	-0.069	-1.599	0.111	-0.084	-1.935	0.054
Day x Asians Share	0.001	0.529	0.597	0.001	0.377	0.706	-0.000	-0.036	0.971
Covariates									
Natural Log of Population Size	0.678	13.981	<.001	0.689	14.257	<.001	0.700	14.323	<.001
Day x Natural Log of Population Size	0.039	16.547	<.001	0.040	16.893	<.001	0.041	16.890	<.001
Median Income	0.130	2.654	0.008	0.113	2.293	0.022	0.155	3.097	0.002
Day x Income	0.003	1.125	0.261	0.002	0.849	0.396	0.004	1.654	0.099
Population Density	0.096	2.413	0.016	0.095	2.372	0.018	0.127	3.400	0.001
Day x Population Density	0.002	0.852	0.395	0.002	1.242	0.215	0.005	2.635	0.009
Proportion Elderly	0.057	1.290	0.198	0.055	1.255	0.210	0.069	1.540	0.124
Day x Proportion Elderly	0.003	1.198	0.232	0.003	1.273	0.204	0.004	1.581	0.115

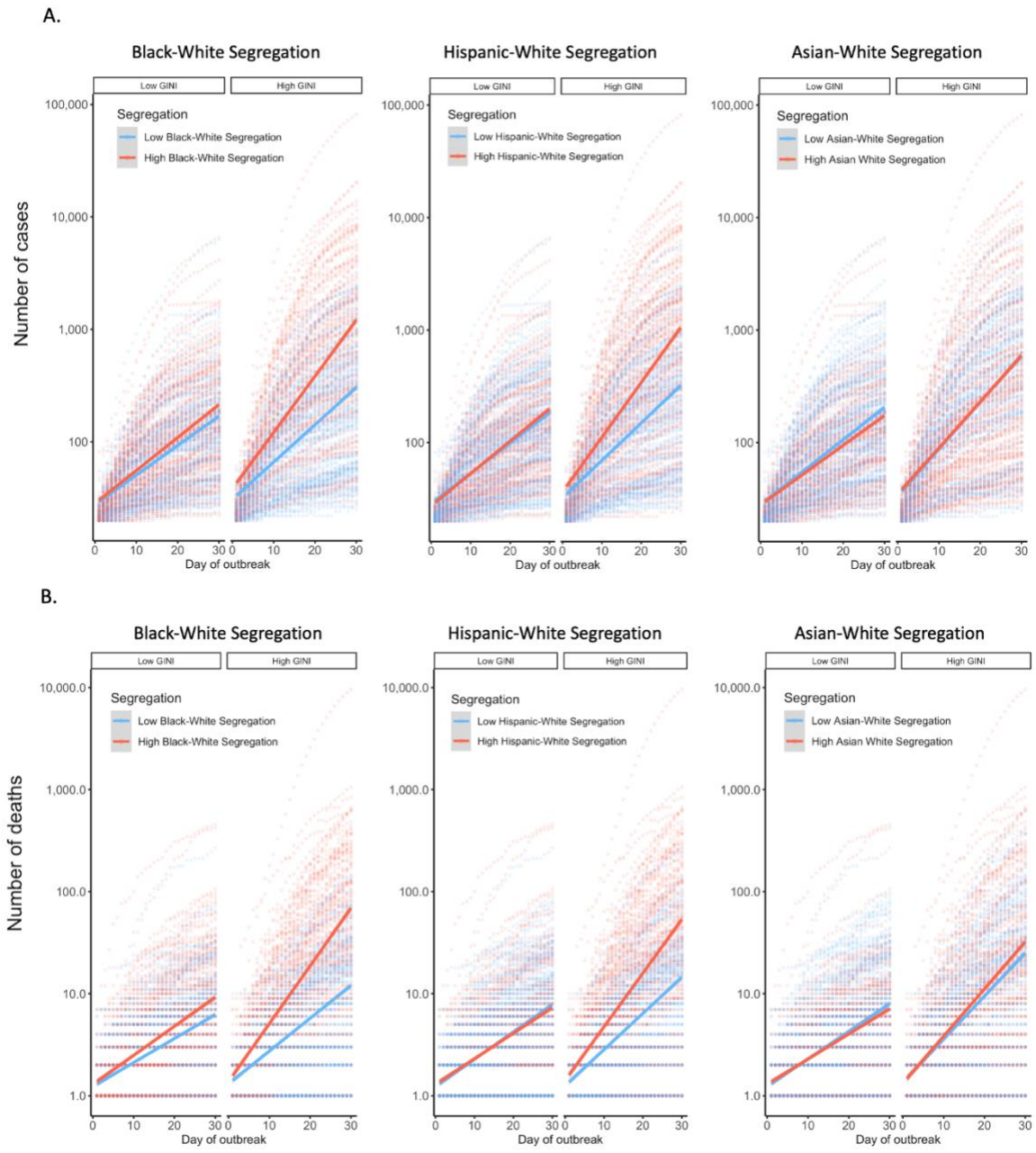

Figure 3-1 The growth of confirmed COVID-19 cases (A) and deaths (B) on a log-scale during the first 30 days of the county-wide outbreaks.

The growth trend of each of the 535 counties (case analysis) and 495 counties (death analysis) under the 100 largest U.S. metropolitan areas are plotted with dotted lines, as a function of high vs. low Gini (median split) and high vs. low racial segregation (median split). The solid lines in the figure are the best fit line across all data points within each of the conditions defined by the combination of Gini and segregation.

Study 2

The findings of Study 1 illustrate how structural inequality underlying systemic racism may exacerbate COVID-19 fatalities. While this study elucidates the mechanism of the lethal consequences of systemic racism at the collective level, this study by itself does not provide any insight on how the subjective perception of discrimination is associated with health outcomes at the individual level. Study 2 is thus set out to address this question.

Racial discrimination, as demonstrated in Study 1, entails substantial economic (Kraus et al., 2017), residential (Williams & Collins, 2001), and medical (Hall et al., 2021) disadvantages, and it is thus unpleasant and threatening to the individual. Hence, many previous studies found that perceived discrimination is associated with various health problems (Pascoe & Richman, 2009; Williams et al., 2003; Williams & Mohammed, 2009). However, it should be noted that some studies in the literature found no such association, or even a reversed pattern, especially among Black Americans (Fuller-Rowell et al., 2012; Gibbons et al., 2021; Krieger & Sidney, 1996), and especially when long-term health outcome measures (e.g., all-cause mortality) are tested. Prior theorization has thus suggested that although perceived discrimination is stressful to the individual at the given moment, the willingness to acknowledge and report the discrimination could signal resilience of the individual (Crocker & Major, 1989; Fuller-Rowell et al., 2012; Major et al., 2002). So far, not much is known about what psychological or behavioral processes may constitute this resilience. It is also unclear whether this resilience can lead to positive long-term health outcomes, such as reduced mortality risk. In this study, I aim to fill this gap by testing the hypothesis that resilience among Black Americans against perceived racial discrimination is reflected in more constructive coping, increased purpose in life, and greater

educational attainment. Moreover, I examine whether this resilience, in turn, would predict reduced mortality risk.

Perceived Discrimination and Health Outcomes

Previous studies have used various health outcome measures, such as subjective wellbeing (Schmitt et al., 2014), depression and anxiety (Pascoe & Richman, 2009; Schulz et al., 2006), and biological stress markers, such as pro-inflammatory cytokines (Stepanikova et al., 2017) and blood pressure (Dolezsar et al., 2014), and found that perceived discrimination is stressful for both Black and White Americans. Anti-Black discrimination is particularly pervasive, and thus, it is plausible that the stress caused by discrimination contributes to the wide health disparity between Blacks and Whites in the U.S. (Williams & Mohammed, 2009).

Notably, however, there are some exceptions to the general pattern linking perceived discrimination to poor health, especially among Black Americans. This paradoxical pattern seems to show up in certain biological markers of health as well as mortality risk, all of which arguably reflect processes that unfold over a relatively long period, especially compared to subjective health, the most common health measure in this literature. For example, one study tested working-class Black adults and found that systolic blood pressure was lower for those who reported discrimination than for those who did not (Krieger & Sidney, 1996). Another study found that the reported level of daily discrimination was associated with flatter (unhealthy) diurnal cortisol rhythm for White Americans, but this association was steeper (healthier) for Black Americans (Fuller-Rowell et al., 2012). Demonstrating a similar pattern for mortality risk, LaVeist et al. (2001) found that Black Americans who reported recent experience of discrimination ("during the past month") and blamed the social system for it had a lower mortality risk over a 13-year period than those who reported no recent experience of

discrimination. In another study, Barnes et al. (2008) found that perceived discrimination predicted increased mortality for both Black and White Americans, but this effect was weaker for Black Americans than for White Americans.

Perceived Discrimination and Resilience Among Black Americans

Why does perceived discrimination sometimes predict better health outcomes for Black Americans when it is stressful and unpleasant? These seemingly puzzling findings may become sensible when understood in the context of American society. It is increasingly clear that anti-Black discrimination is systemic. Discriminatory practices can be clearly observed in how Black people are treated in classrooms (Lopez & Jean-Marie, 2021) and courtrooms (Sommers & Ellsworth, 2000), how they are approached by the police (Voigt et al., 2017), and how they are segregated into poor neighborhoods (Massey, 1990). Anti-Black discrimination is institutionalized, and thus, inherent in the social and cultural systems of the U.S. Hence, it is virtually impossible for Black Americans to escape discriminatory treatment by society.

Under such racialized social reality, reporting discriminatory treatments may carry unique meanings among Black Americans. Prior work has suggested that when discrimination undeniably exists in one's daily life, the willingness to acknowledge and report discrimination indicates a well-adjusted and resilient psyche (Fuller-Rowell et al., 2012; Major et al., 2002; Sellers et al., 1998). This resilience may be reflected and reinforced through several psychological and behavioral processes. First, recognizing negative events and unfair treatments as due to discrimination and reporting the discrimination signify that one is well-equipped to deal with discrimination through constructive processes such as active coping and positive reappraisal (Crocker & Major, 1989; Major et al., 2002). These processes could be more effective in dealing with discrimination, compared to if one is unwilling to recognize and report discrimination,

which may reflect avoidance, suppression, or denial. Second, in part because it is highly stressful and painful, the explicit acknowledgment of race-based discrimination may motivate Black Americans to identify with their group (McCarthy & Yancey, 1971), thereby confronting racism (Czopp et al., 2006) to seek justice through social movements and collective actions (Leach & Allen, 2017). The resulting boost in psychological well-being (Branscombe et al., 1999), particularly the sense of purpose in life (to combat racism on behalf of one's group), is known to have various salubrious effect on health (Hill & Turiano, 2014; E. S. Kim et al., 2014). In addition, the active coping effort and the sense of purpose may be linked to the aspiration to pursue higher education (Blum et al., 2012; Hill et al., 2016; McKnight & Kashdan, 2009), which would give Black Americans greater power, status, and voice to address social injustice. Altogether, the constructive coping effort, purpose in life, and educational attainment may be critical components of resilience against racism among Black Americans. This resilience, in turn, may entail various health benefits. It is possible that these health benefits of resilience can sometimes accumulate and eventually outweigh the negative health impacts of racism, resulting in better health in the long run, including reduced mortality.

The existing evidence supports that each of the three putative components of resilience predicts better health. First, prior work distinguishes between problem-focused coping, which emphasizes active coping and positive reinterpretation, and emotion-focused coping, which involves venting of emotions and denial (Carver et al., 1989). This work shows that problem-focused coping is more effective in mitigating psychological distress linked to adversities (Carver et al., 1989; John & Gross, 2004; Jorgensen & Thibodeau, 2007), including racism (Kaholokula et al., 2017; Pittman, 2011). Second, another line of work shows that having a purpose in life is a robust predictor of reduced mortality risk (Alimujiang et al., 2019; Boyle et

al., 2009; E. S. Kim et al., 2014). Third, it has been well-established that educational attainment is a strong predictor of various health outcomes, including reduced mortality (Hummer & Hernandez, 2013; Zajacova & Lawrence, 2018). In combination, the cumulative evidence makes it likely that the reporting of discrimination predicts reduced mortality risk as it in part reflects resilience among Black Americans.

The Present Study

In the present study, I used large community samples of both Black and White Americans from the Midlife in the U.S. (MIDUS) study (Kessler et al., 1999; Ryff et al., 2003) to test my predictions. I first tested whether perceived discrimination is associated with resilience as measured by more constructive coping, greater purpose in life, and higher educational attainment among Black Americans. I then tested whether perceived discrimination prospectively predicted reduced mortality risk among Black Americans, and whether this link is explained by the degree of resilience. The same analysis is applied to White Americans as well.

Method

Participants

Participants of the current study were drawn from the MIDUS Study, a national study on health and wellbeing. The details of the data collection and the participant characteristics have been documented in prior studies. In brief, the first wave of collection (called M1) started in 1995-1996, and it recruited a national probabilistic sample of 7108 adults through random digit dialing. The data collection involves a 30-min phone interview and two self-administered questionnaires on demographic information, life experience, and a series of psychosocial constructs. This sample was later followed up during 2004-2006 (called M2), where 4963 participants were re-examined. The M2 collection also recruited a new Black American city

sample (N = 592) from Milwaukee, WI to examine health and wellbeing among racial minorities. The main national sample and Black American city sample were then followed up (called M3) during 2013-2014 and during 2016-2017, respectively, where 3294 of the main national sample and 389 of the Black American city sample were re-examined. Lastly, a separate national probabilistic sample (N = 3577) were recruited during 2012-2013 (called MR) as an attempt to replenish the original MIDUS cohort. The MR main national sample was accompanied by a Black American city sample from Milwaukee (N = 508) as well. Hence, the present study used the four samples to examine and internal replicate the relationship between perceived discrimination and the three proposed components of resilience, among Black[15] and White Americans. The longitudinal cohort was used to examine if perceived discrimination predicts mortality risk among Black and White Americans. See Table 3.3 for sample characteristics, including sample sizes for Black and White Americans.

Measures

Perceived discrimination. The perceived lifetime discrimination of each participant was measured. Participants were asked *how many times* in their life they had been discriminated against because of such things as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics. They answered this question for 11 items that describe possible ways that one can be discriminated against, such as being discouraged by a teacher or advisor from seeking higher education, not hired for a job, being hassled by the police, being denied or provided inferior medical care. For each participant, the score for perceived lifetime discrimination was constructed by counting the number of items that the participant

[15] Starting from M2, participants were asked to identify more than one racial origin, if they have more than one. Hence, for those whose primary racial origin was White, but the secondary racial origin was Black, we categorized them as Blacks. This was because the hypodescent, or the "one drop" rule, as identified by prior literature (Ho et al., 2011).

answered 1 time or higher. Hence, the possible score ranges from 0 to 11, where 0 refers to no discrimination experienced at all, and 11 refers to being discriminated against in all 11 domains. Of note, the perceived discrimination measure itself did not ask for the basis of discrimination. Nevertheless, participants were asked to indicate after the question the main reason for the discrimination they reported. Across the four waves of collection, about 65% of Black participants and about 32% of White participants volunteered their responses. Among these participants, about 81% of Black participants indicated race/ethnicity as the single most important reason for discrimination, whereas White participants referred primarily to age (31%), sex (37%), and weight/height (19%).

Purpose in life. Purpose in life was measured as one of the domains for eudaimonic psychological wellbeing. It was measured with seven items (only three items were used in M1). Sample items include "I live life one day at a time and don't really think about the future", "Some people wander aimlessly through life, but I am not one of them" (reverse-coded), "I sometimes feel as if I've done all there is to do in life". Participants rated each item using a 7-point Likert scale, 1 indicates "strongly agree" and 7 indicated "strongly disagree". All items except the reverse-coded ones were reversed, so that a higher score reflects a higher standing on the scale. The items for the scale were summed to produce the final score of the purpose in life. The reliability (assessed using Cronbach's alpha) of the scale was adequate for all the samples ($\alpha > 0.65$) except for M1, where only three items were used.

Coping. Two types of coping were measured, namely, problem-focused coping and emotion-focused coping. Problem-focused coping scale included 12 items encompassing three domains: positive reinterpretation and growth, planning, and active coping. Emotion-focused coping scale included 12 items encompassing another three domains: focuses on and venting of

emotion, denial, and behavioral disengagement. Participants were asked what they generally do and feel when they experience stressful situations, and they rated each item on a 4-point Likert scale, 1 indicates "a lot" and 4 indicated "not at all". Sample item for "positive reinterpretation and growth" includes "I try to see it in a different light, to make it seem more positive. Sample item for "planning" includes "I try to come up with a strategy about what to do". Sample item for "active coping" includes "I concentrate my efforts on doing something about it". Sample item for "Focuses on and venting of emotion" includes "I get upset and let my emotions out". Sample item for "Denial" includes "I pretend that it hasn't really happened". Sample item for "Behavioral Disengagement" includes "I admit to myself that I can't deal with it, and quit trying". Items in both problem-focused coping and emotion-focused coping scale were summed after reversed, so higher score indicates higher standing on the scale. The reliability for both scales across the samples were high (αs > .80). The difference score between the two coping scales (subtracting emotion-focused coping from problem-focused coping) was calculated to quantify the degree of "constructive coping". Note that the coping scale was not included in M1, so it was only analyzed in the remaining three samples (M2, M3, and MR).

Education. Educational attainment was measured as the highest level of education completed, ranging from 1 (No school/some grade school) to 12 (PhD or other professional degree).

Covariates

Age, sex, and income were included as covariates in all the analyses in the present work. Total personal income was used in M2, M3, and MR. Total household income was used in M1. A few other health-related covariates that may influence the analysis of mortality were also included. The details of these covariates are shown below.

Health behaviors. Two variables measuring health behaviors, alcohol consumption and smoking status, were included. Alcohol consumption was measured as the average number of alcohol drinks consumed per week. Smoking status was coded as a categorical variable with 0 being current smoker, and 1 being non-smoker.

Health status. A few measures of health status were also included. In particular, BMI was computed from weight and height measurements obtained by clinic staff. In addition, the extent of functional limitations self-reported by the participants was included. Participants rated on a scale of 1 to 4 (1 – A lot, 4 – Not at all) whether their health limits them to carry on 7 daily activities (e.g., lifting or carrying groceries, climbing several flights of stairs, walking several blocks). I calculated the mean of all items, after reversing them, so that higher number indicated a greater extent of functional limitations.

Mental health status. I also controlled for depressive tendencies or anxiety symptoms of the participants. For depression, participants were asked during the past 12 months, when they felt sad, blue, or depressed for two weeks or more, if they experienced the following seven symptoms: (a) lose interest in most things, (b) feel more tired out or low on energy than is usual, (c) lose your appetite or appetite increased, (d) have more trouble falling asleep than usual, (e) have a lot more trouble concentrating than usual, (f) feel down on yourself, no good, or worthless, and (g) think a lot about death. The total number of "yes" was counted to construct the score for depressed affect, ranging from 0 to 7. If they reported that they didn't feel sad, blue, or depressed for two weeks or more at all, it was scored as 0. For anxiety, participants were asked how often in the past 12 months that they (a) were restless because of your worry, (b) were keyed up, on edge, or had a lot of nervous energy, (c) were irritable because of your worry, (d) had trouble falling asleep, (e) had trouble staying asleep because of your worry, (f) had trouble

keeping your mind on what you were doing, (g) had trouble remembering things because of your worry, (h) were low on energy, (i) tired easily because of your worry, and (j) had sore or arching muscles because of tension. They rated each item on a four-point scale, ranging from 1 – most days, 2 – about half the days, 3 – less than half the days, to 4 – never. The final score for anxiety was calculated by counting the number of items that they rated "most days".

Statistical Analysis

I used multiple regression to test whether perceived discrimination may predict coping, purpose in life, and education among Black and White Americans across the four MIDUS waves. In the main analysis presented here, I report the result by aggregating the three resilience measures as a single composite score and used it as the outcome. In particular, coping, purpose in life, and education were standardized within each racial group and averaged to create a composite score of resilience[16]. The three putative measures of resilience were positively correlated with one another for both Black and White Americans (*r*s > .20), leading to an adequate reliability of the resilience composite for both Black and White Americans (αs = 0.63 and 0.59, respectively[17]). For all analyses, I first used perceived discrimination as the only predictor of the resilience composite score, followed by controlling for the covariates, including age, sex, and income. I also tested whether perceived discrimination may predict coping, purpose in life, and education separately, among both Black and White Americans. Overall, the results of testing each variable separately were consistent with what was found using the resilience composite, and none of the variable uniquely drove the effect observed for the resilience composite. The results for this analysis are included in Appendix B.

[16] Because the coping scale was not included in M1, only purpose in life and education were aggregated for the analysis on M1 sample.
[17] This reliability score was based on M2 sample. The reliability was similar for M3 and MR.

Mortality Analysis

I tested whether perceived discrimination at the baseline time point predicted increased risk of mortality (or reduced likelihood of survival) thereafter, separately for Black and White Americans. Because there was a lack of Black participants at the M1, I chose M2 as the baseline time point, when the first large sample of Black Americans were recruited from Milwaukee, WI. As mentioned above, both the main national sample and the Black American sample of M2 were followed up at M3. In addition, their vital status was kept track of since M2. The mortality information of the participants in the MIDUS study, most recently updated in 2020, was obtained. The deceased status was verified using the National Death Index (NDI) Plus search. The mortality information includes the cause of death, as well as the month and year of the death of participants who are known to have deceased. Among White Americans, 1017 out of 4345 participants have deceased since the baseline. Among Black Americans, 200 out of 834 participants have deceased since the baseline.

The Cox proportional hazards model (Cox, 1972) was used to analyze the time to death as a function of perceived discrimination. The time duration to death (number of days) was calculated by taking the difference between the date when participants completed the initial phone interview at M2, the baseline, and the date when the participants deceased. Because there is no date information for the mortality data, only month and year, we used the first day of the month for all deceased participants to calculate the time to death. For those participants whose mortality information was missing, they were censored using the date of the phone interview at M3 (i.e., the time duration from the M2 phone interview and M3 phone interview was calculated), if they were actually followed up at M3. If they weren't followed up at M3, they

were dropped from the analysis because there is no information at all regarding their vital status after the baseline collection.

Mediation Analysis

I also performed a mediation analysis testing if the composite resilience variable serves as a significant mediator in the relationship between perceived discrimination and mortality. I used the Monte Carlo Method for Assessing Mediation as documented in MacKinnon et al. (2004). Briefly, the method is based on the assumption that the coefficient of the *a* and *b* path (the beta coefficient by regressing mediator on predictor, and the beta coefficient by regressing outcome on the mediator, respectively) are normally distributed. A repeated random draw from the joint distribution of *a* and *b* was performed 10000 times, creating the distribution of *a*b*, the mediation path. The 95% confidence interval was determined based on the distribution, and it was used to determine the significance of the mediation effect.

The mediation analysis was performed for both Black and White Americans. I assessed the mediation effect first controlling for demographic variables and then additionally controlling for the health-related variables.

Results

Perceived Discrimination and Resilience

As can be seen in Fig. 3.2, the relationship between perceived discrimination and the resilience composite score was different for Black and White Americans. Among Black Americans, the association was significantly positive [M1: $b = 0.100$, $t(299) = 5.515$, $p < .001$; M2: $b = 0.064$, $t(735) = 5.631$, $p < .001$; M3: $b = 0.078$, $t(487) = 5.172$, $p < .001$; MR: $b = 0.047$, $t(681) = 3.320$, $p < .001$]. However, it was either virtually zero or even sometimes negative for White Americans [M1: $b = 0.006$, $t(5451) = 0.689$, $p = .491$; M2: $b = -0.010$, $t(3455) = -1.084$, p

= .278; M3: b = -0.025, t(2407) = -2.284, p = .022; MR: b = -0.041, t(2011) = -3.684, p < .001]. When the resilience composite score was regressed on both race and perceived discrimination, as well as their interaction term, the interaction between the two variables was significant in all waves of data, ps < .001. The results above remained unchanged when age, sex, and income were included as covariates (see Appendix B for detailed coefficients).

Perceived Discrimination and Mortality Risk

I found that among Black Americans, perceived lifetime discrimination predicted a reduced risk of mortality (see Table 3.4 for beta coefficients). This was the case when age, sex, and income was controlled (Hazard Ratio [HR] = 0.918, 95% confidence interval [CI] = [0.857, 0.983], p = .015), and when health-related variables including alcohol usage, smoking status, BMI, depression and anxiety symptoms, and functional limitation were additionally controlled (HR = 0.920, 95% CI = [0.858, 0.987], p = .020). The HR suggests that in the full model, a one-point increase on the perceived discrimination measure (i.e., one additional domain of discrimination experienced) conferred approximately an 8% decrease in mortality risk.

In contrast, among White Americans, perceived lifetime discrimination predicted an increase in mortality risk when controlling for age, sex, and income (HR = 1.087, 95% CI = [1.021, 1.157], p = .009). However, this effect became non-significant when health-related variables were additionally controlled (HR = 1.046, 95% CI = [0.979, 1.117], p = .181). The HR in the full model suggests that a one-point increase in perceived lifetime discrimination yielded approximately a 5% increase in mortality risk, albeit not significantly. See Table 3.4 for beta coefficients of the model.

I plotted the estimated survival probability and the 95% CI as a function of time from the baseline for both White and Black Americans (Fig. 3.3 and 3.4). As evident in the figures, the

effect of perceived discrimination on mortality risk was markedly different among White versus Black Americans. The interaction between race and perceived lifetime discrimination was significant both when controlling for age, sex, and income, $b = 0.008$, $z = 3.406$, $p < .001$, and when all covariates were controlled, $b = 0.006$, $z = 2.582$, $p = .010$.

The Mediating Role of Resilience

We then tested whether the relationship between perceived discrimination and mortality risk was mediated by the resilience composite (comprised of constructive coping, purpose, and educational attainment) for each racial group. We found that among Black Americans, perceived discrimination predicted a higher resilience, which in turn, predicted reduced mortality risk. This was the case both when controlling for demographic variables only, $b = -0.022$, 95% CI = [-0.040, -0.009], and when additionally controlling for health-related variables, $b = -0.023$, 95% CI = [-0.041, -0.007]. This mediation, however, was not significant among White Americans, either controlled for demographic variables, $b = 0.004$, 95% CI = [-0.002, 0.010], or additionally controlling for health-related variables, $b = -0.003$, 95% CI = [-0.008, 0.0001]. The mediation graph can be found in Fig. 3.5.

Discussion

The current data shows that perceived discrimination is a reliable predictor of reduced mortality risk for Black Americans, consistent with a few prior studies offering similar evidence (Fuller-Rowell et al., 2012; Gibbons et al., 2021; Krieger & Sidney, 1996). This finding might seem paradoxical since many other studies show that perceived discrimination is linked reliably to poorer health outcomes (Williams & Mohammed, 2009). However, most of these studies examine self-report measures of health (e.g., subjective wellbeing, depression, and anxiety).

Thus, although perceived discrimination is experienced as stressful at the moment, it may predict better health in the long run, including reducing mortality risks.

In particular, I hypothesized and showed that the perception and reporting of discrimination among Black Americans, which is largely race-based, reflect a greater resilience in the face of oppression and injustice. They are motivated to cope with the adversities in life in a more problem-oriented (vs. emotion-focused) fashion. They may also hold future goals and purposes beyond their immediate life circumstances (e.g., addressing social injustice). The resulting sense of purpose in life may entail a desire to seek more education, which provides a means to increase their status and "voice" in the fight against social injustice. Indeed, these three components of resilience were correlated with one another and, when taken together, they largely accounted for the effect of perceived discrimination in reducing mortality risk. Thus, our findings are consistent with prior work suggesting that the explicit acknowledgment that one is being treated unfairly due to their race may signify one's willingness to face the discrimination and cope with it as a member of the group (Fuller-Rowell et al., 2012; Major et al., 2002; Sellers et al., 1998).

One implication of this line of theorization is that the "protective effect" of perceived discrimination may be stronger for those Black Americans who have a stronger racial identity, as this effect is thought to happen because Black Americans recognize their experience of discrimination as a manifestation of racism against their racial group. Indeed, I found that while overall perceived discrimination predicted reduced mortality risk among Black Americans, this pattern was only significant among those who indicated that they very closely identify with being a member of their racial group (N = 401), but not for those who indicated otherwise (e.g., somewhat closely, not very closely, or not at all closely, N = 166). This finding may indicate that

various components of resilience may be more strongly mobilized when adversity is perceived as group-based and thus "going beyond" each individual's personal attribute (Crocker & Major, 1989; van Zomeren et al., 2008). This analysis may shed light on why perceived discrimination had no comparable effect on White Americans in the present study. Unlike Black Americans, White Americans may be more likely to see discriminatory treatments they receive to be personal rather than systemic.

Our findings are in line with prior evidence that constructive (i.e., problem-focused rather than emotion-focused) coping (Carver et al., 1989; Jorgensen & Thibodeau, 2007; Kaholokula et al., 2017), purpose in life (Hill & Turiano, 2014; McKnight & Kashdan, 2009), and educational attainment (Hummer & Hernandez, 2013; Zajacova & Lawrence, 2018) are all related to better health outcomes. I further showed that the perception of discrimination among Black Americans is positively linked to all these three components of resilience. These findings contribute to the emerging literature on resilience among Black Americans. So far, this work has examined various cultural processes, such as spirituality, religiosity, racial socialization, and racial identity (Christian & Barbarin, 2001; Miller & MacIntosh, 1999; Sellers & Shelton, 2003). We may expect that perceived discrimination is also linked positively with spirituality, religiosity, and other sources of resilience.

Some limitations of our work must be acknowledged. First, the connection between perceived discrimination and the three components of resilience was based on cross-sectional correlations. Future work must use longitudinal designs to test whether perceived discrimination, especially group-based, would increase each of these components of resilience among marginalized group members. Second, the current work is constrained by the resilience measures available in the dataset. There may be other facets of resilience among Black Americans,

including religiosity and racial socialization, that serve as protective factors for health. Future work must address this possibility. Third, because of missing data on the cause of mortality, the current work focused on all-cause mortality rather than mortality only associated with disease processes. Future work should replicate the results of the current study using deceased cases with different internal causes. Fourth, our Black sample came largely from a single city – Milwaukee, WI, the city that contains a high proportion of Black and is has high racial residential segregation (Frey & Myers, 2005). It is possible that segregation offers protection against mortality because of the high social cohesion observed in segregated minority community (Nuru-Jeter & LaVeist, 2011). Future work must address the generalizability of the present findings in other Black populations.

Table 3-3 Demographic information of the participants of the four waves of MIDUS data

	M1	M2	M3	MR
White Americans	N = 5648	N = 4345	N = 2835	N = 2810
Age	47.3 (12.9)	55.6 (12.5)	63.8 (11.4)	51.6 (14.4)
Sex (M/F)	2704/2944	2051/2294	1283/1552	1411/1399
Education	6.9	7.2	7.5	7.9
Income	75295.79	42552.09	57171.08	54717.05
Black Americans	N = 331	N = 834	N = 515	N = 797
Age	44.3 (12.5)	52.3 (12.0)	61.3 (10.6)	44.5 (12.4)
Sex (M/F)	125/206	312/522	178/337	321/476
Education	6.2	5.9	6.2	6.4
Income	49826.92	27245.8	34238.47	28482.62

Note. Because personal income was not directly assessed in M1 sample, we used the total household income instead, hence the larger number. The numbers in parathesis indicate standard deviation.

Table 3-4 Risk of mortality as a function of perceived lifetime discrimination among White and Black Americans

	Dependent variable:			
	Risk of mortality			
	Whites	Whites	Blacks	Blacks
	(1)	(2)	(3)	(4)
Discrimination	0.083***	0.045	−0.086**	−0.083**
	(0.032)	(0.033)	(0.035)	(0.036)
Age	0.062***	0.052***	0.046***	0.044***
	(0.004)	(0.004)	(0.007)	(0.007)
Sex	−0.293***	−0.395***	−0.610***	−0.697***
	(0.080)	(0.089)	(0.164)	(0.188)
Income	−0.00000***	−0.00000	−0.00001**	−0.00000
	(0.00000)	(0.00000)	(0.00000)	(0.00000)
BMI		−0.024***		−0.019*
		(0.007)		(0.011)
Alcohol usage		0.018		−0.021
		(0.025)		(0.053)
Smoking status		−0.213**		0.062
		(0.083)		(0.182)
Depressive tendencies		0.046*		0.004
		(0.024)		(0.053)
Anxiety		−0.019		0.066
		(0.059)		(0.065)
Function limitations		0.445***		0.331***
		(0.047)		(0.086)
Observations	2,822	2,691	590	576
R^2	0.107	0.137	0.108	0.135
Max. Possible R^2	0.959	0.956	0.939	0.938
Note:				*p<0.1; **p<0.05; ***p<0.01

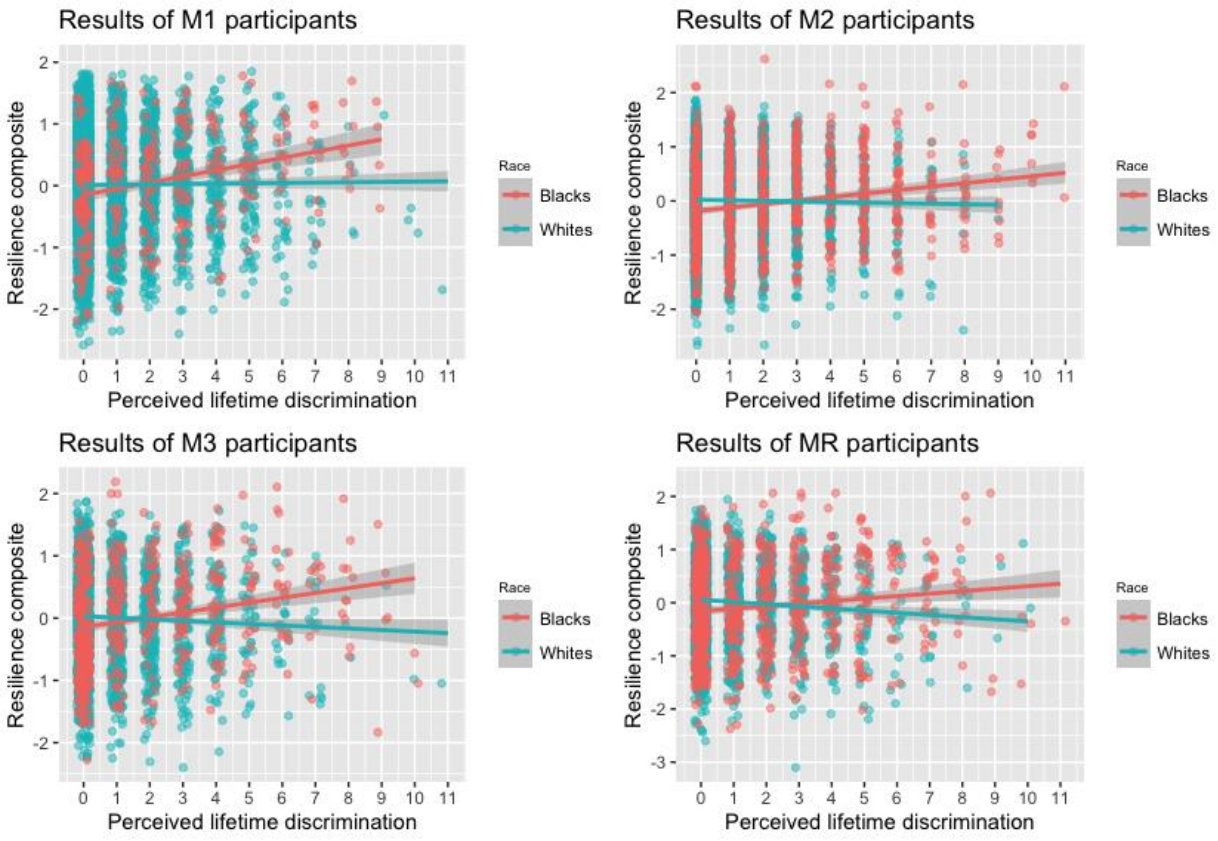

Figure 3-2 Effect of perceived lifetime discrimination on the composite score of resilience across four MIDUS study samples.

Note. Because coping was not measured in M1, the composite score combined only purpose in life and educational attainment in M1.

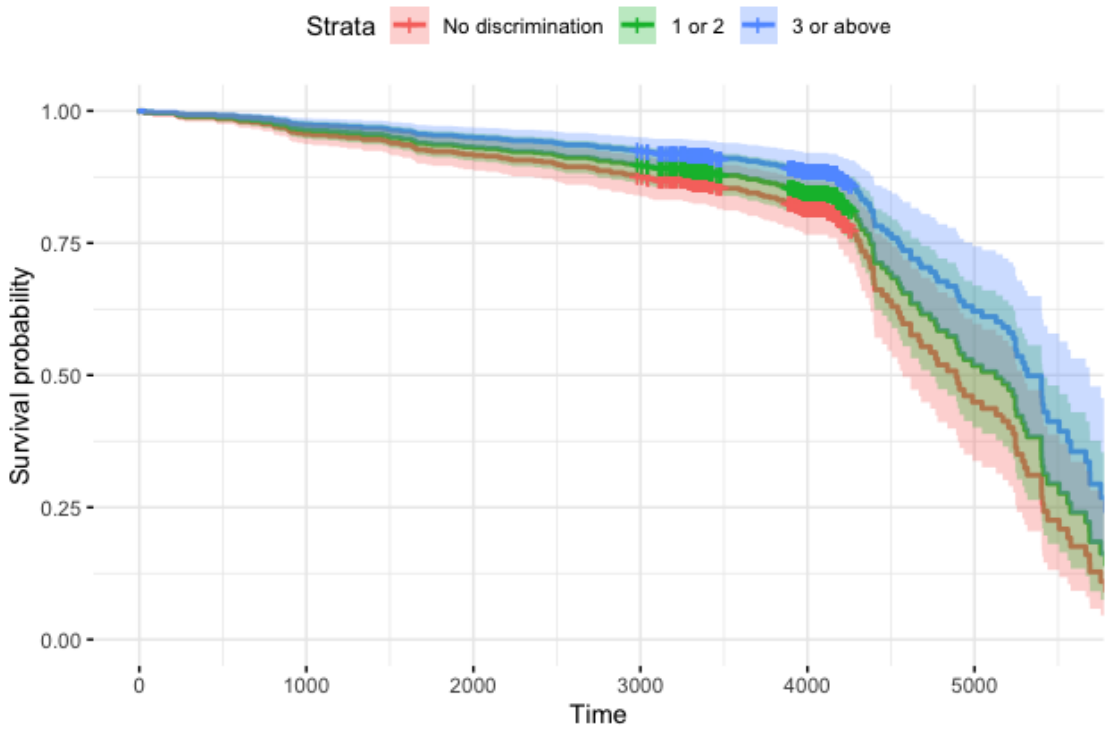

Figure 3-3 Estimated survival probability and its 95% CI as a function of time (number of days) among Black Americans with different levels of perceived lifetime discrimination.

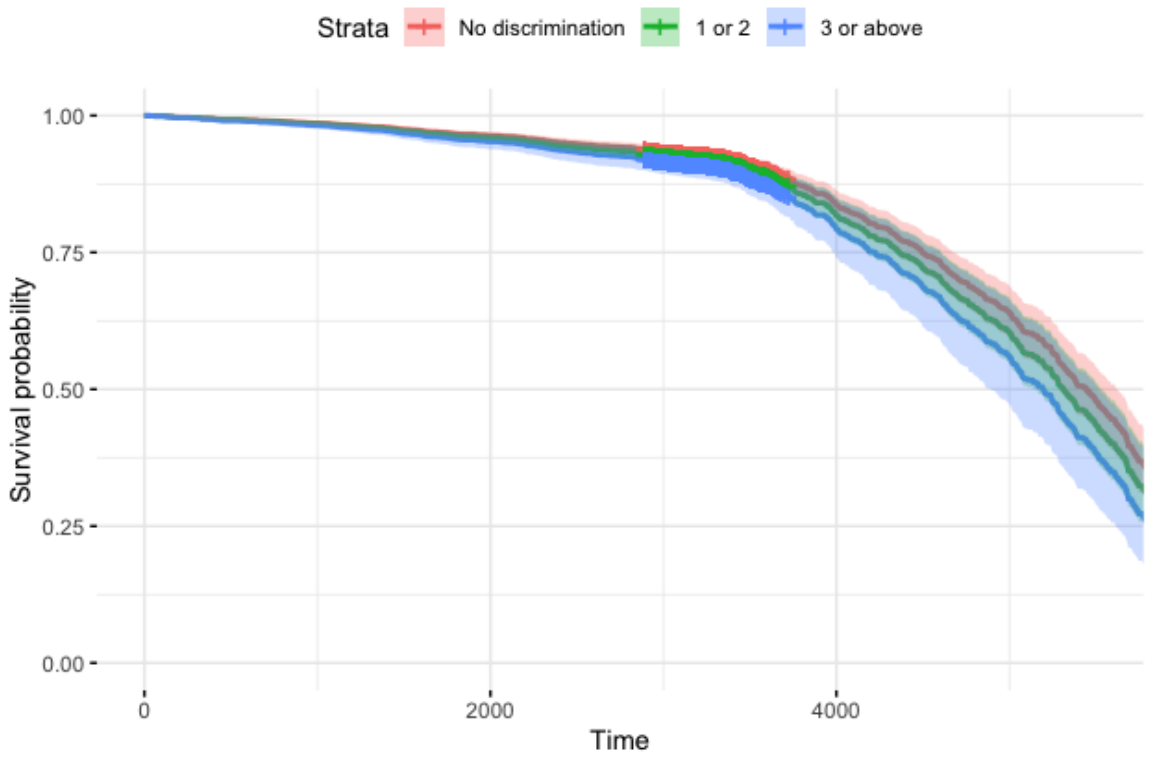

Figure 3-4 Estimated survival probability and its 95% CI as a function of time (number of days) among White Americans with different levels of perceived lifetime discrimination.

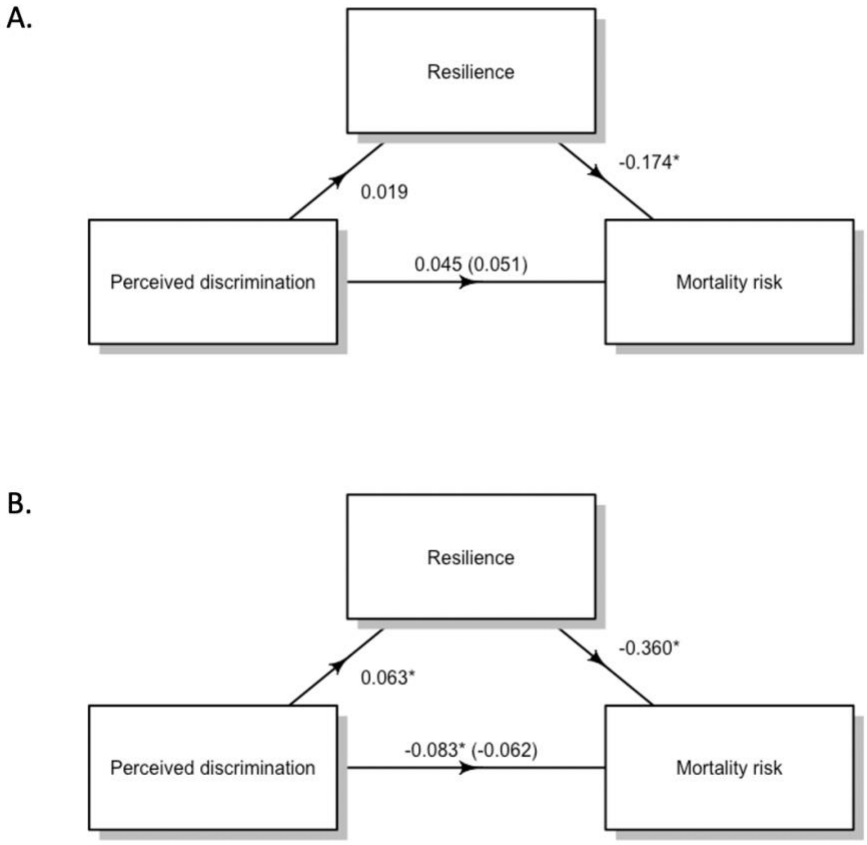

Figure 3-5 Mediation graph depicting the mediating role of resilience in the relationship between perceived discrimination and morality risk, among White Americans (A) and Black Americans (B). The coefficients are based on the model including all covariates.

Chapter 4 Biological Health and Functional Connectivity of the Brain

The first chapter of this dissertation has investigated how sociocultural environment might shape the human brain, and the second chapter of this dissertation has pursued the impact of such processes on the bodily health. In this chapter, I aim to integrate the two lines of work by exploring the interaction between the brain and the body. Specially, I examine how biological health is associated with functional organization of the brain.

The bidirectional link between the nervous system (e.g., the brain) and the non-nervous structures (e.g., the body) of organisms is one of the central themes in decades of research in neuroscience and psychology (Chiel & Beer, 1997; Clark, 1998). It is commonly known that the brain governs various aspects of the body, including both voluntary movements and involuntary regulative functions that are critical for maintaining a healthy state (e.g., homeostasis) of the body. Conversely, it has also been well-established that that the biological health of the body profoundly influences the functioning of the brain (Åberg et al., 2009; Hillman et al., 2008; Juster et al., 2010). Prior work has shown that physiological processes in the body, such as systemic inflammation, are tightly linked to patterns of functional activation and functional connectivity of various brain regions (Kraynak et al., 2018; Marsland et al., 2017; Nusslock et al., 2019). At present, however, little is known about whether biological health of the body also influences functional organization of the brain on a broader, whole-brain level. The present study aims to fill this gap by examining if the biological health of the body is linked to patterns of functional integration and segregation of the whole-brain network.

Biological Health and Functional Connectivity of the Brain

The bidirectional communication between the body and the brain forms the basis for adaptive physiological and psychological processes such as stress coping, pathogen defense, and homeostasis maintenance (Ben-Shaanan et al., 2016; Russo & McGavern, 2015; Segerstrom & Miller, 2004). The brain coordinates with the body via the nervous, neuroimmune, and neuroendocrine pathways in response to various events in the external environments (Dantzer et al., 2000; Pavlov & Tracey, 2017; Quan & Banks, 2007). When these signaling pathways become dysregulated due to the malfunctioning of the biological system of the body, it may also result in disrupted functioning of the brain (Gianaros & Wager, 2015; Weaver et al., 2002). Indeed, prior work has shown that systemic inflammation and cardiovascular malfunctioning are intricately involved in the pathogenesis of neurological and psychiatric disorders, such as Alzheimer's disease, Parkinson's disease, and major depressive disorder (Dantzer et al., 2008; Ferrari & Tarelli, 2011; Glass et al., 2010).

Several correlational and experimental neuroimaging studies have further linked indicators of biological health (e.g., systemic inflammation) to distinctive patterns of functional activation and connectivity of the brain. For instance, it has been shown that chronic low-grade systemic inflammation, quantified by the level of serum interleukin-6 (IL6) or C-reactive protein (CRP), is linked to altered functional activation of several brain regions including the brain stem, midbrain, limbic system and basal ganglia, as well as cortical regions including the anterior cingulate and prefrontal cortex (Harrison, Brydon, Walker, Gray, Steptoe, & Critchley, 2009; Harrison, Brydon, Walker, Gray, Steptoe, Dolan, et al., 2009; Inagaki et al., 2012; Thayer & Sternberg, 2010). Systemic inflammation has also been linked to altered functional connectivity of the executive, default mode, attention, and cortiostriatal network (Felger et al., 2016; Kraynak

et al., 2018; Marsland et al., 2017; Nusslock et al., 2019). Similar evidence has also emerged from experimental studies, where subjects received injection of low-dose endotoxin that induces transient peripheral inflammatory responses. Such manipulation has been found to modulate activations of brain regions underlying various cognitive and affective functions, such as memory, social cognition, and reward processing (Eisenberger et al., 2009; Harrison et al., 2014; Kullmann et al., 2014).

Does Biological Health Influence the Whole-Brain Network?

So far, risk factors of biological health have only been examined with respect to the functional profile of separate brain regions or local networks. The optimal functioning of the brain, however, requires a well-structured organization of those regions and networks across the whole brain (Bullmore & Sporns, 2009, 2012; Rubinov & Sporns, 2010). Recent advances in network neuroscience have revealed that one fundamental feature of the functional organization of the human brain is its *modularity* (Bertolero et al., 2018; He et al., 2009; Newman, 2006). The brain can be partitioned into several distinct communities, or "modules", where intra-module connections are dense and inter-module connections are relatively sparse. Such modular organization leads to *segregation* of different networks where each network specializes in carrying out a distinctive set of functions, while at the same time it preserves *integration* of those networks so information can flow efficiently and be synthesized (J. R. Cohen & D'Esposito, 2016; Meunier et al., 2010; Rubinov & Sporns, 2010). It thus is typically considered as a critical marker of a "healthy" brain, because it ensures efficient information transfer across the whole brain without incurring substantial wiring cost of the connections from a metabolic sense (Laughlin et al., 1998; Lord et al., 2013; Tosh & McNally, 2015).

Prior work has reliably shown that modularity of the functional brain network is strongly associated with cognitive capacities. It predicts not only the general intelligence (Hilger et al., 2017), but also performances in multiple cognitive domains, such as working memory, executive function, and language (Baum et al., 2017; J. R. Cohen & D'Esposito, 2016; E. S. Duncan & Small, 2016). Moreover, conditions that are known to disrupt certain cognitive functions, such as aging and psychiatric disorders, are usually associated with reduced modularity of the whole brain functional network (Contreras et al., 2019; Geerligs et al., 2015; Ye et al., 2015).

The Present Study

The present study thus examined if biological health, measured using various inflammatory and cardiovascular indicators (Kitayama et al., 2015, 2018; Kitayama & Park, 2020), is associated with the organization of the functional brain network. In particular, I applied graph theory analysis on the resting-state fMRI data. I focused on *modularity*, which as mentioned above, quantifies the degree to which the brain can be separated into different modules (Newman, 2006). To explain the potential variation in modularity, I further test the *participation coefficient*, which quantifies the degree of inter-module connections. I examine which module may show increased participation coefficient, thus explains the variation of modularity as a function of health. In addition, given the importance of modularity for cognition, I tested if modularity is reliably linked to cognitive performance among the current participants.

Method

Participants

Participants were drawn from the Midlife in the United States (MIDUS) project, a national longitudinal study on health and well-being (http://www.midus.wisc.edu). In particular, the participants of the present study were from the MIDUS "Refresher" study, which was

designed to replenish the original MIDUS baseline cohort. The "Refresher" study tested a national probability sample of 3,577 participants, as well as an additional 508 African Americans from Milwaukee, WI, who were recruited to increase the number of racial minorities in the broader MIDUS project. One hundred thirty-eight participants from the "Refresher" study later participated in the "Refresher" Neuroscience study, in which they completed various cognitive and emotional tasks and underwent psychophysiological assessments and MRI scanning of the brain. These participants also underwent comprehensive biological assessments during the same session as part of the "Refresher" Biomarker study. Among the 138 participants, 11 were not eligible for MRI scanning due to failure to meet inclusion criteria (e.g., no history of neurological disorders, no magnetic metal or medical devices in the body, no claustrophobia, ability to lie down on one's back for two hours). For the remaining 127 participants, one was excluded due to left-handedness, two were excluded due to missing data from the biological assessments, and six were excluded due to missing resting-state fMRI scan. Among the remaining 118 participants (64 females), 80 were from the national probability sample, and 38 were from the Milwaukee African American sample. The average age was 48.23 years with ages ranging between 26 and 76 years. There were 74 White Americans, 38 Black Americans, 2 Native Americans, 1 Asian American, and 3 of other races. All participants provided informed consent before the study procedures. Data and brain images are publicly available from the MIDUS website upon request.

Procedures

Participants were tested at one of the three sites: University of California, Los Angeles; University of Wisconsin; or Georgetown University. They first completed the "Refresher" Biomarker study, which was an overnight session where they provided blood, saliva, and urine

samples for biological assessments, as well as completed a few questionnaires assessing psychosocial experience. On the second day following the biomarker collection, they completed the "Refresher" neuroscience study, where they underwent psychophysiological assessments, cognitive test, and the MRI scanning.

Biomarkers

Following prior work (Kitayama et al., 2015, 2018; Kitayama & Park, 2020), I focused on four biomarkers to quantify biological health. Two biomarkers were used to index inflammation (i.e., IL-6, CRP), and two were used to index cardiovascular malfunction (i.e., systolic blood pressure [SBP], ratio of total-to-high-density lipoprotein cholesterol [RTHDL]). IL-6 and CRP were measured from blood serum. Serum IL-6 levels were measured using high-sensitivity enzyme-linked immunosorbent assay (ELISA; Quantikine, R&D Systems, Minneapolis, MN), and serum CRP levels were determined using the BNII nephelometer from Dade Behring utilizing a particle enhanced immunonephelometric assay. Blood pressure was measured by clinical nurses three times in a seated position allowing a maximum of 30 seconds between each measurement. The two most similar SBP recordings were then averaged. Lastly, the cholesterol assays were performed using a Roche Cobas analyzer (Roche Diagnostics, Indianapolis, IN).

To reduce the influence of potential outliers, I winsorized extreme values of the four biomarkers at three standard deviations above or below the mean (IL-6: n = 2, CRP: n = 2, RTHDL: n = 1, SBP: n = 0). Because IL-6, CRP, and RTHDL were positively skewed, they were nature log transformed. Following the procedures adopted in prior work (Kitayama et al., 2015, 2018; Kitayama & Park, 2020), I performed the principle component analysis (PCA) and found that the four biomarkers loaded onto a single factor, which accounted for 46.2% of variance.

Therefore, further analysis was performed using the factor score obtained from the PCA, supplemented by analysis on each biomarker separately.

Image Acquisition

MRI data of the present study were acquired on a 3 T scanner (MR750 GE Healthcare, Waukesha, WI) using an 8-channel head coil. The blood oxygen level-dependent (BOLD) signal during the 8 min resting-state was acquired using a T2*-weighted gradient-echo echo-planar imaging (EPI) pulse sequence (240 volumes, TR = 2000 ms, TE = 20 ms, flip angle = 60 degrees, field of view = 220 mm, 96 x 64 matrix, 3 mm slice thickness with 1 mm gap, 40 interleaved sagittal slices, and ASSET parallel imaging with an acceleration factor of 2). A T1-weighted anatomical image was acquired using a three-dimensional magnetization-prepared rapid gradient-echo sequence (TR = 8.2 ms, TE = 3.2 ms, flip angle = 12 degrees, field of view = 256 mm, 256 x 256 matrix, 160 1mm axial slices per volume, inversion time = 450 ms). The scanning session also acquired field map images, diffusion-weighted data. and perfusion data.

Image Preprocesssing

The preprocessing and analysis of the resting-state fMRI data were carried out in the Connectivity Toolbox (CONN; Whitfield-Gabrieli & Nieto-Castanon, 2012). The preprocessing steps were implemented with SPM12 (Wellcome Department of Cognitive Neurology, London). The initial four volumes were first removed to only retain data acquired after the scanner stabilized. Functional data were then realigned, where all volumes were coregistered and resampled to the first volume. Detection of outlier scans was then performed based on variations in the observed global BOLD signal and the amount of head motion in the scanner (see more details below). Functional and anatomical data were normalized into the standard MNI space and segmented into different tissue classes: gray matter (GM), white matter (WM), and cerebrospinal

fluid (CSF). Functional data were resampled to 2 mm isotropic voxels and anatomical data were resampled to 1 mm isotropic voxels. Lastly, functional data were smoothed using a Gaussian kernel with 8 mm full-width at half-maximum.

Detection of outlier scans was carried out using Artifact Detection Tools (ART; www.nitrc.org/projects/artifact_detect/). For each subject, a volume was identified as an outlier if (a) framewise displacement was greater than 0.2 mm in the composite motion involving three translational and three rotational displacements, and (b) variation of global mean signal was greater than 3 SD. These thresholds were conservative because previous studies have shown that even tiny subject motion (0.5 mm) in the scanner can have pervasive impact on resting-state fMRI data (Power et al., 2012, 2014). Subjects were excluded from further analysis if more than 50% of their data (118 volumes) were identified as outliers. This left us with 74 subjects, with an average of 197 clean volumes.

The functional data were further denoised using linear regression and the anatomical component-based noise correction procedure (Behzadi et al., 2007; Muschelli et al., 2014), implemented in CONN. In particular, five principal components were extracted from the signals of the cerebral WM and CSF segments of each subject. These principal components were entered into the linear regression of the functional time series, together with the 12 motion parameters (three translational and rotational displacements, as well as their first-order temporal derivatives), regressors for the outlier scans (coded as "1" for the outlier scan and "0"s for the rest; number of regressors is the same as number of outlier scans), and a session-onset regressor, which is a constant and linear BOLD signal trend convolved with a canonical hemodynamic response function. The residual functional time series were further band-pass filtered (0.008 Hz $< f <$ 0.09 Hz) to retain slow-frequency fluctuations.

Functional Connectivity Analysis

I then carried out the whole-brain functional connectivity analysis. I adopted a commonly used functional atlas that consists of 264 coordinates (Power et al., 2011), and I constructed a 5 mm radius sphere around each coordinate, resulting in 264 functional ROIs. The ROIs were examined for their overlap with the functional data of the current sample, following a similar procedure from previous studies (Geerligs et al., 2015; Iordan et al., 2018). In particular, for each subject, I calculated a mean functional volume that is free from the motion artifacts, using ART, and I binarized this mean functional volume, thresholded at > 50% mean signal intensity. A sample-level mask was then created by taking the logical conjunction of the mean functional volumes of all the subjects. ROIs that had less than 25 voxels (about 50% of their volume) overlapping with this sample-level mask were excluded from the analysis. Towards this end, 243 ROIs were retained.

To carry out graph theory analysis at the whole-brain level, the whole-brain network was constructed by taking the Pearson correlation coefficients between the BOLD time series of all pairs of functional ROIs, followed by Fisher r-to-z transformation, resulting in a 243 x 243 undirected connectivity matrix. The diagonal of the connectivity matrix was set to zero.

Before calculating graph theory parameters (e.g., modularity) of the whole-brain network, the connectivity matrix of each subject was thresholded so as to exclude false-positive connections (edges) (Garrison et al., 2015; Zalesky et al., 2016). In particular, I used density-based thresholding, which preserved the same number of edges across subjects. Calculations of graph theory parameters were performed across a range of thresholds, so the results were not dependent on a particular threshold. The connectivity matrix was thresholded to retain 2% to 10% of the strongest connections, with 1% increments (the network was severely fragmented at

1% of strongest connections, and thus was not used). This range is consistent with that used by Power et al. (2011) in generating the functional atlas, as well as the range used in several other studies (Geerligs et al., 2015; Iordan et al., 2018). There were on average 56, 32, 19, 11, 7, 4, 2, 1, and 1 disconnected node(s) at the 2% to 10% threshold, respectively. At each threshold, the connectivity matrix of each subject was binarized such that edges passed the threshold were coded as 1 and those did not were coded as 0. Graph theory parameters were calculated using the binarized network at each threshold and then averaged across the thresholds.

Modularity. I calculated the modularity index (Q) to quantify the degree of which the whole-brain network can be cleanly partitioned into different modules, or sub-network. Networks with high modularity have dense within-module connections yet sparse inter-module connections. The modularity index is arithmetically defined as follows:

$$Q = \frac{1}{2m} \sum_{ij} [A_{ij} - \gamma \frac{k_i k_j}{2m}] \delta(c_i, c_j)$$

where m is the number of edges of the network, A is the connectivity matrix, i and j are nodes of the network, γ is the resolution parameter (set at 1 in the present study), k_i and k_j are the number of connections of node i and j in a random network, and δ is an indicator that equals to 1 if i and j are assigned to the same module or 0 if otherwise. I calculated the modularity index for the whole-brain network of each subject by running the Louvain community detection algorithm 500 times with iterative community fine-tuning, and then averaged the score across the runs (Blondel et al., 2008; Sun et al., 2009). The modularity score was then transformed to reduce skewness and standardized before further analysis.

Group-level community detection. To explain the variation in modularity as a function of the levels of biomarkers, I performed community detection to identify the sample-level community structure (i.e., partitioning the nodes into distinct communities, or modules). This

community structure was used for further analyses at the level of the module, as well as for displaying the results. I used the Louvain algorithm together with consensus clustering to avoid the degeneracy issue (i.e., non-deterministic solution of the partitioning) of the Louvain algorithm (Blondel et al., 2008; Lancichinetti & Fortunato, 2012). The algorithm was applied at the initial weighted connectivity matrix before the thresholding (but with only positive edges) to obtain threshold-independent community structure. I first performed consensus clustering for each subject to achieve a robust partition at the individual-level, and then the individual-level partitions were used to identify group-level consensus partition. Specifically, for each subject, the Louvain algorithm was run 500 times with iterative community fine-tuning on the connectivity matrix. The resulting 500 partitions were used to construct an agreement matrix, indexing for every pair of nodes, the proportion of the 500 runs that they were assigned to the same module. The agreement matrix was then thresholded at above 50%, and the consensus partition was generated by running the Louvain algorithm (500 runs) on the agreement matrix. This step was iteratively applied (agreement matrix recalculated and rethresholded) until a single representative partition was generated. The group-level consensus partition was then generated by calculating an agreement matrix of all individual-level partition, and the Louvain algorithm was used to generate consensus partition based on this agreement matrix, with the same procedure described above.

Participation coefficients. To assess the degree of inter-module connectivity of different nodes and modules which leads to the reduced modularity, the participation coefficient was calculated (Guimerà & Amaral, 2005). It quantifies the extent to which connections of a node are widely distributed across different modules as opposed to concentrated within its own module. Participation coefficient of a node *i* is arithmetically defined as follows:

$$P(i) = 1 - \sum_{s=1}^{N_m} \left(\frac{k_i(s)}{k_i} \right)^2$$

where *s* is the current module, N_m is the total number of modules in the network, $k_i(s)$ is the number of connections node *i* has with nodes in module *s*, and *ki* is the degree (total number of connections) of node *i*. The participation coefficient of a module was calculated by averaging the participation coefficient of all nodes within the module.

Control Variables

I included in the present investigation several covariates that are known to influence the level of inflammation and cardiovascular risk of the body (O'Connor et al., 2009) or to modulate functional connectivity. First, several demographic variables were controlled for, which included age, gender, and race. Race was coded as a set of dummy variable with the White group as the reference group. I also controlled for educational attainment. Educational attainment was coded as an ordinal variable, ranging from 1 (*eighth grade, junior high school*) to 12 (*PhD, or other professional degree*). In addition, I controlled for several health-related variables, which included alcohol consumption, smoking status, medication, and waist-to-hip ratio (WHR). Alcohol consumption was measured as the average number of alcohol drinks consumed per week. Smoking status was coded as a categorical variable with 1 being current smoker, and 0 being non-smoker. Medication use was coded as a categorical variable with 1 being currently on medications that are known to alter the level of biomarkers (e.g., antihypertensive, antilipidemic, corticosteroid), and 0 being not on those medications. WHR was used to control for variations in visceral adiposity. It was measured by clinical nurses.

Cognitive Tests

Participants in the present study also completed a series of cognitive tests from the Cambridge Neuropsychological Test Automated Battery (CANTAB), which includes assessment of attention, psychomotor speed, executive function, emotion and social cognition. Because prior work has shown that modularity is associated with overall cognitive capacities, I tested this association in the present study. Details of the procedures and scoring cognitive tests can be found in the Appendix C. In brief, across the seven cognitive tasks that were administered, I standardized and averaged the level of performance to create an index of the general cognitive capacity.

Statistical Analysis

The relationship between the level of biomarkers and the graph-theory parameters (e.g., modularity, participation coefficient) of the functional brain network was examined using multiple regression. I first tested the relationship without covariates (base model), followed by controlling for the demographic variables (age, sex, race, and education) and then the health-related variables (alcohol consumption, smoking status, medication use, and WHR).

I first examined the parameter at the whole-brain level – modularity. Next, I tested the parameter at the node level – participation coefficient. The participant coefficient was aggregated across nodes within each of the identified modules to form the module-level parameter. Because five modules were identified among the current sample, I used Bonferroni correction to counteract the issue of multiple-comparison. The threshold of significance was thus set to $p < .01$ for the module-level analysis.

In addition, the relationship between modularity and cognitive performance was tested. The same stepwise multiple regression model was fitted. Because I have predicted that the level of biomarkers may predict modularity, which in turn predicts cognitive performance, I also

tested whether modularity may mediate the link between the level of biomarkers and cognitive performance. The mediation was examined using the "mediate" function from the *psych* package in R, with 5000 bootstrap resamples.

Results

Community Structure

The group-level consensus partition reveals five modules (see Fig. 4.1): the frontoparietal control network (FPCN), the default-mode network (DMN), the cingulo-opercular/salience network (COS), the sensorimotor network (SMN), and the visual network (VN). This community structure is consistent with the results of community detection by prior work (Geerligs et al., 2015; Iordan et al., 2018; Power et al., 2011). There were 37, 66, 47, 51, and 40 nodes within the FPCN, DMN, COS, SMN, and VN, respectively.

Biological Health and Modularity

I found that elevated level of the biomarkers predicted reduced modularity of the whole-brain network, b = -.358, 95% CI = [-.577, -.139], $t(72)$ = -3.252, p = .002. This relationship remained unchanged controlling for demographic variable, b = -.234, 95% CI = [-.459, -.010], $t(65)$ = -2.083, p = .041; and it remained unchanged when additionally controlling for health-related variables, b = -.269, 95% CI = [-.504, -.035], $t(61)$ = -2.295, p = .025 (Fig. 4.2).

There were also some notable effects of the covariates. First, consistent with previous literature on the relationship between aging and the modularity of the brain, age predicted reduced modularity, b = -.001, $t(61)$ = -2.896, p = .005. Second, educational attainment predicted increased modularity of the whole-brain network, b = .003, $t(61)$ = 2.432, p = .018, consistent with previous finding that modularity is positively linked to intellectual performance.

Participation Coefficient

Across the five modules, there was no significant relationship between the level of biomarkers and the participation coefficient of the FPCN, COS, SMN, and DMN. However, the level of biomarkers was positively correlated with the participation coefficient of the VN (Fig. 4.3). The relationship held in the base model, b = .008, 95% CI = [.003, .013], $t(72) = 3.077$, p = .003; after controlling for demographic variables, b = .009, 95% CI = [.004, .015], $t(65) = 3.247$, p = .002; and after controlling for all covariates, b = .009, 95% CI = [.003, .015], $t(61) = 2.942$, p = .005. This suggests that the reduced modularity linked to heightened level of biomarkers were likely due to the increased inter-module connectivity of the visual network.

Modularity and Cognitive Performance

The level of cognitive performance was significantly associated with modularity in the base model, b = .207, 95% CI = [.072, .341], $t(72) = 3.068$, p = .003. This relationship was attenuated when controlling for demographic variables, b = .081, 95% CI = [-.045, .207], $t(65) = 1.279$, p = .205, but it was significant when all covariates were included, b = .131, 95% CI = [.005, .257], $t(61) = 2.081$, p = .042 (Fig. 4.4).

The mediation effect (biological health → modularity → cognitive performance) was then tested. As mentioned above, biological health was negatively associated with modularity (path 'a'), b = -.36, $t(72) = -3.25$, p = .002. Modularity was positively associated with cognitive performance controlling for biological health (path 'b'), b = .18, $t(71) = 2.46$, p = .016. Biological health was negatively associated with cognitive performance, b = -.15, $t(72) = 2.09$, p = .040, but the effect disappeared when modularity was added to the model, b = -.08, $t(71) = -1.14$, p = .260. The mediation effect (biological health → modularity → cognitive performance) was statistically significant, b = -0.064, 95% CI = [-0.136, -0.008]. However, it should be noted

that this mediation effect was no longer significant when the effect of covariates was regressed out, b = -0.034, 95% CI = [-0.094, 0.002].

Discussion

The aim of the present study was to investigate whether biological health of the body is associated with functional connectivity of the whole-brain network. Towards this end, I found that poor biological health is linked to reduced modularity, a fundamental feature of the brain network that describes its cost-efficiency (Newman, 2006). This reduction in modularity is accompanied by a higher participation coefficient of the visual network, suggesting that brain regions of the visual network show increased connections with regions of other functional networks. Moreover, consistent with prior work documenting the importance of modularity for an optimally functioning brain (Bertolero et al., 2018; J. R. Cohen & D'Esposito, 2016), I found that reduced modularity is related to worse cognitive performance. The reduction in modularity also mediates the relationship between biological health and cognitive performance. Caution is warranted in interpreting this mediation effect, since the mediation became non-significant when all covariates are added to the model, suggesting that there may exist other pathways that explain the debilitating effect of poor biological health on cognition.

The present study contributes to the aging literature which has shown that as one ages, the modularity of the brain tends to go down (Geerligs et al., 2015). The reduction in modularity might be one of the reasons of the cognitive decline in certain domains commonly observed through both healthy and pathological aging. However, this literature has solely focused on the effect of age itself and has ignored the physiological effect of adversities and stressors that could accumulate over the life course on one's body. The present study demonstrates that biological health, which could be an indicator of stresses accumulated in the body (Cole, 2014), also leads

to compromised brain functioning. It is possible that biological health in part accounts for the effect of aging on brain functioning, as health typically worsens as one ages. Nevertheless, it should be noted that in the current data, both biological health and age predict modularity even after the two were simultaneously entered to the model. This suggests that biological and age each has unique effect on brain functioning.

The present finding is in line with earlier empirical evidence that an aging brain tends to show neural dedifferentiation and compensation (Reuter-Lorenz et al., 2000; Reuter-Lorenz & Cappell, 2008). For instance, when performing a same cognitive task, older brains are more likely to show bilateral activation when the activation in younger brains is typically lateralized (Reuter-Lorenz et al., 2000). Moreover, older brains also tend to show increased connectivity between brain regions from different functional networks (Geerligs, Maurits, et al., 2012). There is no consensus on whether these altered patterns of functional activation and connectivity represent adaptive compensatory mechanisms for cognition, or instead they signal emerging cognitive deficits. In the current data, the reduction in modularity was associated with worse cognitive performance, which may suggest that the reduced modularity linked to poor biological health signals functional disorganization of the brain rather than functional compensation. One reason this may be the case is that the reduced modularity in the current data is driven by increased inter-module connections from the visual network. The adaptive neural compensation observed in prior work is usually accompanied by increased connections from regions within the association cortex, such as certain prefrontal regions and parietal regions (Benson et al., 2018; Geerligs, Saliasi, et al., 2012). These regions by default have diverse connections across different modules (in healthy brain) and are responsible for functional integration in the brain (Gordon et al., 2018). Hence, it is possible that inter-module connections from the visual network

substantially increase the wiring cost of the brain and are not able to restore healthy brain functions. Future work should test this possibility.

Another contribution of the present study is to extend the growing literature of human social genomics and health (Cole, 2014). This line of work has documented that various adverse social-environment conditions, such as low socioeconomic status, social exclusion, and chronic stress, can mobilize various epigenetic changes in the body (Cole et al., 2011). These changes have been referred to as the conversed transcriptional response to adversity (CTRA), and they involve an increased expression of proinflammatory genes, and a decreased expression of genes coding innate antiviral responses. The CTRA gene expression in turn can undermine health by inducing inflammation-related chronic diseases such as diabetes and atherosclerosis (Cole, 2013). The present study moved one step further by showing that poor biological health (including systemic inflammation), the downstream consequence of stress and adversity, is also negatively associated with "brain health". This finding is consistent with the thesis that the biological system, including the brain and the body, is embedded in and intricately shaped by the social environment.

The present study largely adopts the perspective that better cognitive performance on a task is optimal and worse performance is defective, a common view of cognitive psychology. This perspective, however, has been challenged by an emerging view that cognitive capacities can be considered as "healthy" or optimal only within a specific environmental context, and that cognitive biases or deficits may sometimes be functional when considering the unique life history of the individual (Gigerenzer, 2008; Haselton et al., 2015). Hence, it may be problematic to examine a single composite index of overall cognitive capacities according to this perspective. One limitation of the present work is that the cognitive battery being used is relatively brief and

contains only one task for a specific cognitive domain. It thus does not enable a highly reliable assessment of each cognitive domain (though see Appendix C for an examination with each task as a function of modularity). This gap needs to be addressed by future work testing specific cognitive capacity while considering the different ecological constraints of the social context one is embedded in.

A few limitations of the present study point to future directions for this line of work. First, the current data is cross sectional. While it is reasonable to assume that poor biological health engenders various neuroimmune and neuroendocrine pathways that compromises the functioning of the brain, the reverse direction cannot be ruled out. It is possible that when the brain is not working in an optimal fashion, it may lead to poor biological health through mental disorders and engagement in unhealthy behaviors. Future work using longitudinal design will better elucidate the causal relationship. Such investigation will also provide informative insight into potential intervention to improve cognition. Prior work has already showed that physical exercise, while improving bodily health, is also beneficial for cognition (Erickson et al., 2011). It remains to be tested whether certain neurophysiological pathway is implicated in this process, and whether interventions on cardiovascular and inflammatory systems can directly influence cognition. Second, the present study only examined functional connectivity using the resting-state fMRI scan. Although modularity and other markers of the functional organization are reliable predictors of cognition when assessed at rest (Bertolero et al., 2018), they do not capture the unique state of the brain while performing different cognitive tasks. Future work should use task-based fMRI to test how biological health is related to different domains of cognition, and how this link is mediated by functional activation and connectivity of the brain. Third, future work should also use diffusion-weighted MRI to examine the *structural* connectivity of the

brain. This can inform whether biological health may be linked to the organization of brain networks at the more fundamental level.

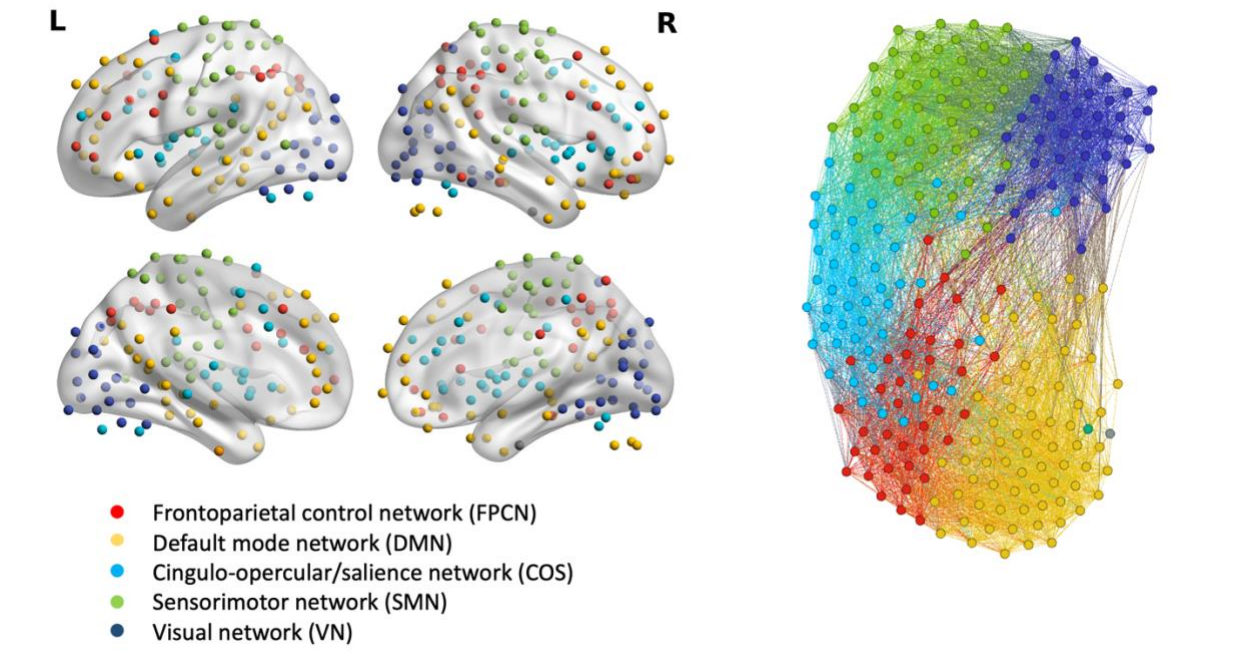

Figure 4-1 Community structure of the current sample derived from group-level consensus partitioning.

Nodes are presented on both the cortical surface (left), and in the force-directed graph (right). The force-directed graph was constructed using the 20% strongest connections. Nodes are color-coded by the assigned module (five in total), and within-module connections are in the same color as the module.

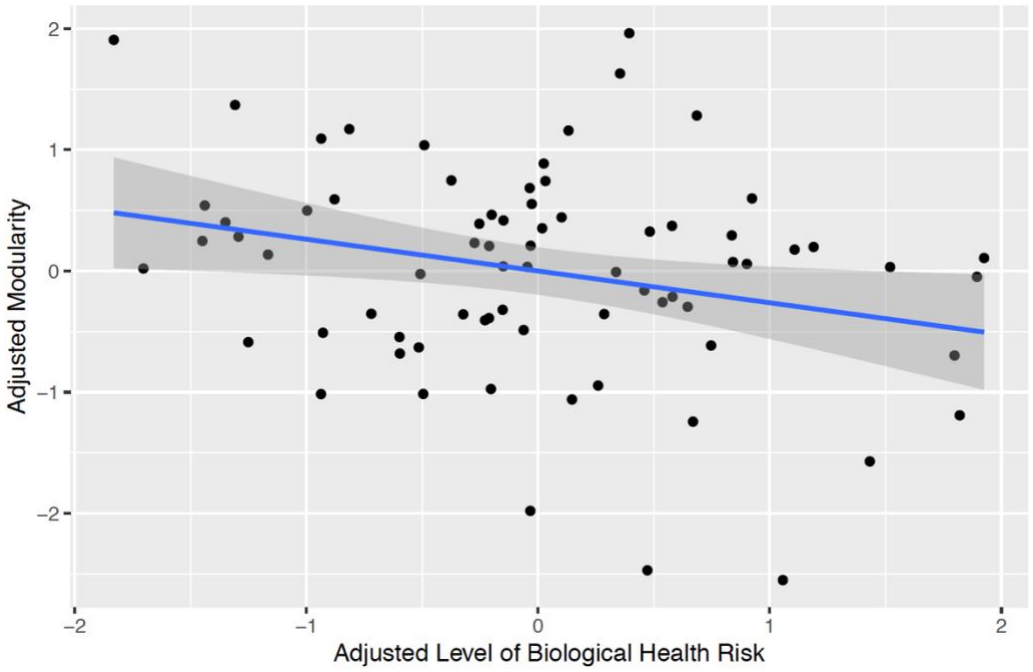

Figure 4-2 The correlation between the level of biomarkers and the modularity of the whole-brain network. Both variables were adjusted by regressing out the effects of the covariates.

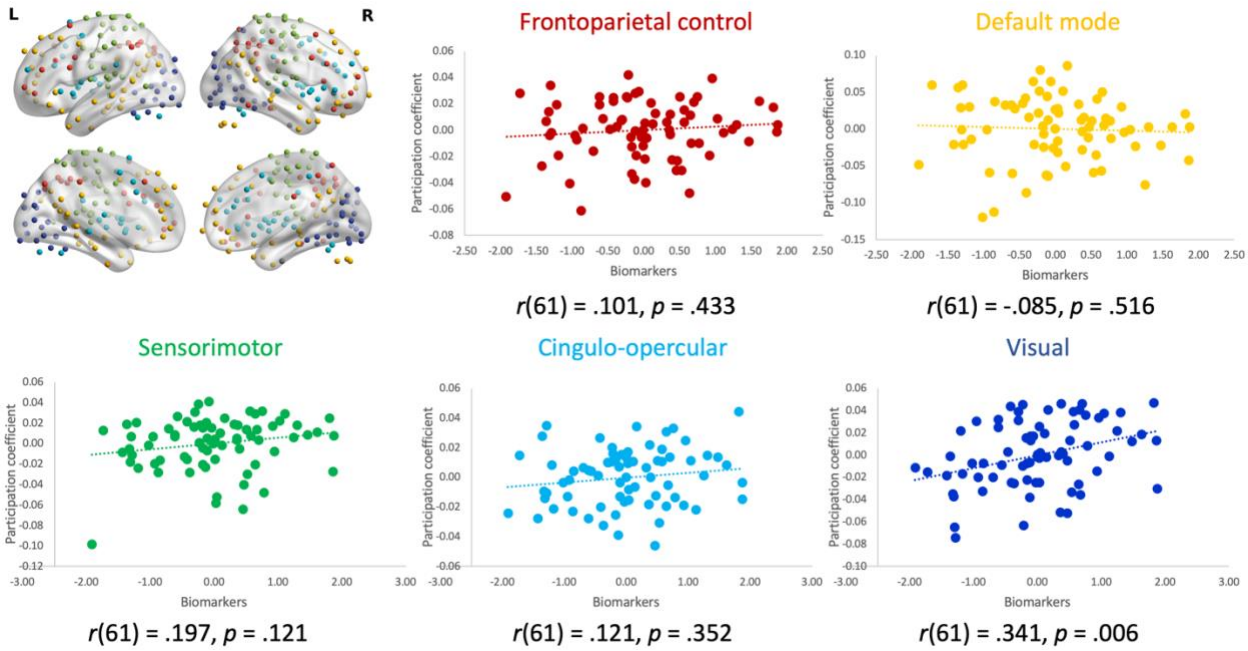

Figure 4-3 The relationship between the participation coefficient of each of the modules and the level of biomarkers.

Each scatterplot illustrates the relationship between the participation coefficient of the respective module (aggregated across all nodes within the module) and the level of biomarkers. In each plot, both variables were adjusted by regressing out the effects of the covariates.

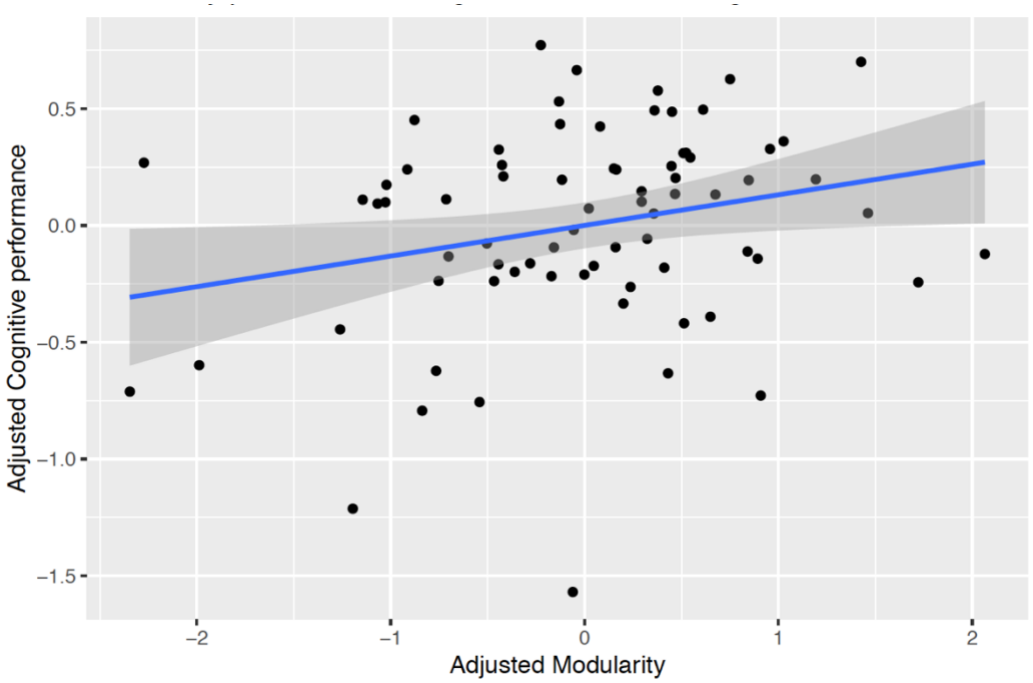

Figure 4-4 The correlation between modularity and the overall cognitive performance. Both variables were adjusted by regressing out the effects of the covariates.

Chapter 5 General Discussion

Social and behavioral scientists have long believed that the sociocultural environment profoundly shapes human behaviors. It is only recently, however, that the field has realized that such influences extend to various physiological and neural processes underlying behaviors. The mutual constitution of these biological processes and the sociocultural environment is most likely to be mediated by the plastic change of the body and brain as they interact with the sociocultural environment throughout human development. Hence, what were once thought of as two opposing forces of influence over behavioral and psychological processes – the "nature" (biological processes) and "nurture" (environment) – have turned out to be not completely separate and independent. In particular, recent work has shown that "nature can be nurtured" (Holliday et al., 1990; Kitayama & Yu, 2020; Yu et al., 2019). This dissertation built upon this emerging theoretical perspective and examined three different but related questions.

In chapter two, I took a cultural neuroscience approach and investigated how morphometrical (or structural) properties of the human brain can be shaped by culture. Importantly, instead of only testing cultural differences in brain structures, from which the direction of the effect would remain unknown, I took advantage of the "plasticity alleles" which would have the potential to inform causality. Building on prior work showing that individuals carrying the "plasticity alleles" are more sensitive to cultural influences and thus exhibit the more typical psychological profiles of the culture (Kitayama et al., 2014; Tompson et al., 2018), I further showed that individuals carrying the "plasticity alleles" also exhibit greater cultural

differences in GM volume of several cortical regions. This finding adds further evidence to the possibility that engagement with culture can change the structural aspects of the brain (Kitayama & Salvador, 2017). Moreover, I found that the GM volume of these regions are correlated with independence or interdependent self-construal, which suggests that cultural difference in brain structures is related to the fundamental way one perceives and construes the self. Taken together, this study contributes to the cultural psychology literature by providing another convincing example that human brain is inherently socioculturally patterned. Culture is thus "embrained".

In chapter three, I focus on the inequality aspect of sociocultural environment and its consequences for biological health. In the first study, I examined the growth trend in COVID-19 cases and deaths during the COVID-19 pandemic, and I tested which dimensions of structural inequality increased the growth of COVID-19 fatalities. Towards this end, I found that two structural factors, racial residential segregation and income disparity, were extremely relevant. When a given region is highly segregated (by race) and has a high degree of income inequality, there will exist large minority enclaves as well as poor White enclaves that are deprived of various socioeconomic and medical resources. Individuals from these communities will thus be more vulnerable to the virus. Indeed, I found that segregation and income inequality jointly predicted higher growth rates of cases and deaths at the early stage of the pandemic in large U.S cities. In the second study of this chapter, I examined how subjective perception of racial discrimination is associated with health. While the experience of racial discrimination in and by itself is stressful (which is likely reflected in poor subjective health), I found that the reporting of discrimination could also reflect resilience among Black Americans against systemic racism. This resilience may entail more constructive coping efforts, greater purpose in life to deal with adversities, and greater aspiration to pursue higher education. The resilience, in turn, predicted

reduced mortality risk among Black Americans. This study suggests that individuals are not passive recipients of discrimination, and it is important to adequately consider how individuals interpret, make meaning of, and cope with the experience of discrimination.

Lastly, in chapter four, I examined the interconnections between brain and biological health. I found that biological health is associated with the fundamental fashion of how the human brain is functionally organized. In particular, poor biological health is related to reduced modularity of the whole-brain functional network, a hallmark of optimal organization of a network that describes its cost efficiency (Newman, 2006). Importantly, this link remains robust after controlling for age, a variable that is strongly associated with decreased modularity. Hence, this finding suggests that stress and adversities accumulated throughout the life course, reflected as reduced biological health, have a strong impact on brain functioning over and above the effect of natural aging. The nervous system and non-nervous structures of the organism, while coordinating with each other through bidirectional pathways, are simultaneously embedded in and attuned to the sociocultural environment.

Limitations and Future Directions

Several future lines of research may be derived from the work presented in this dissertation. Many of these future directions could result from a further integration of the main theme or methodology of each chapter. First, the present work examines how culture may shape the structural aspects of the brain. While this analysis is novel, the main dependent variable, GM volume, is a rather crude index of the functioning and organization of the brain. One interesting avenue of future research could be to examine how culture and different cultural dimensions, such as self-construal, are associated with functional connectivity of the brain network. For instance, as discussed in chapter two, independent individuals value and express personal

choices, preferences, and goal, and they have a clear sense of self. These behaviors are known to be governed by the executive network centered around the prefrontal regions of the brain (Northoff & Bermpohl, 2004; O'Doherty, 2011). Hence, it is plausible to expect that independent self-construal is related to the importance, or "centrality", of the executive network in the brain. Another direction of research could derive from recent work on culture and health. It has been shown that a better attunement between people and their cultural context, or "culture fit", is a robust predictor of biological health (Levine et al., 2016). Independent individuals are thus healthier in independent cultures, and interdependent individuals are healthier in interdependent cultures. It is unclear whether this pattern could be extended to index of "brain health", such as the modularity index tested in chapter four. Future work should test this possibility.

Second, in chapter three, I have investigated resilience among Black Americans against racial discrimination and how it can buffer the mortality risk. The focus of the study was on potential psychological components of resilience. Extant literature on resilience among Black Americans has focused on many other components of resilience, one of them being the "cultural processes". For instance, several studies have shown that factors such as racial socialization, racial identity, and religiosity are critical components of resilience and are reliable predictors of better educational and health outcomes among Black Americans (Neblett et al., 2006; Sellers et al., 2003). It might be obvious that the cultural processes examined in this literature are quite different from what have been focused on by the cultural psychology literature, although both lines of work seem to define "culture" in a similar fashion. Therefore, a better integration of the literature could yield informative insights. For instance, Black Americans are known to have a higher level of racial identity. It remains to be tested how the identification with one's own racial group is related to self-construal (e.g., interdependence), and whether certain self-construal could

be an antecedent or a component of resilience that promotes biological health among racial minorities.

Third, although the link between perceived discrimination and biological health has been well studied, no work so far has examined whether perceived discrimination is linked to indices of "brain health", such as cortical atrophy or functional organization of the brain. Opposing predictions could be made here. On the one hand, because perceived discrimination is unpleasant and stressful, it may lead to dysregulation of cortisol in the body, if such stress becomes chronic. This in turn can lead to atrophy of the brain since cortisol is excitotoxic to neurons (Sapolsky, 1994). On the other hand, to the extent that the perception and reporting of discrimination reflect resilience and various constructive coping processes, as demonstrated in chapter three, perceived discrimination may not necessarily predict compromised brain health and cognition. Future research should elucidate this link between perceived discrimination and brain.

Lastly, it should be acknowledged that many data used in the present dissertation is correlational in nature. While this is limited by the fact that some variables being tested, such as culture, cannot be manipulated, future longitudinal or intervention work could be extremely useful. For instance, future work can longitudinally follow up individuals immigrating to other cultures and examine whether changes in the brain can be detected. Such work will have the potential to extend the current work by further clarifying causality of relationships and elucidating mechanism of the effects.

Appendices

Appendix A: Chapter 3 Study 1 Supplementary Materials

Robustness Checks and Additional Analyses

To check the robustness of the findings in study 1, several variations of the main analysis were carried out. In the first variation, we additionally controlled for the potential effects of state-wide lockdowns. We computed the number of days during the 30-day period after the state-wide lock-down[18]. When this variable was entered as an additional covariate, it predicted a less steep increase of cases, b = -.006, p < .001 (Black-White segregation model), b = -.005, p < .001 (Hispanic-White segregation model), b = -.005, p < .001 (Asian-White segregation model). It also predicted a less steep growth of deaths, b = -.004, p = .023 (Black-White segregation model), b = -.004, p = .024 (Hispanic-White segregation model), b = -.003, p = .052 (Asian-White segregation model). Importantly, our key effects in the main analysis reported above remain unchanged (see Table A1 and A2).

In the second variation, we analyzed only the first 15 days (rather than 30 days) of the outbreaks. The focus on the shorter period may be desirable since various confounding factors, particularly reporting biases, are less likely to change systematically if the period is shorter. One downside of this analysis is that the data are reduced in half, thus making the estimation of growth curves less reliable compared to the 30-day analysis. The day x segregation interaction (Blacks and Hispanics) remained significant for both cases and deaths. The day x Gini x segregation interaction (Blacks and Hispanics) remained significant for deaths, although it was no longer significant for cases (Tables A3 and A4).

[18] The date at which the stay at home order was made effective in each state, as well as the first and last day of data used for each county are made available at the OSF website: https://osf.io/qm697/?view_only=c30e3ce756904c529c36feb2e028958b.

In the third variation, we used 100 cases (rather than 20 cases) to define the first day of county-wise outbreaks for the analysis of cases. This alternate cutoff is equally reasonable and has been used in prior work (Berg et al., 2020). All of our key effects remain unchanged (Table A5).

Fourth, because the Black-White segregation and the Hispanic-White segregation were strongly correlated ($r = .59$, $p < .0001$), we averaged the two indices to form a single index of segregation. When we ran the standard models reported in the main analyses, all effects replicated, and the 3-way interactions were highly significant for both cases and deaths (Tables A6 and A7).

Table A-1 Regression coefficients for confirmed COVID-19 cases for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models, after controlling for the potential effect of lockdown.

The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	4.523	100.619	<.001	4.532	100.458	<.001	4.530	93.554	<.001
Variables of Interest									
Day	0.071	32.426	<.001	0.071	34.072	<.001	0.071	29.659	<.001
Segregation	0.175	3.809	<.001	0.173	3.797	<.001	0.067	1.320	0.189
Day x Segregation	0.010	4.305	<.001	0.011	5.016	<.001	0.003	1.310	0.193
GINI	0.011	0.439	0.661	0.017	0.694	0.488	0.025	1.053	0.293
Day x GINI	0.000	0.166	0.868	0.000	0.220	0.826	0.001	0.928	0.354
Segregation x GINI	0.036	1.739	0.083	0.005	0.240	0.811	-0.023	-1.016	0.310
Day x Segregation x GINI	0.003	2.869	0.004	0.003	2.456	0.014	-0.000	-0.117	0.907
Minority Share									
Blacks Share	0.121	4.256	<.001	0.121	4.252	<.001	0.124	4.287	<.001
Day x Blacks Share	0.006	4.287	<.001	0.007	4.535	<.001	0.006	4.178	<.001
Hispanics Share	0.071	2.312	0.021	0.048	1.605	0.109	0.065	2.018	0.044
Day x Hispanics Share	0.005	3.463	0.001	0.004	2.437	0.015	0.005	3.037	0.003
Asians Share	-0.037	-1.401	0.162	-0.041	-1.557	0.120	-0.045	-1.687	0.092
Day x Asians Share	0.002	1.159	0.247	0.001	1.021	0.308	0.001	0.731	0.465
Covariates									
Natural Log of Population Size	0.743	23.416	<.001	0.757	24.035	<.001	0.760	23.939	<.001
Day x Natural Log of Population Size	0.030	18.227	<.001	0.031	19.059	<.001	0.032	18.736	<.001
Median Income	0.016	0.556	0.579	0.009	0.315	0.753	0.023	0.796	0.427

Day x Income	0.000	0.196	0.845	-0.000	-0.110	0.912	0.001	0.449	0.654
Population Density	0.060	2.636	0.009	0.075	3.293	0.001	0.082	3.838	<.001
Day x Population Density	0.002	2.009	0.045	0.003	2.277	0.023	0.004	3.470	0.001
Proportion Elderly	-0.002	-0.065	0.948	-0.002	-0.059	0.953	0.006	0.217	0.828
Day x Proportion Elderly	-0.000	-0.161	0.872	-0.000	-0.169	0.866	0.000	0.160	0.873
Duration after lockdown	-0.076	-3.214	0.001	-0.071	-2.981	0.003	-0.067	-2.799	0.005
Day x Duration after lockdown	-0.006	-4.652	<.001	-0.005	-4.301	<.001	-0.005	-4.132	<.001

Table A-2 Regression coefficients for COVID-19 deaths for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models, after controlling for the potential effect of lockdown.

The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	1.293	23.900	<.001	1.304	23.829	<.001	1.302	22.707	<.001
Variables of Interest									
Day	0.068	23.874	<.001	0.068	22.894	<.001	0.068	21.771	<.001
Segregation	0.177	3.161	0.002	0.155	2.738	0.007	0.093	1.491	0.139
Day x Segregation	0.012	4.291	<.001	0.010	3.162	0.002	0.007	2.006	0.047
GINI	0.100	2.328	0.020	0.100	2.356	0.019	0.121	2.847	0.005
Day x GINI	0.003	1.263	0.207	0.004	1.637	0.102	0.005	2.246	0.025
Segregation x GINI	0.043	1.239	0.216	0.052	1.432	0.153	-0.029	-0.747	0.456
Day x Segregation x GINI	0.007	4.198	<.001	0.006	3.264	0.001	0.001	0.618	0.537
Minority Share									
Blacks Share	0.166	3.751	<.001	0.164	3.693	<.001	0.166	3.673	<.001
Day x Blacks Share	0.006	2.800	0.005	0.007	2.898	0.004	0.006	2.613	0.009
Hispanics Share	0.018	0.407	0.685	-0.019	-0.436	0.663	0.010	0.201	0.841
Day x Hispanics Share	0.001	0.312	0.756	-0.002	-0.799	0.425	0.001	0.244	0.807
Asians Share	-0.061	-1.453	0.147	-0.061	-1.459	0.145	-0.072	-1.726	0.085
Day x Asians Share	0.001	0.468	0.640	0.001	0.319	0.750	-0.000	-0.102	0.919
Covariates									
Natural Log of Population Size	0.771	15.677	<.001	0.780	15.941	<.001	0.792	16.093	<.001
Day x Natural Log of Population Size	0.038	15.098	<.001	0.039	15.453	<.001	0.040	15.575	<.001
Median Income	0.134	2.831	0.005	0.121	2.530	0.012	0.154	3.198	0.001
Day x Income	0.003	1.078	0.281	0.002	0.778	0.437	0.004	1.630	0.104
Population Density	0.113	2.947	0.003	0.113	2.930	0.004	0.142	3.954	<.001

Day x Population Density	0.001	0.688	0.492	0.002	1.059	0.290	0.005	2.496	0.013
Proportion Elderly	0.083	1.946	0.052	0.082	1.911	0.057	0.093	2.144	0.033
Day x Proportion Elderly	0.002	0.970	0.333	0.002	1.050	0.294	0.003	1.412	0.159
Duration after lockdown	0.211	6.151	<.001	0.211	6.143	<.001	0.220	6.358	<.001
Day x Duration after lockdown	-0.004	-2.278	0.023	-0.004	-2.260	0.024	-0.003	-1.951	0.052

Table A-3 Regression coefficients for confirmed COVID-19 cases for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right). The analysis focused on the first 15 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	4.000	126.450	<.001	4.008	124.372	<.001	4.007	120.252	<.001
Variables of Interest									
Day	0.109	30.148	<.001	0.110	30.384	<.001	0.110	28.694	<.001
Segregation	0.098	3.001	0.003	0.088	2.680	0.009	0.039	1.091	0.278
Day x Segregation	0.012	3.234	0.002	0.012	3.208	0.002	0.005	1.153	0.251
GINI	0.013	0.650	0.516	0.018	0.901	0.368	0.020	1.028	0.305
Day x GINI	0.001	0.519	0.604	0.001	0.662	0.508	0.002	0.964	0.336
Segregation x GINI	0.010	0.582	0.561	-0.017	-0.966	0.334	-0.025	-1.360	0.174
Day x Segregation x GINI	0.001	0.821	0.412	-0.000	-0.207	0.836	-0.003	-1.400	0.162
Minority Share									
Blacks Share	0.081	3.625	<.001	0.077	3.444	0.001	0.082	3.621	<.001
Day x Blacks Share	0.010	3.956	<.001	0.009	3.855	<.001	0.010	3.959	<.001
Hispanics Share	0.021	0.884	0.377	0.009	0.373	0.709	0.014	0.574	0.567
Day x Hispanics Share	0.004	1.580	0.115	0.002	0.948	0.344	0.003	1.242	0.215
Asians Share	-0.054	-2.577	0.010	-0.056	-2.694	0.007	-0.058	-2.774	0.006
Day x Asians Share	-0.003	-1.151	0.250	-0.003	-1.255	0.210	-0.003	-1.361	0.174
Covariates									
Natural Log of Population Size	0.543	23.994	<.001	0.549	24.386	<.001	0.550	24.319	<.001
Day x Natural Log of Population Size	0.061	24.642	<.001	0.061	25.047	<.001	0.061	24.985	<.001
Median Income	0.022	0.973	0.331	0.018	0.784	0.434	0.027	1.160	0.247
Day x Income	0.002	0.874	0.382	0.002	0.629	0.529	0.003	1.041	0.298

Population Density	0.048	2.613	0.009	0.061	3.286	0.001	0.058	3.387	0.001
Day x Population Density	0.008	4.037	<.001	0.009	4.488	<.001	0.009	4.996	<.001
Proportion Elderly	0.002	0.093	0.926	0.001	0.070	0.944	0.005	0.240	0.810
Day x Proportion Elderly	0.002	0.857	0.392	0.002	0.811	0.418	0.002	1.009	0.313

Table A-4 Regression coefficients for COVID-19 deaths for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models. The analysis focused on the first 15 days of county-wise outbreaks.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	0.804	20.247	<.001	0.816	20.625	<.001	0.812	19.721	<.001
Variables of Interest									
Day	0.091	20.313	<.001	0.092	19.690	<.001	0.092	18.992	<.001
Segregation	0.128	3.044	0.003	0.118	2.755	0.007	0.097	2.070	0.041
Day x Segregation	0.017	3.679	<.001	0.013	2.588	0.011	0.012	2.202	0.030
GINI	0.077	1.861	0.063	0.069	1.673	0.095	0.079	1.948	0.052
Day x GINI	0.007	1.881	0.061	0.008	1.950	0.052	0.010	2.583	0.010
Segregation x GINI	-0.004	-0.128	0.898	0.022	0.632	0.528	-0.030	-0.804	0.422
Day x Segregation x GINI	0.006	2.011	0.045	0.007	2.144	0.033	-0.004	-1.260	0.208
Minority Share									
Blacks Share	0.094	2.398	0.017	0.086	2.191	0.029	0.091	2.278	0.023
Day x Blacks Share	0.010	2.665	0.008	0.010	2.644	0.009	0.011	2.712	0.007
Hispanics Share	0.019	0.494	0.621	-0.013	-0.343	0.732	0.017	0.417	0.677
Day x Hispanics Share	0.003	0.730	0.466	-0.001	-0.238	0.812	0.003	0.634	0.527
Asians Share	-0.082	-2.126	0.034	-0.079	-2.055	0.040	-0.093	-2.403	0.017
Day x Asians Share	-0.004	-1.158	0.247	-0.005	-1.202	0.230	-0.006	-1.591	0.112
Covariates									
Natural Log of Population Size	0.384	8.873	<.001	0.388	9.028	<.001	0.398	9.202	<.001
Day x Natural Log of Population Size	0.046	10.859	<.001	0.047	11.113	<.001	0.048	11.284	<.001
Median Income	0.115	2.710	0.007	0.102	2.346	0.019	0.142	3.277	0.001
Day x Income	0.010	2.337	0.020	0.009	2.081	0.038	0.013	2.959	0.003
Population Density	0.085	2.277	0.023	0.078	2.087	0.037	0.098	2.790	0.006
Day x Population Density	0.006	1.815	0.070	0.007	1.872	0.062	0.011	3.196	0.001
Proportion Elderly	0.044	1.157	0.248	0.042	1.099	0.273	0.053	1.366	0.173

Day x Proportion Elderly	0.007	1.768	0.078	0.007	1.804	0.072	0.008	2.119	0.035

Table A-5 Regression coefficients for confirmed COVID-19 cases for the Black segregation (left), Hispanic segregation (middle) and Asian segregation (right) models. The analysis focused on the first 30 days of county-wise outbreaks, with the first day as when more than 100 cases (instead of 20 cases) were reported.

Predictor	b	t	p	b	t	p	b	t	p
Intercept	5.407	123.874	<.001	5.413	130.121	<.001	5.413	114.327	<.001
Variables of Interest									
Day	0.037	18.725	<.001	0.038	20.847	<.001	0.037	16.967	<.001
Segregation	0.161	3.717	<.001	0.188	4.566	<.001	0.061	1.232	0.221
Day x Segregation	0.008	4.120	<.001	0.010	5.327	<.001	0.003	1.375	0.172
GINI	0.042	1.441	0.150	0.045	1.567	0.118	0.069	2.421	0.016
Day x GINI	0.002	1.732	0.084	0.002	1.846	0.066	0.003	2.710	0.007
Segregation x GINI	0.070	3.062	0.002	0.068	2.873	0.004	0.011	0.448	0.655
Day x Segregation x GINI	0.003	3.380	0.001	0.003	3.464	0.001	0.001	0.871	0.385
Minority Share									
Blacks Share	0.135	4.314	<.001	0.143	4.591	<.001	0.133	4.100	<.001
Day x Blacks Share	0.005	3.878	<.001	0.005	4.298	<.001	0.005	3.626	<.001
Hispanics Share	0.099	3.119	0.002	0.064	2.116	0.035	0.095	2.810	0.005
Day x Hispanics Share	0.006	4.514	<.001	0.004	3.263	0.001	0.006	4.304	<.001
Asians Share	0.010	0.378	0.706	0.007	0.244	0.808	-0.000	-0.010	0.992
Day x Asians Share	0.002	1.526	0.128	0.001	1.270	0.205	0.001	1.177	0.240
Covariates									
Natural Log of Population Size	0.569	16.085	<.001	0.579	16.466	<.001	0.582	16.084	<.001
Day x Natural Log of Population Size	0.026	18.687	<.001	0.026	18.995	<.001	0.027	18.681	<.001
Median Income	0.017	0.533	0.595	0.004	0.126	0.900	0.024	0.749	0.454
Day x Income	0.000	0.375	0.708	0.000	0.060	0.952	0.001	0.499	0.618

Population Density	0.063	2.717	0.007	0.066	2.802	0.005	0.092	4.153	<.001
Day x Population Density	0.002	2.239	0.026	0.002	2.225	0.027	0.003	3.684	<.001
Proportion Elderly	-0.006	-0.197	0.844	-0.007	-0.245	0.807	0.002	0.078	0.938
Day x Proportion Elderly	0.001	0.581	0.561	0.000	0.364	0.716	0.001	0.971	0.332

Table A-6 Regression coefficients for confirmed COVID-19 cases with Black segregation and Hispanic segregation combined as a single segregation index. The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p
Intercept	4.515	102.195	<.001
Variables of interest			
Day	0.070	33.594	<.001
Segregation	0.208	4.126	<.001
Day x Segregation	0.012	4.996	<.001
GINI	0.019	0.756	0.450
Day x GINI	0.001	0.423	0.673
Segregation x GINI	0.025	1.047	0.296
Day x Segregation x GINI	0.004	2.838	0.005
Minority Share			
Blacks Share	0.133	4.719	<.001
Day x Blacks Share	0.007	5.042	<.001
Hispanics Share	0.059	1.945	0.053
Day x Hispanics Share	0.004	2.936	0.004
Asians Share	-0.033	-1.267	0.206
Day x Asians Share	0.002	1.359	0.175
Covariates			
Natural Log of Population Size	0.793	27.841	<.001
Day x Natural Log of Population Size	0.034	22.875	<.001
Median Income	0.020	0.679	0.497
Day x Income	0.001	0.450	0.653
Population Density	0.070	2.999	0.003
Day x Population Density	0.003	2.135	0.033
Proportion Elderly	0.006	0.219	0.827
Day x Proportion Elderly	0.000	0.224	0.822

Table A-7 Regression coefficients for COVID-19 deaths with Black segregation and Hispanic segregation combined as a single segregation index. The analysis focused on the first 30 days of county-wise outbreaks.

Predictor	b	t	p
Intercept	1.313	24.249	<.001
Variables of Interest			
Day	0.067	23.569	<.001
Segregation	0.247	3.939	<.001
Day x Segregation	0.013	3.973	<.001
GINI	0.084	1.887	0.060
Day x GINI	0.003	1.284	0.200
Segregation x GINI	0.070	1.693	0.091
Day x Segregation x GINI	0.008	4.090	<.001
Minority Share			
Blacks Share	0.145	3.194	0.002
Day x Blacks Share	0.007	3.094	0.002
Hispanics Share	0.006	0.140	0.889
Day x Hispanics Share	-0.001	-0.333	0.739
Asians Share	-0.066	-1.530	0.127
Day x Asians Share	0.001	0.566	0.572
Covariates			
Natural Log of Population Size	0.680	14.111	<.001
Day x Natural Log of Population Size	0.040	16.746	<.001
Median Income	0.116	2.365	0.018
Day x Income	0.002	0.837	0.403
Population Density	0.088	2.173	0.030
Day x Population Density	0.001	0.651	0.515
Proportion Elderly	0.055	1.253	0.211
Day x Proportion Elderly	0.003	1.199	0.231

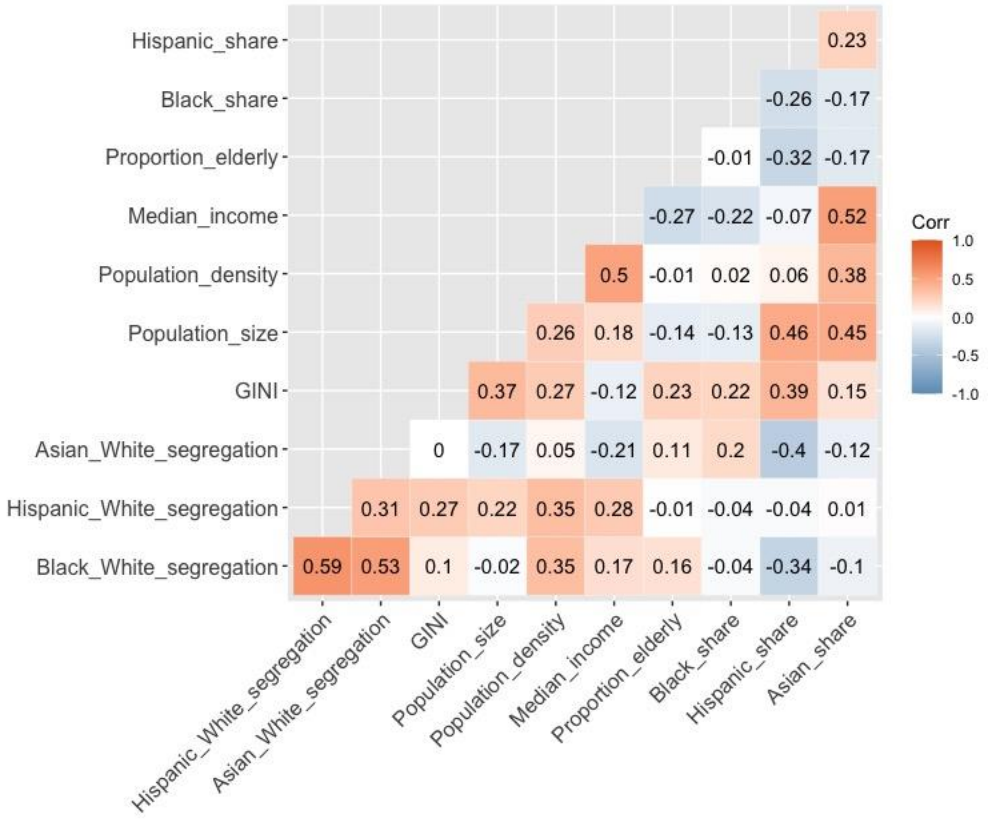

Figure A-1 Correlation matrix of the predictors included in the present analysis. The three segregation indices are at the MSA-level, whereas the rest are at the county-level. The correlations were computed by aggregating the county-level predictors across counties to form MSA-level predictors.

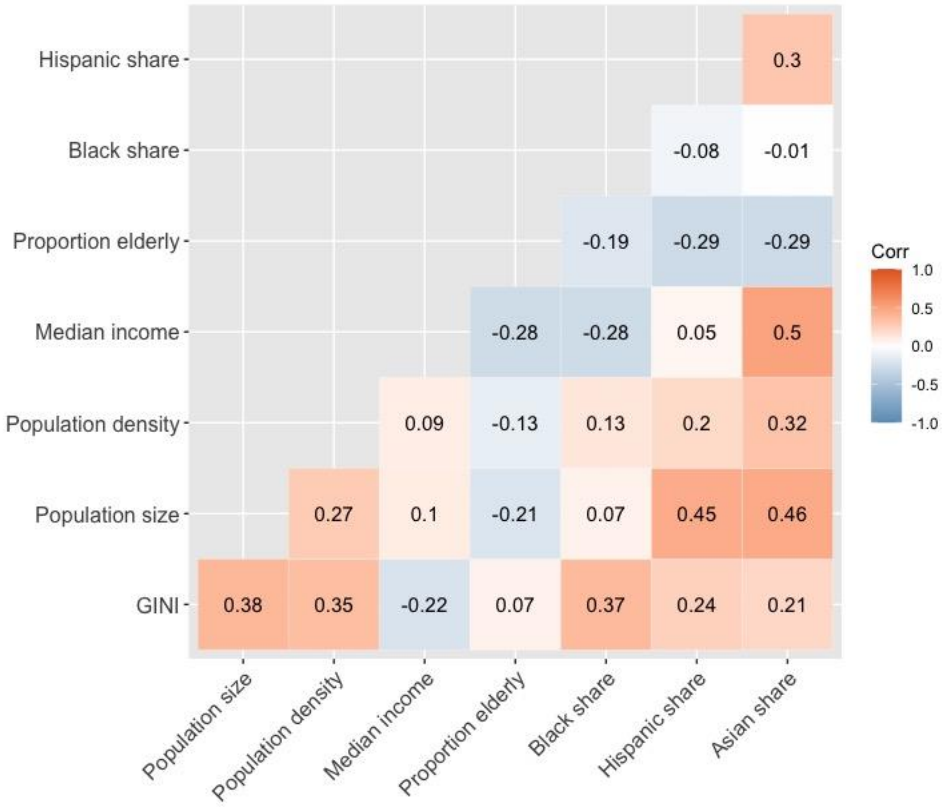

Figure A-2 Correlation matrix of the county-level predictors included in the present analysis.

Appendix B: Chapter 3 Study 2 Supplementary Materials

Table A-8 Resilience composite as a function of perceived lifetime discrimination among White and Black Americans in M1.

	Dependent variable:			
	Composite score			
	White Americans	White Americans	Black Americans	Black Americans
	(1)	(2)	(3)	(4)
Discrimination	0.006	−0.002	0.100***	0.071***
	(0.008)	(0.008)	(0.018)	(0.019)
Age		−0.007***		−0.011***
		(0.001)		(0.003)
Sex		−0.073***		0.087
		(0.020)		(0.092)
Income		0.00000***		0.00000***
		(0.00000)		(0.00000)
Constant	0.010	0.217***	−0.151**	0.007
	(0.012)	(0.053)	(0.059)	(0.244)
Observations	5,453	5,316	301	289
R^2	0.0001	0.112	0.092	0.209
Adjusted R^2	−0.0001	0.111	0.089	0.198
Note:				*p<0.1; **p<0.05; ***p<0.01

Table A-9 Resilience composite as a function of perceived lifetime discrimination among White and Black Americans in M2.

	Dependent variable:			
	Composite score			
	White Americans	White Americans	Black Americans	Black Americans
	(1)	(2)	(3)	(4)
Discrimination	−0.010	−0.011	0.064***	0.057***
	(0.009)	(0.009)	(0.011)	(0.011)
Age		−0.002*		0.001
		(0.001)		(0.002)
Sex		0.024		0.124**
		(0.026)		(0.057)
Income		0.00001***		0.00001***
		(0.00000)		(0.00000)
Constant	0.021	−0.149*	−0.187***	−0.706***
	(0.015)	(0.080)	(0.039)	(0.165)
Observations	3,457	3,239	737	705
R^2	0.0003	0.106	0.041	0.153
Adjusted R^2	0.0001	0.105	0.040	0.148
Note:				*p<0.1; **p<0.05; ***p<0.01

Table A-10 Resilience composite as a function of perceived lifetime discrimination among White and Black Americans in M3.

	Dependent variable:			
	Composite score			
	White Americans	White Americans	Black Americans	Black Americans
	(1)	(2)	(3)	(4)
Discrimination	−0.025**	−0.017	0.078***	0.062***
	(0.011)	(0.011)	(0.015)	(0.014)
Age		−0.004***		−0.0004
		(0.001)		(0.003)
Sex		0.007		0.086
		(0.031)		(0.070)
Income		0.00000***		0.00001***
		(0.00000)		(0.00000)
Constant	0.031*	0.090	−0.144***	−0.541**
	(0.017)	(0.110)	(0.045)	(0.228)
Observations	2,409	2,253	489	475
R^2	0.002	0.108	0.052	0.213
Adjusted R^2	0.002	0.106	0.050	0.206
Note:				*p<0.1; **p<0.05; ***p<0.01

Table A-11 Resilience composite as a function of perceived lifetime discrimination among White and Black Americans in MR.

	Dependent variable:			
	Composite score			
	White Americans	White Americans	Black Americans	Black Americans
	(1)	(2)	(3)	(4)
Discrimination	−0.041***	−0.033***	0.047***	0.035***
	(0.011)	(0.011)	(0.014)	(0.013)
Age		−0.003***		0.001
		(0.001)		(0.003)
Sex		0.082**		0.286***
		(0.033)		(0.060)
Income		0.00000***		0.00001***
		(0.00000)		(0.00000)
Constant	0.054***	−0.182**	−0.158***	−0.917***
	(0.019)	(0.086)	(0.043)	(0.148)
Observations	2,013	1,933	683	668
R^2	0.007	0.119	0.016	0.188
Adjusted R^2	0.006	0.117	0.014	0.183
Note:				*p<0.1; **p<0.05; ***p<0.01

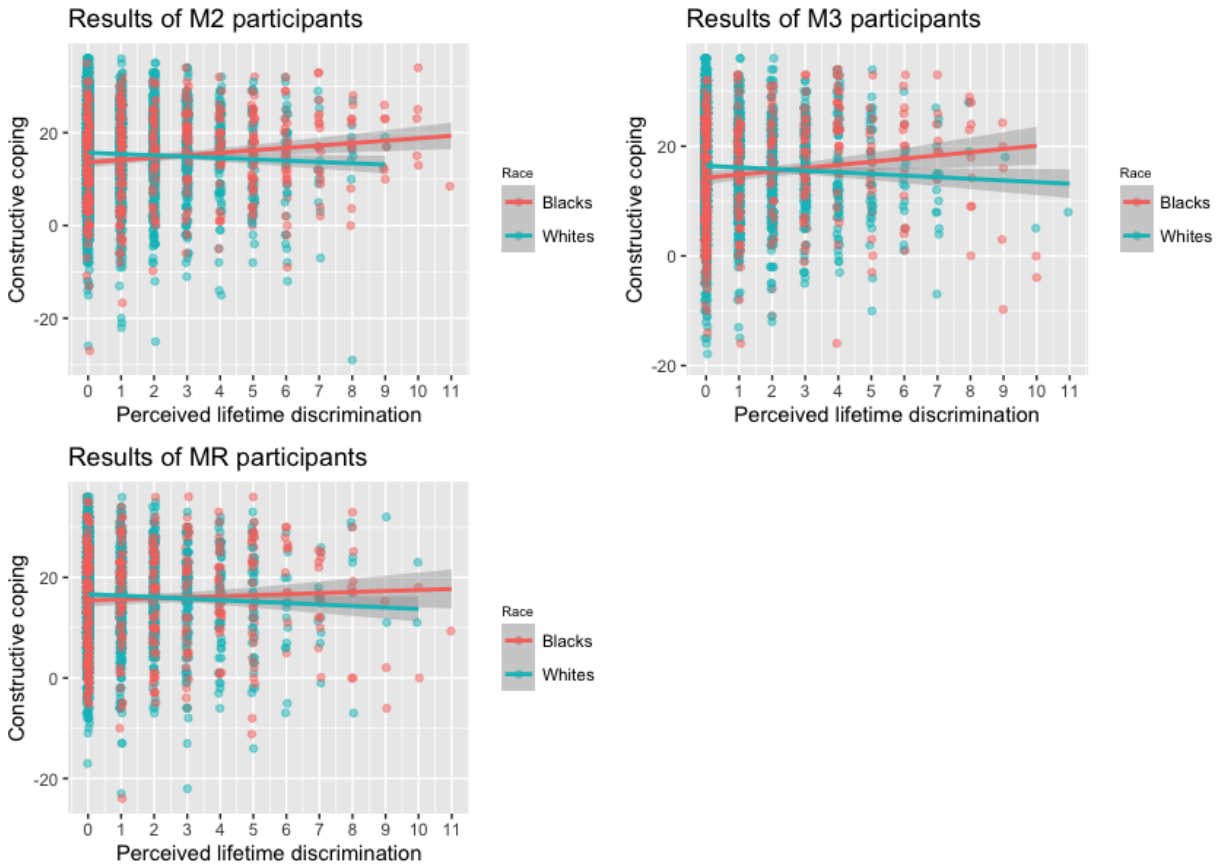

Figure A-3 Effect of perceived lifetime discrimination on constructive coping across three MIDUS study samples with coping assessed. Across the four samples, perceived discrimination predicted greater constructive coping among Black Americans, *p*s < .05, whereas it predicted less constructive coping among White Americans, *p*s < .05. The racial difference in the effect was significant across the four samples, *p*s < .05.

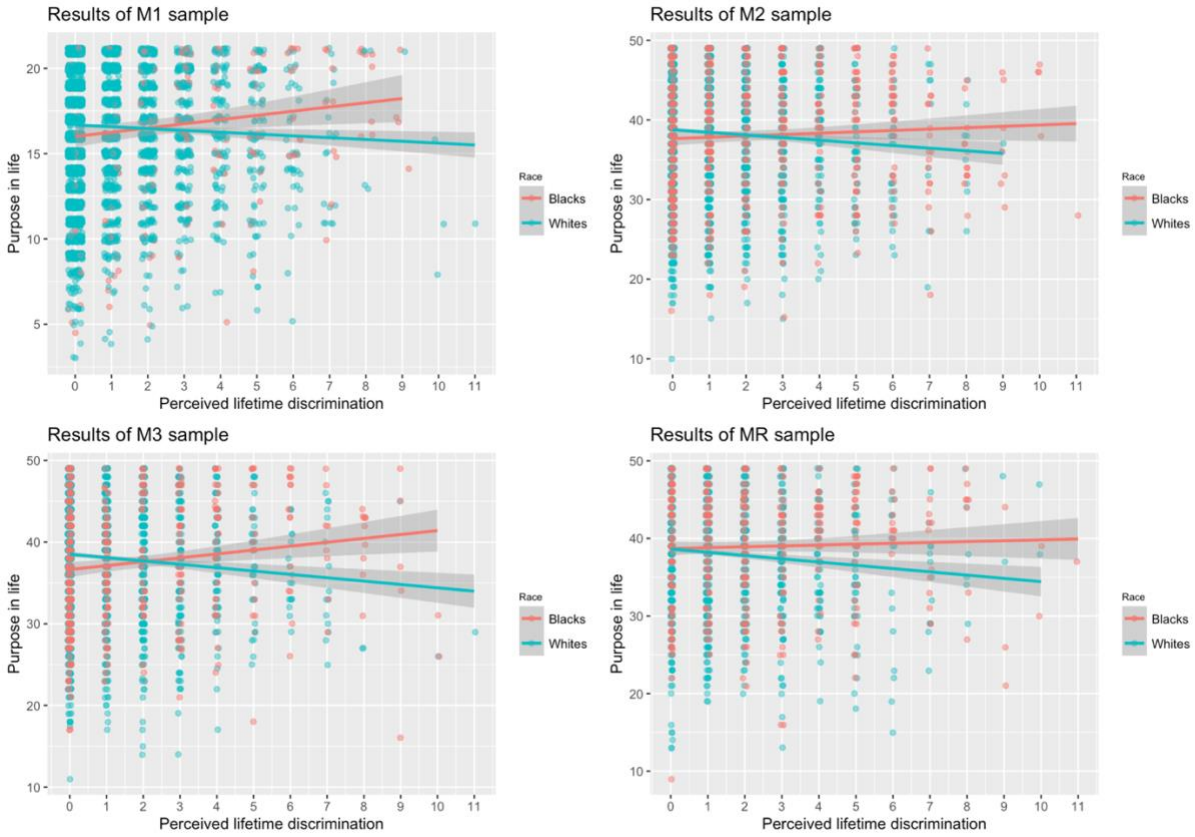

Figure A-4 Effect of perceived lifetime discrimination on purpose in life across four MIDUS study samples. Perceived discrimination predicted reduced purpose in life among White Americans, *p*s < .001. It did not predict reduced purpose, and in some cases (M1 and M3) it predicted increased purpose among Black Americans, *p*s < .05. The racial difference in the effect was significant across the four samples, *p*s < .05.

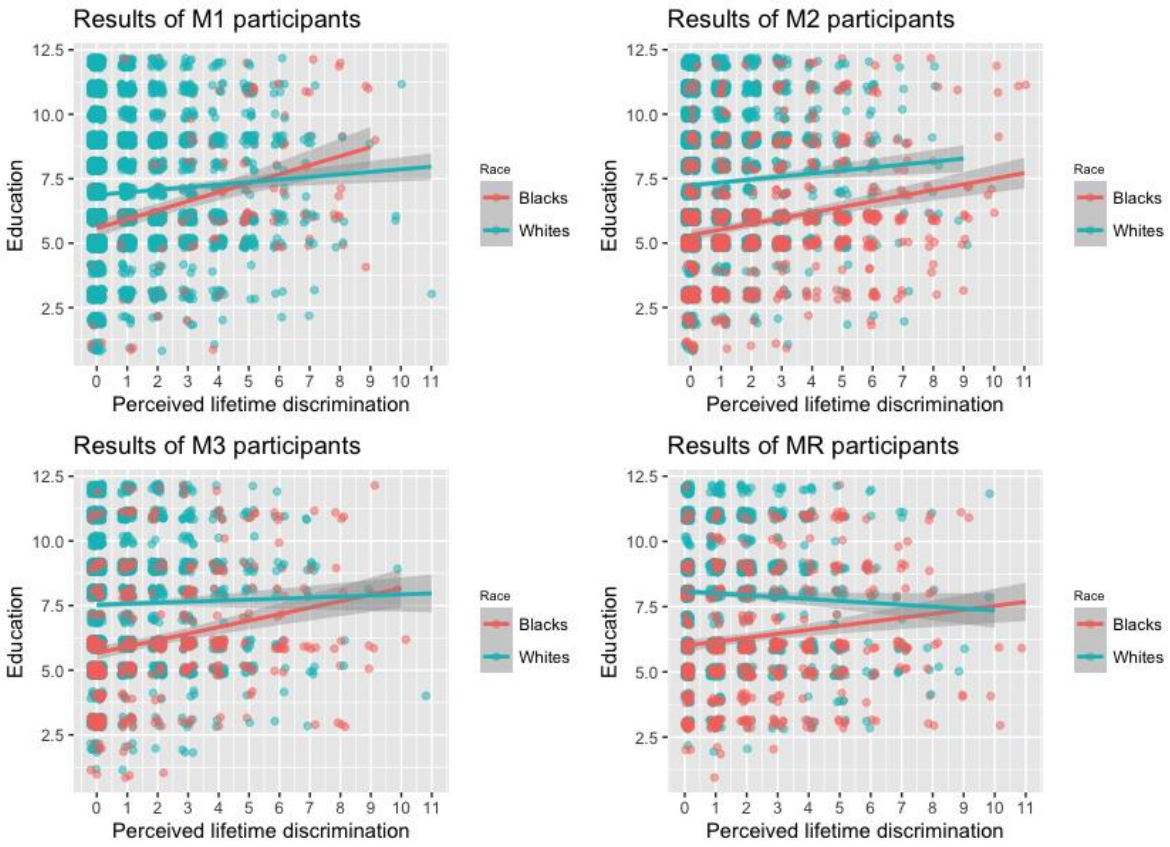

Figure A-5 Effect of perceived lifetime discrimination on educational attainment across four MIDUS study samples. Perceived discrimination predicted greater educational attainment among Black Americans across the four samples, *p*s < .001. This effect was attenuated and reversed among White Americans. The racial difference in the effect was significant across the four samples, *p*s < .05.

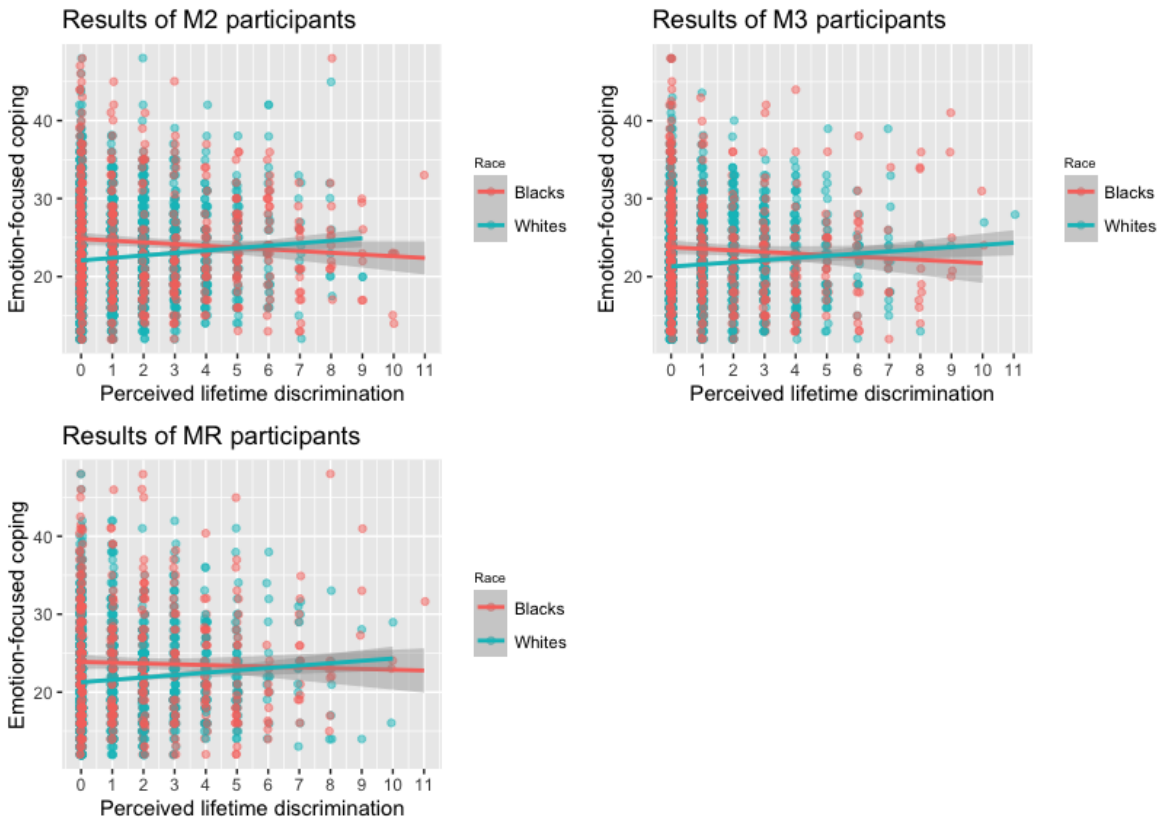

Figure A-6 Effect of perceived lifetime discrimination on emotion-focused coping across three MIDUS study samples. Perceived discrimination predicted more emotion-focused coping among White Americans across the three samples, *p*s < .001. This effect was absent among Black Americans, *p*s > .20. The racial difference in the effect was significant, *p*s < .05, except in MR, where it was marginal, *p* = .069.

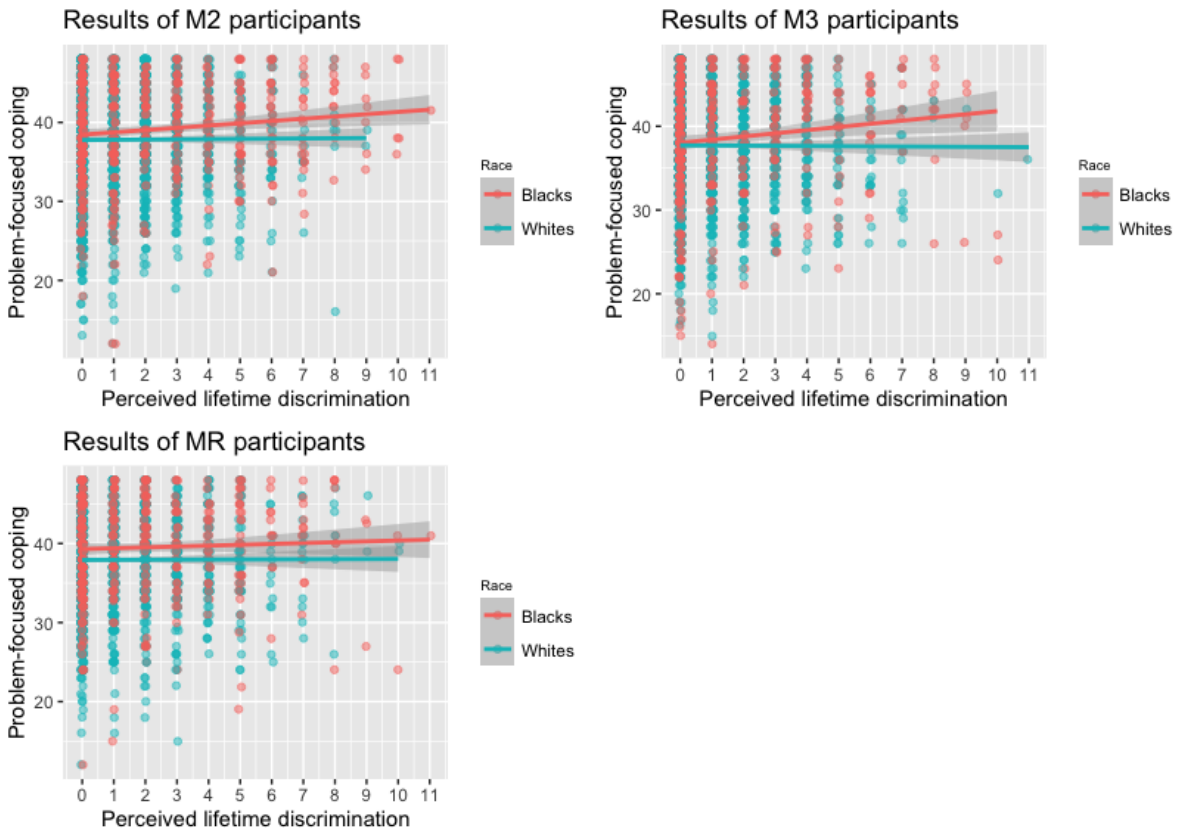

Figure A-7 Effect of perceived lifetime discrimination on problem-focused coping across three MIDUS study samples. Perceived discrimination predicted more problem-focused coping among Black Americans in M2 and M3, *p*s < .05. The effect was not significant in MR. This effect was absent among White Americans, *p*s > .20. The racial difference in the effect was significant in M3, $p = .036$, and it was marginal in M2, $p = .089$.

Appendix C: Chapter 4 Supplementary Materials

In Chapter 3, I presented results on participants' cognitive performance. The cognitive performance was measured using various cognitive tests. Here, I include detailed procedures about each of the tests, as well as the how performance on each of the tasks was quantified. A detailed documentation of the tasks can be found in the MIDUS study series at the ICPSR website of the University of Michigan: https://www.icpsr.umich.edu/web/pages/NACDA/midus.html.

Participants of the present study completed the Cambridge Neuropsychological Test Automated Battery (CANTAB). Tests of the CANTAB were self-administered using a Portable SlimBook Panel Touchscreen PC and presspad, equipped with CANTAB software version 5.0. There are seven cognitive tests in total, three assessing attention and psychomotor speed, three assessing executive function, and one assessing emotion and social cognition. In most cases, the tests are completed in the order as below, except for the Cambridge Gambling Task, which was completed on a separate occasion.

Motor Screening Task

The motor screening task (MOT) was used to measure sensorimotor deficits. It also served the purpose of screening a lack of comprehension, so as to ensure that valid data can be collected from the participants. All participants in the present study were able to complete the MOT[19] adequately. In this test, colored crosses appeared in random locations on the screen successively. Participants were asked to click on the cross as quickly and accurately as possible.

[19] One participant missed the session where CANTAB was administered. However, this participant completed the Cambridge Gambling Task and was able to perform above average.

Performance was measured by the mean latency (i.e., reaction time) across trials. The lower the latency, the higher the performance.

Intra-Extra Dimensional Set Shift

The Intra-Extra Dimensional Set Shift (IED) test measures the ability to learn rules and rule reversals. This test is similar to the Wisconsin Card Sorting test.

Participants during this test see color-filled shapes, white lines, or combination of the two (white lines overlying color-filled shapes). The test starts by presenting two simple color-filled shapes, and participants must learn which one is correct through trial-and-error. The stimuli and/or rules change after six correct responses. The change of rule is initially intra-dimensional (ID), such that color-filled shapes remain the only relevant dimension for solving the trial. In later trials extra-dimensional (ED) rule shift occurs, such that white lines become the only relevant dimension. Participants can progress through different stages after six consecutive correct responses. The test terminates if at any stage the participant fails to produce six consecutive correct responses. Performance was measured as the number of ED errors. The more the errors, the lower the performance.

Affective Go/No-Go

The Affective Go/No-Go (AGN) test assesses affective bias on information processing. In each block, a series of words are presented successively. Each block features words from two of three different affective categories: positive, negative, and neutral. One affective is the target category and the other is the distractor category in each block. Participants are asked to press the screen only for words from the target category. Performance was assessed as the mean latency across correct trials. The lower the latency, the better the performance.

Information Sampling Task

The Information Sampling Task (IST) tests impulsivity and decision making. Each trial starts with a 5x5 array of gray boxes and two larger colored panels (two difference colors) below these boxes. Each gray box can be clicked to reveal its color, which is one of the two colors of the panels below. Participants are to guess which color under the gray boxes is in the majority. They must select the gray boxes one at a time, and the boxes remain open after selected. When participants have made their decision about which color is in the majority, they must select the panel of that color at the bottom of the screen to indicate their choice. They are then informed whether their choice is correct or not, and all boxes reveal their color. There are two conditions. In the fixed win condition, 100 points are awarded for a correct decision, no matter how many boxes have been opened. In the decreasing win condition, 250 points can be won for a correct decision at the beginning, and it decreases by 10 points for every box selected. In both conditions, an incorrect decision costs 100 points. Performance is assessed by the quality of decision, which is quantified as the probability that the color chosen by the subject at the point of decision would be correct, based only on the evidence available to the subject at that time.

Attention Switching Task

The Attention Switching Task (AST) is a test of the ability to switch attention between the direction or location of an arrow on screen. It is similar to the Stroop test. In each trial, the participants see an arrow. The arrow might appear on the left or the right side of the screen. Participants are asked to press the left or right button, either according to the direction the arrow is pointing, or according to the physical location of the arrow. Performance was assessed by the number of incorrect trials. The more the errors, the lower the performance.

The Emotion Recognition Task

The Emotion Recognition Task (ERT) measures the ability to discriminate six basic emotions (sadness, happiness, fear, anger, disgust, and surprise) based on facial expressions. On each trial, a face showing a specific emotion is displayed. The expression of the emotion varies in its magnitude from trial to trial. Each face is displayed for 200 ms and then immediately covered up. Participants are then asked to choose among the six emotions the one that was shown. Performance was measured by the number of correct trials.

The Cambridge Gambling Task

The Cambridge Gambling Task (CGT) assesses decision-making and risk-taking. On each trial, participants see a row of ten boxes across the top of the screen, some of which are red and some of which are blue (see below). The participants are to guess whether a yellow token is hidden in a red box or a blue box, and they make their choice by choosing one of the two rectangles containing either the word “Red” or “Blue”. Participants can decide on how much points they place for the bet. There are two conditions. In the ascending bet condition, participants start with a number of points, and the points gradually go up until they place the bet. In the descending bet condition, participants start with a number of points, and the points gradually go down until they place the bet. Participants are asked to accumulate as many points as possible. Performance was measured as the quality of the decision, which is the proportion of the trials on which the participant chooses to gamble on the more likely outcome.

Modularity and Performance of Each Cognitive Task

Modularity tended to show a positive correlation with cognitive performance across several domains, controlling for age, sex, education, race, alcohol consumption, smoking status, and medication use. It is worth noting the correlation failed to achieve statistical significance in most tasks, likely due to the low reliability when a cognitive capacity is measured with only a

single brief task. Modularity was positively associated with a quicker reaction on the Affective Go/No-Go task, b = .291, t(61) = 2.240, p = .029. Modularity tended to be positively associated with a higher accuracy on the Attention Switching Task, b = .194, t(61) = 1.591, p = .117, and a quicker reaction on the Motor Screening Task, b = .156, t(61) = 1.230, p = .173. Modularity showed a weak association with the number of correct extra-dimension shifts on the Intra-Extra Dimensional Set Shift Task, b = .117, t(61) = .923, p = .360, the quality of decision on the Information Sampling Task, b = -.135, t(61) = 1.034, p = .305, the number of correct recognition on the Emotion Recognition Task, b = -.007, t(61) = -.070, p = .944, and the quality of decision on the Cambridge Gambling Task, b = .022, t(61) = .180, p = .858. Overall, it appears that in the current data, modularity is associated with better processing speed and attentional control, but it does not appear to be associated with executive function and social cognition.

Bibliography

Åberg, M. A. I., Pedersen, N. L., Torén, K., Svartengren, M., Bäckstrand, B., Johnsson, T., Cooper-Kuhn, C. M., Åberg, N. D., Nilsson, M., & Kuhn, H. G. (2009). Cardiovascular fitness is associated with cognition in young adulthood. *Proceedings of the National Academy of Sciences*, *106*(49), 20906–20911. https://doi.org/10.1073/pnas.0905307106

Akee, R., Jones, M. R., & Porter, S. R. (2019). Race Matters: Income Shares, Income Inequality, and Income Mobility for All U.S. Races. *Demography*, *56*(3), 999–1021. https://doi.org/10.1007/s13524-019-00773-7

Alimujiang, A., Wiensch, A., Boss, J., Fleischer, N. L., Mondul, A. M., McLean, K., Mukherjee, B., & Pearce, C. L. (2019). Association Between Life Purpose and Mortality Among US Adults Older Than 50 Years. *JAMA Network Open*, *2*(5), e194270. https://doi.org/10.1001/jamanetworkopen.2019.4270

Anderson, R. M. (1982). *The Population Dynamics of Infectious Diseases: Theory and Applications*. Springer US. https://doi.org/10.1007/978-1-4899-2901-3

Apperly, I. A., Samson, D., Chiavarino, C., & Humphreys, G. W. (2004). Frontal and Temporo-Parietal Lobe Contributions to Theory of Mind: Neuropsychological Evidence from a False-Belief Task with Reduced Language and Executive Demands. *Journal of Cognitive Neuroscience*, *16*(10), 1773–1784. https://doi.org/10.1162/0898929042947928

Asghari, V., Sanyal, S., Buchwaldt, S., Paterson, A., Jovanovic, V., & Van Tol, H. H. (1995). Modulation of intracellular cyclic AMP levels by different human dopamine D4 receptor variants. *Journal of Neurochemistry*, *65*(3), 1157–1165. https://doi.org/10.1046/j.1471-4159.1995.65031157.x

Ashburner, J. (2007). A fast diffeomorphic image registration algorithm. *NeuroImage*, *38*(1), 95–113. https://doi.org/10.1016/j.neuroimage.2007.07.007

Ashburner, J., & Friston, K. J. (2000). Voxel-Based Morphometry—The Methods. *NeuroImage*, *11*(6), 805–821. https://doi.org/10.1006/nimg.2000.0582

Barnes, L. L., de Leon, C. F. M., Lewis, T. T., Bienias, J. L., Wilson, R. S., & Evans, D. A. (2008). Perceived Discrimination and Mortality in a Population-Based Study of Older Adults. *American Journal of Public Health*, *98*(7), 1241–1247. https://doi.org/10.2105/AJPH.2007.114397

Bates, D., Kliegl, R., Vasishth, S., & Baayen, H. (2018). Parsimonious Mixed Models. *ArXiv:1506.04967 [Stat]*. http://arxiv.org/abs/1506.04967

Bates, D., Mächler, M., Bolker, B., & Walker, S. (2015). Fitting Linear Mixed-Effects Models Using lme4. *Journal of Statistical Software*, *67*(1), 1–48. https://doi.org/10.18637/jss.v067.i01

Baum, G. L., Ciric, R., Roalf, D. R., Betzel, R. F., Moore, T. M., Shinohara, R. T., Kahn, A. E., Vandekar, S. N., Rupert, P. E., Quarmley, M., Cook, P. A., Elliott, M. A., Ruparel, K., Gur, R. E., Gur, R. C., Bassett, D. S., & Satterthwaite, T. D. (2017). Modular Segregation

of Structural Brain Networks Supports the Development of Executive Function in Youth. *Current Biology*, *27*(11), 1561-1572.e8. https://doi.org/10.1016/j.cub.2017.04.051

Behzadi, Y., Restom, K., Liau, J., & Liu, T. T. (2007). A component based noise correction method (CompCor) for BOLD and perfusion based fMRI. *NeuroImage*, *37*(1), 90–101. https://doi.org/10.1016/j.neuroimage.2007.04.042

Belsky, J., & Pluess, M. (2009a). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*(6), 885–908. https://doi.org/10.1037/a0017376

Belsky, J., & Pluess, M. (2009b). The Nature (and Nurture?) of Plasticity in Early Human Development. *Perspectives on Psychological Science*, *4*(4), 345–351. https://doi.org/10.1111/j.1745-6924.2009.01136.x

Ben-Shaanan, T. L., Azulay-Debby, H., Dubovik, T., Starosvetsky, E., Korin, B., Schiller, M., Green, N. L., Admon, Y., Hakim, F., Shen-Orr, S. S., & Rolls, A. (2016). Activation of the reward system boosts innate and adaptive immunity. *Nature Medicine*, *22*(8), 940–944. https://doi.org/10.1038/nm.4133

Benson, G., Hildebrandt, A., Lange, C., Schwarz, C., Köbe, T., Sommer, W., Flöel, A., & Wirth, M. (2018). Functional connectivity in cognitive control networks mitigates the impact of white matter lesions in the elderly. *Alzheimer's Research & Therapy*, *10*(1), 109. https://doi.org/10.1186/s13195-018-0434-3

Berg, M. K., Yu, Q., Salvador, C. E., Melani, I., & Kitayama, S. (2020). Mandated Bacillus Calmette-Guérin (BCG) vaccination predicts flattened curves for the spread of COVID-19. *Science Advances*, *6*(32), eabc1463. https://doi.org/10.1126/sciadv.abc1463

Bertolero, M. A., Yeo, B. T. T., Bassett, D. S., & D'Esposito, M. (2018). A mechanistic model of connector hubs, modularity and cognition. *Nature Human Behaviour*, *2*(10), 765–777. https://doi.org/10.1038/s41562-018-0420-6

Bischoff, K., & Reardon, S. F. (2013). Residential Segregation by Income, 1970-2009. *US2010 Project*. https://cepa.stanford.edu/content/residential-segregation-income-1970-2009

Blatter, D. D., Bigler, E. D., Gale, S. D., Johnson, S. C., Anderson, C. V., Burnett, B. M., Parker, N., Kurth, S., & Horn, S. D. (1995). Quantitative volumetric analysis of brain MR: Normative database spanning 5 decades of life. *AJNR. American Journal of Neuroradiology*, *16*(2), 241–251.

Bleidorn, W., Kandler, C., Hülsheger, U. R., Riemann, R., Angleitner, A., & Spinath, F. M. (2010). Nature and nurture of the interplay between personality traits and major life goals. *Journal of Personality and Social Psychology*, *99*(2), 366–379. https://doi.org/10.1037/a0019982

Blondel, V. D., Guillaume, J.-L., Lambiotte, R., & Lefebvre, E. (2008). Fast unfolding of communities in large networks. *Journal of Statistical Mechanics: Theory and Experiment*, *2008*(10), P10008. https://doi.org/10.1088/1742-5468/2008/10/P10008

Blum, S., Brow, M., & Silver, R. C. (2012). Coping. In V. S. Ramachandran (Ed.), *Encyclopedia of Human Behavior (Second Edition)* (pp. 596–601). Academic Press. https://doi.org/10.1016/B978-0-12-375000-6.00110-5

Boyle, P. A., Barnes, L. L., Buchman, A. S., & Bennett, D. A. (2009). Purpose in Life Is Associated With Mortality Among Community-Dwelling Older Persons. *Psychosomatic Medicine*, *71*(5), 574–579. https://doi.org/10.1097/PSY.0b013e3181a5a7c0

Branscombe, N. R., Schmitt, M. T., & Harvey, R. D. (1999). Perceiving pervasive discrimination among African Americans: Implications for group identification and well-being. *Journal*

of Personality and Social Psychology, *77*(1), 135–149. https://doi.org/10.1037/0022-3514.77.1.135
Bullmore, E., & Sporns, O. (2009). Complex brain networks: Graph theoretical analysis of structural and functional systems. *Nature Reviews Neuroscience*, *10*(3), 186–198. https://doi.org/10.1038/nrn2575
Bullmore, E., & Sporns, O. (2012). The economy of brain network organization. *Nature Reviews Neuroscience*, *13*(5), 336–349. https://doi.org/10.1038/nrn3214
Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology*, *56*(2), 267–283. https://doi.org/10.1037//0022-3514.56.2.267
Case, A., & Deaton, A. (2015). Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century. *Proceedings of the National Academy of Sciences*, *112*(49), 15078–15083. https://doi.org/10.1073/pnas.1518393112
Case, A., & Deaton, A. (2021). *Deaths of Despair and the Future of Capitalism*. Princeton University Press.
Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science (New York, N.Y.)*, *301*(5631), 386–389. https://doi.org/10.1126/science.1083968
Centers for Disease Control and Prevention. (2020, February 11). *COVID-19 Hospitalization and Death by Race/Ethnicity*. Centers for Disease Control and Prevention. https://www.cdc.gov/coronavirus/2019-ncov/covid-data/investigations-discovery/hospitalization-death-by-race-ethnicity.html
Chee, M. W. L., Zheng, H., Goh, J. O. S., Park, D., & Sutton, B. P. (2010). Brain Structure in Young and Old East Asians and Westerners: Comparisons of Structural Volume and Cortical Thickness. *Journal of Cognitive Neuroscience*, *23*(5), 1065–1079. https://doi.org/10.1162/jocn.2010.21513
Chiel, H. J., & Beer, R. D. (1997). The brain has a body: Adaptive behavior emerges from interactions of nervous system, body and environment. *Trends in Neurosciences*, *20*(12), 553–557. https://doi.org/10.1016/S0166-2236(97)01149-1
Christian, M. D., & Barbarin, O. A. (2001). Cultural resources and psychological adjustment of African American children: Effects of spirituality and racial attribution. *Journal of Black Psychology*, *27*(1), 43–63. https://doi.org/10.1177/0095798401027001003
Clark, A. (1998). *Being There: Putting Brain, Body, and World Together Again*. The MIT Press. https://mitpress.mit.edu/books/being-there
Cohen, D., & Gunz, A. (2002). As Seen by the Other …: Perspectives on the Self in the Memories and Emotional Perceptions of Easterners and Westerners. *Psychological Science*, *13*(1), 55–59. https://doi.org/10.1111/1467-9280.00409
Cohen, J. R., & D'Esposito, M. (2016). The Segregation and Integration of Distinct Brain Networks and Their Relationship to Cognition. *The Journal of Neuroscience*, *36*(48), 12083–12094. https://doi.org/10.1523/JNEUROSCI.2965-15.2016
Cole, S. W. (2013). Social Regulation of Human Gene Expression: Mechanisms and Implications for Public Health. *American Journal of Public Health*, *103*(S1), S84–S92. https://doi.org/10.2105/AJPH.2012.301183
Cole, S. W. (2014). Human social genomics. *PLoS Genetics*, *10*(8), e1004601. https://doi.org/10.1371/journal.pgen.1004601

Cole, S. W., Hawkley, L. C., Arevalo, J. M. G., & Cacioppo, J. T. (2011). Transcript origin analysis identifies antigen-presenting cells as primary targets of socially regulated gene expression in leukocytes. *Proceedings of the National Academy of Sciences*, *108*(7), 3080–3085. https://doi.org/10.1073/pnas.1014218108

Collins, C. A., & Williams, D. R. (1999a). Segregation and Mortality: The Deadly Effects of Racism? *Sociological Forum*, *14*(3), 495–523. https://doi.org/10.1023/A:1021403820451

Collins, C. A., & Williams, D. R. (1999b). Segregation and Mortality: The Deadly Effects of Racism? *Sociological Forum*, *14*(3), 495–523. https://doi.org/10.1023/A:1021403820451

Collins, W. A., Maccoby, E. E., Steinberg, L., Hetherington, E. M., & Bornstein, M. H. (2000). Contemporary research on parenting: The case for nature and nurture. *American Psychologist*, *55*(2), 218–232. https://doi.org/10.1037/0003-066X.55.2.218

Contreras, J. A., Avena-Koenigsberger, A., Risacher, S. L., West, J. D., Tallman, E., McDonald, B. C., Farlow, M. R., Apostolova, L. G., Goñi, J., Dzemidzic, M., Wu, Y.-C., Kessler, D., Jeub, L., Fortunato, S., Saykin, A. J., & Sporns, O. (2019). Resting state network modularity along the prodromal late onset Alzheimer's disease continuum. *NeuroImage: Clinical*, *22*, 101687. https://doi.org/10.1016/j.nicl.2019.101687

Cooper, R. S., Kennelly, J. F., Durazo-Arvizu, R., Oh, H. J., Kaplan, G., & Lynch, J. (2001). Relationship between premature mortality and socioeconomic factors in black and white populations of US metropolitan areas. *Public Health Reports*, *116*(5), 464–473.

Coulton, C. J., Chow, J., Wang, E. C., & Su, M. (1996). Geographic Concentration of Affluence and Poverty in 100 Metropolitan Areas, 1990. *Urban Affairs Review*, *32*(2), 186–216. https://doi.org/10.1177/107808749603200203

Cox, D. R. (1972). Regression Models and Life-Tables. *Journal of the Royal Statistical Society. Series B (Methodological)*, *34*(2), 187–220.

Crocker, J., & Major, B. (1989). Social stigma and self-esteem: The self-protective properties of stigma. *Psychological Review*, *96*(4), 608–630. https://doi.org/10.1037/0033-295X.96.4.608

Czopp, A. M., Monteith, M. J., & Mark, A. Y. (2006). Standing up for a change: Reducing bias through interpersonal confrontation. *Journal of Personality and Social Psychology*, *90*(5), 784–803. https://doi.org/10.1037/0022-3514.90.5.784

Dantzer, R., Konsman, J. P., Bluthé, R. M., & Kelley, K. W. (2000). Neural and humoral pathways of communication from the immune system to the brain: Parallel or convergent? *Autonomic Neuroscience: Basic & Clinical*, *85*(1–3), 60–65. https://doi.org/10.1016/S1566-0702(00)00220-4

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews. Neuroscience*, *9*(1), 46–56. https://doi.org/10.1038/nrn2297

Davies, G., Tenesa, A., Payton, A., Yang, J., Harris, S. E., Liewald, D., Ke, X., Le Hellard, S., Christoforou, A., Luciano, M., McGhee, K., Lopez, L., Gow, A. J., Corley, J., Redmond, P., Fox, H. C., Haggarty, P., Whalley, L. J., McNeill, G., … Deary, I. J. (2011). Genome-wide association studies establish that human intelligence is highly heritable and polygenic. *Molecular Psychiatry*, *16*(10), 996–1005. https://doi.org/10.1038/mp.2011.85

Delaney, M. B., & Reed, L. (2015). Recognizing And Responding to a New Era of Infectious and Communicable Diseases. *Journal of Emergency Nursing*, *41*(2), 138–140. https://doi.org/10.1016/j.jen.2014.12.009

Devos, T., & Banaji, M. R. (2005). American = White? *Journal of Personality and Social Psychology*, *88*(3), 447–466. https://doi.org/10.1037/0022-3514.88.3.447

Diez-Roux, A. V., Link, B. G., & Northridge, M. E. (2000). A multilevel analysis of income inequality and cardiovascular disease risk factors. *Social Science & Medicine*, *50*(5), 673–687. https://doi.org/10.1016/S0277-9536(99)00320-2

Dolezsar, C. M., McGrath, J. J., Herzig, A. J. M., & Miller, S. B. (2014). Perceived racial discrimination and hypertension: A comprehensive systematic review. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association*, *33*(1), 20–34. https://doi.org/10.1037/a0033718

Draganski, B., Gaser, C., Busch, V., Schuierer, G., Bogdahn, U., & May, A. (2004). Changes in grey matter induced by training. *Nature*, *427*(6972), 311–312. https://doi.org/10.1038/427311a

Duncan, E. S., & Small, S. L. (2016). Increased Modularity of Resting State Networks Supports Improved Narrative Production in Aphasia Recovery. *Brain Connectivity*, *6*(7), 524–529. https://doi.org/10.1089/brain.2016.0437

Duncan, O. D., & Duncan, B. (1955). A Methodological Analysis of Segregation Indexes. *American Sociological Review*, *20*(2), 210–217. JSTOR. https://doi.org/10.2307/2088328

Eisenberger, N. I., Inagaki, T. K., Rameson, L. T., Mashal, N. M., & Irwin, M. R. (2009). An fMRI study of cytokine-induced depressed mood and social pain: The role of sex differences. *NeuroImage*, *47*(3), 881–890. https://doi.org/10.1016/j.neuroimage.2009.04.040

Epstein, R. (2008). Parahippocampal and retrosplenial contributions to human spatial navigation. *Trends in Cognitive Sciences*, *12*(10), 388–396. https://doi.org/10.1016/j.tics.2008.07.004

Erickson, K. I., Voss, M. W., Prakash, R. S., Basak, C., Szabo, A., Chaddock, L., Kim, J. S., Heo, S., Alves, H., White, S. M., Wojcicki, T. R., Mailey, E., Vieira, V. J., Martin, S. A., Pence, B. D., Woods, J. A., McAuley, E., & Kramer, A. F. (2011). Exercise training increases size of hippocampus and improves memory. *Proceedings of the National Academy of Sciences*, *108*(7), 3017–3022. https://doi.org/10.1073/pnas.1015950108

Evans, G. W., & Schamberg, M. A. (2009). Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences*, *106*(16), 6545–6549. https://doi.org/10.1073/pnas.0811910106

Felger, J. C., Li, Z., Haroon, E., Woolwine, B. J., Jung, M. Y., Hu, X., & Miller, A. H. (2016). Inflammation is associated with decreased functional connectivity within corticostriatal reward circuitry in depression. *Molecular Psychiatry*, *21*(10), 1358–1365. https://doi.org/10.1038/mp.2015.168

Ferrari, C. C., & Tarelli, R. (2011). Parkinson's disease and systemic inflammation. *Parkinson's Disease*, *2011*, 436813. https://doi.org/10.4061/2011/436813

Frey, W. H., & Myers, D. (2005). *Racial segregation in us metropolitan areas and cities, 1990-2000: Patterns, trends, and explanations. Population Studies Center Research Report 05-573*.

Fuller-Rowell, T. E., Doan, S. N., & Eccles, J. S. (2012). Differential effects of perceived discrimination on the diurnal cortisol rhythm of African Americans and Whites. *Psychoneuroendocrinology*, *37*(1), 107–118. https://doi.org/10.1016/j.psyneuen.2011.05.011

Garrison, K. A., Scheinost, D., Finn, E. S., Shen, X., & Constable, R. T. (2015). The (in)stability of functional brain network measures across thresholds. *NeuroImage*, *118*, 651–661. https://doi.org/10.1016/j.neuroimage.2015.05.046

Gaser, C., & Schlaug, G. (2003). Brain Structures Differ between Musicians and Non-Musicians. *Journal of Neuroscience*, *23*(27), 9240–9245. https://doi.org/10.1523/JNEUROSCI.23-27-09240.2003

Gaskin, D. J., Dinwiddie, G. Y., Chan, K. S., & McCleary, R. (2012). Residential Segregation and Disparities in Healthcare Services Utilization. *Medical Care Research and Review*, *69*(2), 158–175. https://doi.org/10.1177/1077558711420263

Geerligs, L., Maurits, N. M., Renken, R. J., & Lorist, M. M. (2012). Reduced specificity of functional connectivity in the aging brain during task performance. *Human Brain Mapping*, *35*(1), 319–330. https://doi.org/10.1002/hbm.22175

Geerligs, L., Renken, R. J., Saliasi, E., Maurits, N. M., & Lorist, M. M. (2015). A Brain-Wide Study of Age-Related Changes in Functional Connectivity. *Cerebral Cortex*, *25*(7), 1987–1999. https://doi.org/10.1093/cercor/bhu012

Geerligs, L., Saliasi, E., Maurits, N. M., & Lorist, M. M. (2012). Compensation through increased functional connectivity: Neural correlates of inhibition in old and young. *Journal of Cognitive Neuroscience*, *24*(10), 2057–2069. https://doi.org/10.1162/jocn_a_00270

Geronimus, A. T., Pearson, J. A., Linnenbringer, E., Schulz, A. J., Reyes, A. G., Epel, E. S., Lin, J., & Blackburn, E. H. (2015). Race-Ethnicity, Poverty, Urban Stressors, and Telomere Length in a Detroit Community-based Sample. *Journal of Health and Social Behavior*, *56*(2), 199–224. https://doi.org/10.1177/0022146515582100

Gianaros, P. J., & Wager, T. D. (2015). Brain-Body Pathways Linking Psychological Stress and Physical Health. *Current Directions in Psychological Science*, *24*(4), 313–321. https://doi.org/10.1177/0963721415581476

Gibbons, F. X., Gerrard, M., Fleischli, M. E., Simons, R. L., & Kingsbury, J. H. (2021). Perceived Racial Discrimination and Healthy Behavior among African Americans. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *40*(3), 155–165. https://doi.org/10.1037/hea0001056

Gigerenzer, G. (2008). Why Heuristics Work. *Perspectives on Psychological Science*, *3*(1), 20–29. https://doi.org/10.1111/j.1745-6916.2008.00058.x

Glass, C. K., Saijo, K., Winner, B., Marchetto, M. C., & Gage, F. H. (2010). Mechanisms Underlying Inflammation in Neurodegeneration. *Cell*, *140*(6), 918–934. https://doi.org/10.1016/j.cell.2010.02.016

Glazer, J., King, A., Yoon, C., Liberzon, I., & Kitayama, S. (2020). DRD4 polymorphisms modulate reward positivity and P3a in a gambling task: Exploring a genetic basis for cultural learning. *Psychophysiology*, *57*(10), e13623. https://doi.org/10.1111/psyp.13623

Goodman, M., Lyons, S., Dean, L. T., Arroyo, C., & Hipp, J. A. (2018). How Segregation Makes Us Fat: Food Behaviors and Food Environment as Mediators of the Relationship Between Residential Segregation and Individual Body Mass Index. *Frontiers in Public Health*, *6*. https://doi.org/10.3389/fpubh.2018.00092

Gordon, E. M., Lynch, C. J., Gratton, C., Laumann, T. O., Gilmore, A. W., Greene, D. J., Ortega, M., Nguyen, A. L., Schlaggar, B. L., Petersen, S. E., Dosenbach, N. U. F., & Nelson, S. M. (2018). Three Distinct Sets of Connector Hubs Integrate Human Brain Function. *Cell Reports*, *24*(7), 1687-1695.e4. https://doi.org/10.1016/j.celrep.2018.07.050

Goto, S. G., Yee, A., Lowenberg, K., & Lewis, R. S. (2013). Cultural differences in sensitivity to social context: Detecting affective incongruity using the N400. *Social Neuroscience*, *8*(1), 63–74. https://doi.org/10.1080/17470919.2012.739202

Grill-Spector, K., Kourtzi, Z., & Kanwisher, N. (2001). The lateral occipital complex and its role in object recognition. *Vision Research*, *41*(10), 1409–1422. https://doi.org/10.1016/S0042-6989(01)00073-6

Guimerà, R., & Amaral, L. A. N. (2005). Cartography of complex networks: Modules and universal roles. *Journal of Statistical Mechanics: Theory and Experiment*, *2005*(02), P02001. https://doi.org/10.1088/1742-5468/2005/02/P02001

Hall, O. T., Jordan, A., Teater, J., Dixon-Shambley, K., McKiever, M. E., Baek, M., Garcia, S., Rood, K. M., & Fielin, D. A. (2021). Experiences of racial discrimination in the medical setting and associations with medical mistrust and expectations of care among black patients seeking addiction treatment. *Journal of Substance Abuse Treatment*, 108551. https://doi.org/10.1016/j.jsat.2021.108551

Harrison, N. A., Brydon, L., Walker, C., Gray, M. A., Steptoe, A., & Critchley, H. D. (2009). Inflammation Causes Mood Changes Through Alterations in Subgenual Cingulate Activity and Mesolimbic Connectivity. *Biological Psychiatry*, *66*(5), 407–414. https://doi.org/10.1016/j.biopsych.2009.03.015

Harrison, N. A., Brydon, L., Walker, C., Gray, M. A., Steptoe, A., Dolan, R. J., & Critchley, H. D. (2009). Neural Origins of Human Sickness in Interoceptive Responses to Inflammation. *Biological Psychiatry*, *66*(5), 415–422. https://doi.org/10.1016/j.biopsych.2009.03.007

Harrison, N. A., Doeller, C. F., Voon, V., Burgess, N., & Critchley, H. D. (2014). Peripheral inflammation acutely impairs human spatial memory via actions on medial temporal lobe glucose metabolism. *Biological Psychiatry*, *76*(7), 585–593. https://doi.org/10.1016/j.biopsych.2014.01.005

Haselton, M. G., Nettle, D., & Andrews, P. W. (2015). The Evolution of Cognitive Bias. In *The Handbook of Evolutionary Psychology* (pp. 724–746). John Wiley & Sons, Ltd. https://doi.org/10.1002/9780470939376.ch25

Hayasaka, S., & Nichols, T. E. (2004). Combining voxel intensity and cluster extent with permutation test framework. *NeuroImage*, *23*(1), 54–63. https://doi.org/10.1016/j.neuroimage.2004.04.035

He, Y., Wang, J., Wang, L., Chen, Z. J., Yan, C., Yang, H., Tang, H., Zhu, C., Gong, Q., Zang, Y., & Evans, A. C. (2009). Uncovering Intrinsic Modular Organization of Spontaneous Brain Activity in Humans. *PLOS ONE*, *4*(4), e5226. https://doi.org/10.1371/journal.pone.0005226

Hedden, T., & Gabrieli, J. D. E. (2004). Insights into the ageing mind: A view from cognitive neuroscience. *Nature Reviews Neuroscience*, *5*(2), 87–96. https://doi.org/10.1038/nrn1323

Heijmans, B. T., Tobi, E. W., Stein, A. D., Putter, H., Blauw, G. J., Susser, E. S., Slagboom, P. E., & Lumey, L. H. (2008). Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences of the United States of America*, *105*(44), 17046–17049. https://doi.org/10.1073/pnas.0806560105

Heine, S. J. (2001). Self as cultural product: An examination of East Asian and North American selves. *Journal of Personality*, *69*(6), 881–906. https://doi.org/10.1111/1467-6494.696168

Heine, S. J., Lehman, D. R., Markus, H. R., & Kitayama, S. (1999). Is there a universal need for positive self-regard? *Psychological Review*, *106*(4), 766–794. https://doi.org/10.1037/0033-295X.106.4.766

Hilger, K., Ekman, M., Fiebach, C. J., & Basten, U. (2017). Intelligence is associated with the modular structure of intrinsic brain networks. *Scientific Reports*, *7*(1), 16088. https://doi.org/10.1038/s41598-017-15795-7

Hill, P. L., & Turiano, N. A. (2014). Purpose in Life as a Predictor of Mortality across Adulthood. *Psychological Science*, *25*(7), 1482–1486. https://doi.org/10.1177/0956797614531799

Hill, P. L., Turiano, N. A., Mroczek, D. K., & Burrow, A. L. (2016). The Value of a Purposeful Life: Sense of Purpose Predicts Greater Income and Net Worth. *Journal of Research in Personality*, *65*, 38–42. https://doi.org/10.1016/j.jrp.2016.07.003

Hillman, C. H., Erickson, K. I., & Kramer, A. F. (2008). Be smart, exercise your heart: Exercise effects on brain and cognition. *Nature Reviews Neuroscience*, *9*(1), 58–65. https://doi.org/10.1038/nrn2298

Ho, A. K., Sidanius, J., Levin, D. T., & Banaji, M. R. (2011). Evidence for hypodescent and racial hierarchy in the categorization and perception of biracial individuals. *Journal of Personality and Social Psychology*, *100*(3), 492–506. https://doi.org/10.1037/a0021562

Holliday, R., Holliday, R., Monk, M., & Pugh, J. E. (1990). DNA methylation and epigenetic inheritance. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences*, *326*(1235), 329–338. https://doi.org/10.1098/rstb.1990.0015

Huang, C.-M., Doole, R., Wu, C. W., Huang, H.-W., & Chao, Y.-P. (2019). Culture-Related and Individual Differences in Regional Brain Volumes: A Cross-Cultural Voxel-Based Morphometry Study. *Frontiers in Human Neuroscience*, *13*. https://doi.org/10.3389/fnhum.2019.00313

Hummer, R. A., & Hernandez, E. M. (2013). The Effect of Educational Attainment on Adult Mortality in the United States*. *Population Bulletin*, *68*(1), 1–16.

Hunter, M. A., & Robinson, Z. F. (2016). The Sociology of Urban Black America. *Annual Review of Sociology*, *42*(1), 385–405. https://doi.org/10.1146/annurev-soc-081715-074356

Inagaki, T. K., Muscatell, K. A., Irwin, M. R., Cole, S. W., & Eisenberger, N. I. (2012). Inflammation selectively enhances amygdala activity to socially threatening images. *NeuroImage*, *59*(4), 3222–3226. https://doi.org/10.1016/j.neuroimage.2011.10.090

Intrator, J., Tannen, J., & Massey, D. S. (2016). Segregation by race and income in the United States 1970–2010. *Social Science Research*, *60*, 45–60. https://doi.org/10.1016/j.ssresearch.2016.08.003

Iordan, A. D., Cooke, K. A., Moored, K. D., Katz, B., Buschkuehl, M., Jaeggi, S. M., Jonides, J., Peltier, S. J., Polk, T. A., & Reuter-Lorenz, P. A. (2018). Aging and Network Properties: Stability Over Time and Links with Learning during Working Memory Training. *Frontiers in Aging Neuroscience*, *9*. https://doi.org/10.3389/fnagi.2017.00419

John, O. P., & Gross, J. J. (2004). Healthy and Unhealthy Emotion Regulation: Personality Processes, Individual Differences, and Life Span Development. *Journal of Personality*, *72*(6), 1301–1334. https://doi.org/10.1111/j.1467-6494.2004.00298.x

Jorgensen, R. S., & Thibodeau, R. (2007). Defensive avoidance of disapproval: The relationship of a defensive style to physical and mental health. *Harvard Review of Psychiatry*, *15*(1), 9–17. https://doi.org/10.1080/10673220601183923

Juster, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*(1), 2–16. https://doi.org/10.1016/j.neubiorev.2009.10.002

Kaholokula, J. K., Antonio, M. C. K., Ing, C. K. T., Hermosura, A., Hall, K. E., Knight, R., & Wills, T. A. (2017). The effects of perceived racism on psychological distress mediated by venting and disengagement coping in Native Hawaiians. *BMC Psychology*, *5*(1), 2. https://doi.org/10.1186/s40359-017-0171-6

Kawachi, I., Kennedy, B. P., Lochner, K., & Prothrow-Stith, D. (1997). Social capital, income inequality, and mortality. *American Journal of Public Health*, *87*(9), 1491–1498. https://doi.org/10.2105/AJPH.87.9.1491

Kershaw, K. N., & Pender, A. E. (2016). Racial/Ethnic Residential Segregation, Obesity, and Diabetes Mellitus. *Current Diabetes Reports*, *16*(11), 108. https://doi.org/10.1007/s11892-016-0800-0

Kessler, R. C., Mickelson, K. D., & Williams, D. R. (1999). The Prevalence, Distribution, and Mental Health Correlates of Perceived Discrimination in the United States. *Journal of Health and Social Behavior*, *40*(3), 208–230. https://doi.org/10.2307/2676349

Kim, E. S., Strecher, V. J., & Ryff, C. D. (2014). Purpose in life and use of preventive health care services. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(46), 16331–16336. https://doi.org/10.1073/pnas.1414826111

Kim, H., & Markus, H. R. (1999). Deviance or uniqueness, harmony or conformity? A cultural analysis. *Journal of Personality and Social Psychology*, *77*(4), 785–800. https://doi.org/10.1037/0022-3514.77.4.785

Kim, H. S., & Sasaki, J. Y. (2014). Cultural Neuroscience: Biology of the Mind in Cultural Contexts. *Annual Review of Psychology*, *65*(1), 487–514. https://doi.org/10.1146/annurev-psych-010213-115040

Kitayama, S., King, A., Hsu, M., Liberzon, I., & Yoon, C. (2016). Dopamine-System Genes and Cultural Acquisition: The Norm Sensitivity Hypothesis. *Current Opinion in Psychology*, *8*, 167–174. https://doi.org/10.1016/j.copsyc.2015.11.006

Kitayama, S., King, A., Yoon, C., Tompson, S., Huff, S., & Liberzon, I. (2014). The Dopamine D4 Receptor Gene (DRD4) Moderates Cultural Difference in Independent Versus Interdependent Social Orientation. *Psychological Science*, *25*(6), 1169–1177. https://doi.org/10.1177/0956797614528338

Kitayama, S., Markus, H. R., & Kurokawa, M. (2000). Culture, Emotion, and Well-being: Good Feelings in Japan and the United States. *Cognition and Emotion*, *14*(1), 93–124. https://doi.org/10.1080/026999300379003

Kitayama, S., Park, H., Sevincer, A. T., Karasawa, M., & Uskul, A. K. (2009a). A cultural task analysis of implicit independence: Comparing North America, Western Europe, and East Asia. *Journal of Personality and Social Psychology*, *97*(2), 236–255. https://doi.org/10.1037/a0015999

Kitayama, S., Park, H., Sevincer, A. T., Karasawa, M., & Uskul, A. K. (2009b). A cultural task analysis of implicit independence: Comparing North America, Western Europe, and East Asia. *Journal of Personality and Social Psychology*, *97*(2), 236–255. https://doi.org/10.1037/a0015999

Kitayama, S., & Park, J. (2014). Error-related brain activity reveals self-centric motivation: Culture matters. *Journal of Experimental Psychology: General*, *143*(1), 62–70. https://doi.org/10.1037/a0031696

Kitayama, S., & Park, J. (2020). Is Conscientiousness Always Associated With Better Health? A U.S.–Japan Cross-Cultural Examination of Biological Health Risk. *Personality and Social Psychology Bulletin*, 0146167220929824. https://doi.org/10.1177/0146167220929824

Kitayama, S., Park, J., Boylan, J. M., Miyamoto, Y., Levine, C. S., Markus, H. R., Karasawa, M., Coe, C. L., Kawakami, N., Love, G. D., & Ryff, C. D. (2015). Anger Expression and Ill-Health in Two Cultures: An Examination of Inflammation and Cardiovascular Risk. *Psychological Science*, *26*(2), 211–220. https://doi.org/10.1177/0956797614561268

Kitayama, S., Park, J., Miyamoto, Y., Date, H., Boylan, J. M., Markus, H. R., Karasawa, M., Kawakami, N., Coe, C. L., Love, G. D., & Ryff, C. D. (2018). Behavioral Adjustment Moderates the Link Between Neuroticism and Biological Health Risk: A U.S.–Japan Comparison Study: *Personality and Social Psychology Bulletin*. https://doi.org/10.1177/0146167217748603

Kitayama, S., & Salvador, C. E. (2017). Culture Embrained: Going Beyond the Nature-Nurture Dichotomy. *Perspectives on Psychological Science*, *12*(5), 841–854. https://doi.org/10.1177/1745691617707317

Kitayama, S., & Uskul, A. K. (2011). Culture, Mind, and the Brain: Current Evidence and Future Directions. *Annual Review of Psychology*, *62*(1), 419–449. https://doi.org/10.1146/annurev-psych-120709-145357

Kitayama, S., Varnum, M., & Salvador, C. (2019). Cultural Neuroscience. In D. Cohen & S. Kitayama (Eds.), *Handbook of Cultural Psychology, Second Edition* (pp. 79–118). Guilford Publications.

Kitayama, S., Yanagisawa, K., Ito, A., Ueda, R., Uchida, Y., & Abe, N. (2017). Reduced orbitofrontal cortical volume is associated with interdependent self-construal. *Proceedings of the National Academy of Sciences*, *114*(30), 7969–7974. https://doi.org/10.1073/pnas.1704831114

Kitayama, S., & Yu, Q. (2020). Mutual Constitution of Culture and the Mind: Insights from Cultural Neuroscience. In L. J. Kirmayer, C. M. Worthman, S. Kitayama, R. Lemelson, & C. A. Cummings (Eds.), *Culture, Mind, and Brain: Emerging Concepts, Models, and Applications* (pp. 88–119). Cambridge University Press. https://doi.org/10.1017/9781108695374.006

Krall, S. C., Rottschy, C., Oberwelland, E., Bzdok, D., Fox, P. T., Eickhoff, S. B., Fink, G. R., & Konrad, K. (2015). The role of the right temporoparietal junction in attention and social interaction as revealed by ALE meta-analysis. *Brain Structure & Function*, *220*(2), 587–604. https://doi.org/10.1007/s00429-014-0803-z

Kraus, M. W., Onyeador, I. N., Daumeyer, N. M., Rucker, J. M., & Richeson, J. A. (2019). The Misperception of Racial Economic Inequality. *Perspectives on Psychological Science*, *14*(6), 899–921. https://doi.org/10.1177/1745691619863049

Kraus, M. W., Piff, P. K., & Keltner, D. (2009). Social class, sense of control, and social explanation. *Journal of Personality and Social Psychology*, *97*(6), 992–1004. https://doi.org/10.1037/a0016357

Kraus, M. W., Rucker, J. M., & Richeson, J. A. (2017). Americans misperceive racial economic equality. *Proceedings of the National Academy of Sciences*, *114*(39), 10324–10331. https://doi.org/10.1073/pnas.1707719114

Kraynak, T. E., Marsland, A. L., Wager, T. D., & Gianaros, P. J. (2018). Functional neuroanatomy of peripheral inflammatory physiology: A meta-analysis of human neuroimaging studies. *Neuroscience and Biobehavioral Reviews*, *94*, 76–92. https://doi.org/10.1016/j.neubiorev.2018.07.013

Krieger, N., & Sidney, S. (1996). Racial discrimination and blood pressure: The CARDIA Study of young black and white adults. *American Journal of Public Health*, *86*(10), 1370–1378. https://doi.org/10.2105/ajph.86.10.1370

Krys, K., Vignoles, V. L., de Almeida, I., & Uchida, Y. (2022). Outside the "Cultural Binary": Understanding Why Latin American Collectivist Societies Foster Independent Selves. *Perspectives on Psychological Science*, 17456916211029632. https://doi.org/10.1177/17456916211029632

Kullmann, J. S., Grigoleit, J.-S., Wolf, O. T., Engler, H., Oberbeck, R., Elsenbruch, S., Forsting, M., Schedlowski, M., & Gizewski, E. R. (2014). Experimental human endotoxemia enhances brain activity during social cognition. *Social Cognitive and Affective Neuroscience*, *9*(6), 786–793. https://doi.org/10.1093/scan/nst049

Lancichinetti, A., & Fortunato, S. (2012). Consensus clustering in complex networks. *Scientific Reports*, *2*(1), 336. https://doi.org/10.1038/srep00336

Laughlin, S. B., de Ruyter van Steveninck, R. R., & Anderson, J. C. (1998). The metabolic cost of neural information. *Nature Neuroscience*, *1*(1), 36–41. https://doi.org/10.1038/236

LaVeist, T. A., Sellers, R., & Neighbors, H. W. (2001). Perceived racism and self and system blame attribution: Consequences for longevity. *Ethnicity & Disease*, *11*(4), 711–721.

Leach, C. W., & Allen, A. M. (2017). The Social Psychology of the Black Lives Matter Meme and Movement. *Current Directions in Psychological Science*, *26*(6), 543–547. https://doi.org/10.1177/0963721417719319

LeClere, F. B., & Soobader, M. J. (2000). The effect of income inequality on the health of selected US demographic groups. *American Journal of Public Health*, *90*(12), 1892–1897.

Levine, C. S., Miyamoto, Y., Markus, H. R., Rigotti, A., Boylan, J. M., Park, J., Kitayama, S., Karasawa, M., Kawakami, N., Coe, C. L., Love, G. D., & Ryff, C. D. (2016). Culture and Healthy Eating: The Role of Independence and Interdependence in the United States and Japan. *Personality & Social Psychology Bulletin*, *42*(10), 1335–1348. https://doi.org/10.1177/0146167216658645

Lewin, K. (1936). *Principles of topological psychology* (pp. xv, 238). McGraw-Hill. https://doi.org/10.1037/10019-000

Locke, J. (1836). *An Essay Concerning Human Understanding*. T. Tegg and Son.

Lopez, A. E., & Jean-Marie, G. (2021). Challenging Anti-Black Racism in Everyday Teaching, Learning, and Leading: From Theory to Practice. *Journal of School Leadership*, *31*(1–2), 50–65. https://doi.org/10.1177/1052684621993115

Lord, L.-D., Expert, P., Huckins, J. F., & Turkheimer, F. E. (2013). Cerebral energy metabolism and the brain's functional network architecture: An integrative review. *Journal of Cerebral Blood Flow and Metabolism: Official Journal of the International Society of Cerebral Blood Flow and Metabolism*, *33*(9), 1347–1354. https://doi.org/10.1038/jcbfm.2013.94

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence Limits for the Indirect Effect: Distribution of the Product and Resampling Methods. *Multivariate Behavioral Research*, *39*(1), 99–128. https://doi.org/10.1207/s15327906mbr3901_4

Maguire, E. A., Gadian, D. G., Johnsrude, I. S., Good, C. D., Ashburner, J., Frackowiak, R. S. J., & Frith, C. D. (2000). Navigation-related structural change in the hippocampi of taxi drivers. *Proceedings of the National Academy of Sciences of the United States of America*, *97*(8), 4398–4403.

Major, B., Quinton, W. J., & McCoy, S. K. (2002). Antecedents and consequences of attributions to discrimination: Theoretical and empirical advances. In *Advances in Experimental Social Psychology* (Vol. 34, pp. 251–330). Academic Press. https://doi.org/10.1016/S0065-2601(02)80007-7

Maldjian, J. A., Laurienti, P. J., Kraft, R. A., & Burdette, J. H. (2003). An automated method for neuroanatomic and cytoarchitectonic atlas-based interrogation of fMRI data sets. *NeuroImage*, *19*(3), 1233–1239. https://doi.org/10.1016/S1053-8119(03)00169-1

Mani, A., Mullainathan, S., Shafir, E., & Zhao, J. (2013). Poverty Impedes Cognitive Function. *Science*, *341*(6149), 976–980. https://doi.org/10.1126/science.1238041

Markus, H. R., & Kitayama, S. (1991). Culture and the self: Implications for cognition, emotion, and motivation. *Psychological Review*, *98*(2), 224–253. https://doi.org/10.1037/0033-295X.98.2.224

Marsland, A. L., Kuan, D. C.-H., Sheu, L. K., Krajina, K., Kraynak, T. E., Manuck, S. B., & Gianaros, P. J. (2017). Systemic inflammation and resting state connectivity of the default mode network. *Brain, Behavior, and Immunity*, *62*, 162–170. https://doi.org/10.1016/j.bbi.2017.01.013

Massey, D. S. (1990). American Apartheid: Segregation and the Making of the Underclass. *American Journal of Sociology*, *96*(2), 329–357.

Massey, D. S. (1996). The age of extremes: Concentrated affluence and poverty in the twenty-first century. *Demography*, *33*(4), 395–412; discussion 413-416.

Massey, D. S., & Denton, N. A. (1988). The Dimensions of Residential Segregation. *Social Forces*, *67*(2), 281–315. JSTOR. https://doi.org/10.2307/2579183

Massey, D. S., & Denton, N. A. (1989). Hypersegregation in U.S. Metropolitan Areas: Black and Hispanic Segregation Along Five Dimensions. *Demography*, *26*(3), 373–391. https://doi.org/10.2307/2061599

Massey, D. S., & Denton, N. A. (1993). *American Apartheid—Douglas S. Massey, Nancy A. Denton*. Harvard University Press. https://www.hup.harvard.edu/catalog.php?isbn=9780674018211

Massey, D. S., & Eggers, M. L. (1990). The Ecology of Inequality: Minorities and the Concentration of Poverty, 1970-1980. *American Journal of Sociology*, *95*(5), 1153–1188. https://doi.org/10.1086/229425

Massey, D. S., & Fischer, M. J. (2000). How segregation concentrates poverty. *Ethnic and Racial Studies*, *23*(4), 670–691. https://doi.org/10.1080/01419870050033676

Masuda, T., & Nisbett, R. E. (2001). Attending holistically versus analytically: Comparing the context sensitivity of Japanese and Americans. *Journal of Personality and Social Psychology*, *81*(5), 922–934. https://doi.org/10.1037/0022-3514.81.5.922

McCall, L., & Percheski, C. (2010). Income Inequality: New Trends and Research Directions. *Annual Review of Sociology*, *36*(1), 329–347. https://doi.org/10.1146/annurev.soc.012809.102541

McCarthy, J. D., & Yancey, W. L. (1971). Uncle Tom and Mr. Charlie: Metaphysical Pathos in the Study of Racism and Personal Disorganization. *American Journal of Sociology*, *76*(4), 648–672. https://doi.org/10.1086/224976

McKnight, P. E., & Kashdan, T. B. (2009). Purpose in Life as a System that Creates and Sustains Health and Well-Being: An Integrative, Testable Theory. *Review of General Psychology*, *13*(3), 242–251. https://doi.org/10.1037/a0017152

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience*, *24*, 1161–1192. https://doi.org/10.1146/annurev.neuro.24.1.1161

Meunier, D., Lambiotte, R., & Bullmore, E. T. (2010). Modular and Hierarchically Modular Organization of Brain Networks. *Frontiers in Neuroscience*, *4*. https://doi.org/10.3389/fnins.2010.00200

Miller, D. B., & MacIntosh, R. (1999). Promoting resilience in urban African American adolescents: Racial socialization and identity as protective factors. *Social Work Research*, *23*(3), 159–169. https://doi.org/10.1093/swr/23.3.159

Miyamoto, Y. (2017). Culture and social class. *Current Opinion in Psychology*, *18*, 67–72. https://doi.org/10.1016/j.copsyc.2017.07.042

Murray, C. (2012). *Coming Apart: The State of White America, 1960-2010*. Crown Publishing Group.

Muschelli, J., Nebel, M. B., Caffo, B. S., Barber, A. D., Pekar, J. J., & Mostofsky, S. H. (2014). Reduction of motion-related artifacts in resting state fMRI using aCompCor. *NeuroImage*, *96*, 22–35. https://doi.org/10.1016/j.neuroimage.2014.03.028

Na, J., & Kitayama, S. (2011). Spontaneous Trait Inference Is Culture-Specific: Behavioral and Neural Evidence. *Psychological Science*, *22*(8), 1025–1032. https://doi.org/10.1177/0956797611414727

Neblett, E. W., Philip, C. L., Cogburn, C. D., & Sellers, R. M. (2006). African American Adolescents' Discrimination Experiences and Academic Achievement: Racial Socialization as a Cultural Compensatory and Protective Factor. *Journal of Black Psychology*, *32*(2), 199–218. https://doi.org/10.1177/0095798406287072

Newman, M. E. J. (2006). Modularity and community structure in networks. *Proceedings of the National Academy of Sciences of the United States of America*, *103*(23), 8577–8582. https://doi.org/10.1073/pnas.0601602103

Nisbett, R. E., Peng, K., Choi, I., & Norenzayan, A. (2001). Culture and systems of thought: Holistic versus analytic cognition. *Psychological Review*, *108*(2), 291–310.

Northoff, G., & Bermpohl, F. (2004). Cortical midline structures and the self. *Trends in Cognitive Sciences*, *8*(3), 102–107. https://doi.org/10.1016/j.tics.2004.01.004

Nuru-Jeter, A. M., & LaVeist, T. A. (2011). Racial Segregation, Income Inequality, and Mortality in US Metropolitan Areas. *Journal of Urban Health*, *88*(2), 270–282. https://doi.org/10.1007/s11524-010-9524-7

Nusslock, R., Brody, G. H., Armstrong, C. C., Carroll, A. L., Sweet, L. H., Yu, T., Barton, A. W., Hallowell, E. S., Chen, E., Higgins, J. P., Parrish, T. B., Wang, L., & Miller, G. E. (2019). Higher Peripheral Inflammatory Signaling Associated With Lower Resting-State Functional Brain Connectivity in Emotion Regulation and Central Executive Networks. *Biological Psychiatry*, *86*(2), 153–162. https://doi.org/10.1016/j.biopsych.2019.03.968

O'Connor, M.-F., Bower, J. E., Cho, H. J., Creswell, J. D., Dimitrov, S., Hamby, M. E., Hoyt, M. A., Martin, J. L., Robles, T. F., Sloan, E. K., Thomas, K. S., & Irwin, M. R. (2009). To

assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain, Behavior, and Immunity*, *23*(7), 887–897. https://doi.org/10.1016/j.bbi.2009.04.005

O'Doherty, J. P. (2011). Contributions of the ventromedial prefrontal cortex to goal-directed action selection. *Annals of the New York Academy of Sciences*, *1239*, 118–129. https://doi.org/10.1111/j.1749-6632.2011.06290.x

Oishi, S., Kesebir, S., & Diener, E. (2011). Income Inequality and Happiness: *Psychological Science*. https://doi.org/10.1177/0956797611417262

Park, J., Kitayama, S., Markus, H. R., Coe, C. L., Miyamoto, Y., Karasawa, M., Curhan, K. B., Love, G. D., Kawakami, N., Boylan, J. M., & Ryff, C. D. (2013). Social status and anger expression: The cultural moderation hypothesis. *Emotion*, *13*(6), 1122–1131. https://doi.org/10.1037/a0034273

Pascoe, E. A., & Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, *135*(4), 531–554. https://doi.org/10.1037/a0016059

Pavlov, V. A., & Tracey, K. J. (2017). Neural regulation of immunity: Molecular mechanisms and clinical translation. *Nature Neuroscience*, *20*(2), 156–166. https://doi.org/10.1038/nn.4477

Pew Research Center. (2011, July 26). *Wealth Gaps Rise to Record Highs Between Whites, Blacks, Hispanics*. Pew Research Center's Social & Demographic Trends Project. https://www.pewsocialtrends.org/2011/07/26/wealth-gaps-rise-to-record-highs-between-whites-blacks-hispanics/

Pew Research Center. (2016a, June 27). *On Views of Race and Inequality, Blacks and Whites Are Worlds Apart*. Pew Research Center's Social & Demographic Trends Project. https://www.pewsocialtrends.org/2016/06/27/on-views-of-race-and-inequality-blacks-and-whites-are-worlds-apart/

Pew Research Center. (2016b, June 27). *On Views of Race and Inequality, Blacks and Whites Are Worlds Apart: Demographic trends and economic well-being*. https://www.pewsocialtrends.org/2016/06/27/1-demographic-trends-and-economic-well-being/

Pickett, K. E., Kelly, S., Brunner, E., Lobstein, T., & Wilkinson, R. G. (2005). Wider income gaps, wider waistbands? An ecological study of obesity and income inequality. *Journal of Epidemiology & Community Health*, *59*(8), 670–674. https://doi.org/10.1136/jech.2004.028795

Pittman, C. T. (2011). Getting Mad But Ending Up Sad: The Mental Health Consequences for African Americans Using Anger to Cope With Racism. *Journal of Black Studies*, *42*(7), 1106–1124. https://doi.org/10.1177/0021934711401737

Powell, L. M., Slater, S., Mirtcheva, D., Bao, Y., & Chaloupka, F. J. (2007). Food store availability and neighborhood characteristics in the United States. *Preventive Medicine*, *44*(3), 189–195. https://doi.org/10.1016/j.ypmed.2006.08.008

Power, J. D., Barnes, K. A., Snyder, A. Z., Schlaggar, B. L., & Petersen, S. E. (2012). Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *NeuroImage*, *59*(3), 2142–2154. https://doi.org/10.1016/j.neuroimage.2011.10.018

Power, J. D., Cohen, A. L., Nelson, S. M., Wig, G. S., Barnes, K. A., Church, J. A., Vogel, A. C., Laumann, T. O., Miezin, F. M., Schlaggar, B. L., & Petersen, S. E. (2011). Functional

network organization of the human brain. *Neuron*, *72*(4), 665–678. https://doi.org/10.1016/j.neuron.2011.09.006
Power, J. D., Mitra, A., Laumann, T. O., Snyder, A. Z., Schlaggar, B. L., & Petersen, S. E. (2014). Methods to detect, characterize, and remove motion artifact in resting state fMRI. *NeuroImage*, *84*, 320–341. https://doi.org/10.1016/j.neuroimage.2013.08.048
Quan, N., & Banks, W. A. (2007). Brain-immune communication pathways. *Brain, Behavior, and Immunity*, *21*(6), 727–735. https://doi.org/10.1016/j.bbi.2007.05.005
Quillian, L. (2012). Segregation and Poverty Concentration: The Role of Three Segregations. *American Sociological Review*, *77*(3), 354–379. https://doi.org/10.1177/0003122412447793
Reardon, S. F., & Bischoff, K. (2011). Income inequality and income segregation. *AJS; American Journal of Sociology*, *116*(4), 1092–1153. https://doi.org/10.1086/657114
Reuter-Lorenz, P. A., & Cappell, K. A. (2008). Neurocognitive Aging and the Compensation Hypothesis. *Current Directions in Psychological Science*, *17*(3), 177–182. https://doi.org/10.1111/j.1467-8721.2008.00570.x
Reuter-Lorenz, P. A., Jonides, J., Smith, E. E., Hartley, A., Miller, A., Marshuetz, C., & Koeppe, R. A. (2000). Age Differences in the Frontal Lateralization of Verbal and Spatial Working Memory Revealed by PET. *Journal of Cognitive Neuroscience*, *12*(1), 174–187. https://doi.org/10.1162/089892900561814
Rolls, E. T., & Grabenhorst, F. (2008). The orbitofrontal cortex and beyond: From affect to decision-making. *Progress in Neurobiology*, *86*(3), 216–244. https://doi.org/10.1016/j.pneurobio.2008.09.001
Rosenzweig, M. R. (1996). Aspects of the Search for Neural Mechanisms of Memory. *Annual Review of Psychology*, *47*(1), 1–32. https://doi.org/10.1146/annurev.psych.47.1.1
Rosenzweig, M. R., Krech, D., Bennett, E. L., & Zolman, J. F. (1962). Variation in environmental complexity and brain measures. *Journal of Comparative and Physiological Psychology*, *55*(6), 1092–1095. https://doi.org/10.1037/h0042758
Rubinov, M., & Sporns, O. (2010). Complex network measures of brain connectivity: Uses and interpretations. *NeuroImage*, *52*(3), 1059–1069. https://doi.org/10.1016/j.neuroimage.2009.10.003
Russo, M., & McGavern, D. B. (2015). Immune surveillance of the CNS following infection and injury. *Trends in Immunology*, *36*(10), 637–650. https://doi.org/10.1016/j.it.2015.08.002
Ryff, C. D., Keyes, C. L. M., & Hughes, D. L. (2003). Status Inequalities, Perceived Discrimination, and Eudaimonic Well-Being: Do the Challenges of Minority Life Hone Purpose and Growth? *Journal of Health and Social Behavior*, *44*(3), 275–291. https://doi.org/10.2307/1519779
Salvador, C. E., Berg, M. K., Yu, Q., San Martin, A., & Kitayama, S. (2020). Relational Mobility Predicts Faster Spread of COVID-19: A 39-Country Study. *Psychological Science*, *31*(10), 1236–1244. https://doi.org/10.1177/0956797620958118
Samson, D., Apperly, I. A., Chiavarino, C., & Humphreys, G. W. (2004). Left temporoparietal junction is necessary for representing someone else's belief. *Nature Neuroscience*, *7*(5), 499–500. https://doi.org/10.1038/nn1223
San Martin, A., Sinaceur, M., Madi, A., Tompson, S., Maddux, W. W., & Kitayama, S. (2018). Self-assertive interdependence in Arab culture. *Nature Human Behaviour*, *2*(11), 830–837. https://doi.org/10.1038/s41562-018-0435-z

Sapolsky, R. M. (1994). Glucocorticoids, stress and exacerbation of excitotoxic neuron death. *Seminars in Neuroscience*, *6*(5), 323–331. https://doi.org/10.1006/smns.1994.1041

Sasaki, J. Y., Kim, H. S., Mojaverian, T., Kelley, L. D. S., Park, I. Y., & Janušonis, S. (2013). Religion priming differentially increases prosocial behavior among variants of the dopamine D4 receptor (DRD4) gene. *Social Cognitive and Affective Neuroscience*, *8*(2), 209–215. https://doi.org/10.1093/scan/nsr089

Savani, K., Markus, H. R., & Conner, A. L. (2008). Let your preference be your guide? Preferences and choices are more tightly linked for North Americans than for Indians. *Journal of Personality and Social Psychology*, *95*(4), 861–876. https://doi.org/10.1037/a0011618

Savani, K., Rose Markus, H., Naidu, N. V. R., Kumar, S., & Berlia, N. (2010). What Counts as a Choice? U.S. Americans Are More Likely Than Indians to Construe Actions as Choices. *Psychological Science*, *21*(3), 391–398.

Saxe, R., & Kanwisher, N. (2003). People thinking about thinking people: The role of the temporo-parietal junction in "theory of mind." *NeuroImage*, *19*(4), 1835–1842. https://doi.org/10.1016/S1053-8119(03)00230-1

Schmitt, M. T., Branscombe, N. R., Postmes, T., & Garcia, A. (2014). The consequences of perceived discrimination for psychological well-being: A meta-analytic review. *Psychological Bulletin*, *140*(4), 921–948. https://doi.org/10.1037/a0035754

Schulz, A., Gravlee, C. C., Williams, D. R., Israel, B. A., Mentz, G., & Rowe, Z. (2006). Discrimination, Symptoms of Depression, and Self-Rated Health Among African American Women in Detroit: Results From a Longitudinal Analysis. *American Journal of Public Health*, *96*(7), 1265–1270. https://doi.org/10.2105/AJPH.2005.064543

Schurz, M., Radua, J., Aichhorn, M., Richlan, F., & Perner, J. (2014). Fractionating theory of mind: A meta-analysis of functional brain imaging studies. *Neuroscience & Biobehavioral Reviews*, *42*, 9–34. https://doi.org/10.1016/j.neubiorev.2014.01.009

Segerstrom, S. C., & Miller, G. E. (2004). Psychological Stress and the Human Immune System: A Meta-Analytic Study of 30 Years of Inquiry. *Psychological Bulletin*, *130*(4), 601–630. https://doi.org/10.1037/0033-2909.130.4.601

Sellers, R. M., Caldwell, C. H., Schmeelk-Cone, K. H., & Zimmerman, M. A. (2003). Racial identity, racial discrimination, perceived stress, and psychological distress among African American young adults. *Journal of Health and Social Behavior*, *44*(3), 302–317.

Sellers, R. M., & Shelton, J. N. (2003). The role of racial identity in perceived racial discrimination. *Journal of Personality and Social Psychology*, *84*(5), 1079–1092. https://doi.org/10.1037/0022-3514.84.5.1079

Sellers, R. M., Smith, M. A., Shelton, J. N., Rowley, S. A., & Chavous, T. M. (1998). Multidimensional model of racial identity: A reconceptualization of African American racial identity. *Personality and Social Psychology Review: An Official Journal of the Society for Personality and Social Psychology, Inc*, *2*(1), 18–39. https://doi.org/10.1207/s15327957pspr0201_2

Set, E., Saez, I., Zhu, L., Houser, D. E., Myung, N., Zhong, S., Ebstein, R. P., Chew, S. H., & Hsu, M. (2014). Dissociable contribution of prefrontal and striatal dopaminergic genes to learning in economic games. *Proceedings of the National Academy of Sciences of the United States of America*, *111*(26), 9615–9620. https://doi.org/10.1073/pnas.1316259111

Sheese, B. E., Voelker, P. M., Rothbart, M. K., & Posner, M. I. (2007). Parenting quality interacts with genetic variation in dopamine receptor D4 to influence temperament in

early childhood. *Development and Psychopathology*, *19*(4), 1039–1046. https://doi.org/10.1017/S0954579407000521

Silveira, P. P., Gaudreau, H., Atkinson, L., Fleming, A. S., Sokolowski, M. B., Steiner, M., Kennedy, J. L., Meaney, M. J., Levitan, R. D., & Dubé, L. (2016). Genetic Differential Susceptibility to Socioeconomic Status and Childhood Obesogenic Behavior: Why Targeted Prevention May Be the Best Societal Investment. *JAMA Pediatrics*, *170*(4), 359–364. https://doi.org/10.1001/jamapediatrics.2015.4253

Simons, R. L., Lei, M.-K., Beach, S. R. H., Barr, A. B., Simons, L. G., Gibbons, F. X., & Philibert, R. A. (2018). Discrimination, segregation, and chronic inflammation: Testing the weathering explanation for the poor health of Black Americans. *Developmental Psychology*, *54*(10), 1993–2006. https://doi.org/10.1037/dev0000511

Singelis, T. M. (1994). The measurement of independent and interdependent self-construals. *Personality and Social Psychology Bulletin*, *20*(5), 580–591. https://doi.org/10.1177/0146167294205014

Sommers, S. R., & Ellsworth, P. C. (2000). Race in the Courtroom: Perceptions of Guilt and Dispositional Attributions. *Personality and Social Psychology Bulletin*, *26*(11), 1367–1379. https://doi.org/10.1177/0146167200263005

Stepanikova, I., Bateman, L. B., & Oates, G. R. (2017). Systemic Inflammation in Midlife: Race, Socioeconomic Status, and Perceived Discrimination. *American Journal of Preventive Medicine*, *52*(1 Suppl 1), S63–S76. https://doi.org/10.1016/j.amepre.2016.09.026

Stephens, N. M., Markus, H. R., & Phillips, L. T. (2014). Social class culture cycles: How three gateway contexts shape selves and fuel inequality. *Annual Review of Psychology*, *65*, 611–634. https://doi.org/10.1146/annurev-psych-010213-115143

Stephens, N. M., Townsend, S. S. M., Markus, H. R., & Phillips, L. T. (2012). A cultural mismatch: Independent cultural norms produce greater increases in cortisol and more negative emotions among first-generation college students. *Journal of Experimental Social Psychology*, *48*(6), 1389–1393. https://doi.org/10.1016/j.jesp.2012.07.008

Sun, Y., Danila, B., Josić, K., & Bassler, K. E. (2009). Improved community structure detection using a modified fine-tuning strategy. *EPL (Europhysics Letters)*, *86*(2), 28004. https://doi.org/10.1209/0295-5075/86/28004

Suzuki, B. H. (1977). Education and the Socialization of Asian Americans: A Revisionist Analysis of the "Model Minority" Thesis. *Amerasia Journal*, *4*(2), 23–51. https://doi.org/10.17953/amer.4.2.x203l74863857108

Tang, Y., Zhao, L., Lou, Y., Shi, Y., Fang, R., Lin, X., Liu, S., & Toga, A. (2018). Brain structure differences between Chinese and Caucasian cohorts: A comprehensive morphometry study. *Human Brain Mapping*, *39*(5), 2147–2155. https://doi.org/10.1002/hbm.23994

Thayer, J. F., & Sternberg, E. M. (2010). Neural aspects of immunomodulation: Focus on the vagus nerve. *Brain, Behavior, and Immunity*, *24*(8), 1223–1228. https://doi.org/10.1016/j.bbi.2010.07.247

Tompson, S. H., Huff, S. T., Yoon, C., King, A., Liberzon, I., & Kitayama, S. (2018). The dopamine D4 receptor gene (DRD4) modulates cultural variation in emotional experience. *Culture and Brain*, *6*(2), 118–129. https://doi.org/10.1007/s40167-018-0063-5

Tosh, C. R., & McNally, L. (2015). The relative efficiency of modular and non-modular networks of different size. *Proceedings of the Royal Society B: Biological Sciences*, *282*(1802), 20142568. https://doi.org/10.1098/rspb.2014.2568

Triandis, H. C. (1989). The self and social behavior in differing cultural contexts. *Psychological Review*, *96*(3), 506–520. https://doi.org/10.1037/0033-295X.96.3.506

Tsai, J. L., Knutson, B., & Fung, H. H. (2006). Cultural variation in affect valuation. *Journal of Personality and Social Psychology*, *90*(2), 288–307. https://doi.org/10.1037/0022-3514.90.2.288

Uchiyama, R., Spicer, R., & Muthukrishna, M. (2021). Cultural Evolution of Genetic Heritability. *Behavioral and Brain Sciences*, 1–147. https://doi.org/10.1017/S0140525X21000893

U.S. Bureau of Labor Statistics. (2019). *Job Flexibilities and Work Schedules News Release*. https://www.bls.gov/news.release/flex2.htm

U.S. Census Bureau. (2002). *Racial and Ethnics Residential Segregation in the U.S.: 1980-2000*. https://www.census.gov/library/publications/2002/dec/censr-3.html

U.S. Census Bureau. (2020). *Metropolitan and Micropolitan*. https://www.census.gov/programs-surveys/metro-micro/about.html

Usher, T., Gaskin, D. J., Bower, K., Rohde, C., & Thorpe, R. J. (2018). Residential Segregation and Hypertension Prevalence in Black and White Older Adults. *Journal of Applied Gerontology*, *37*(2), 177–202. https://doi.org/10.1177/0733464816638788

van Zomeren, M., Postmes, T., & Spears, R. (2008). Toward an integrative social identity model of collective action: A quantitative research synthesis of three socio-psychological perspectives. *Psychological Bulletin*, *134*(4), 504–535. https://doi.org/10.1037/0033-2909.134.4.504

Varnum, M. E. W., Shi, Z., Chen, A., Qiu, J., & Han, S. (2014). When "Your" reward is the same as "My" reward: Self-construal priming shifts neural responses to own vs. friends' rewards. *NeuroImage*, *87*, 164–169. https://doi.org/10.1016/j.neuroimage.2013.10.042

Voigt, R., Camp, N. P., Prabhakaran, V., Hamilton, W. L., Hetey, R. C., Griffiths, C. M., Jurgens, D., Jurafsky, D., & Eberhardt, J. L. (2017). Language from police body camera footage shows racial disparities in officer respect. *Proceedings of the National Academy of Sciences*, *114*(25), 6521–6526. https://doi.org/10.1073/pnas.1702413114

Vukasović, T., & Bratko, D. (2015). Heritability of personality: A meta-analysis of behavior genetic studies. *Psychological Bulletin*, *141*(4), 769–785. https://doi.org/10.1037/bul0000017

Wang, E., Ding, Y.-C., Flodman, P., Kidd, J. R., Kidd, K. K., Grady, D. L., Ryder, O. A., Spence, M. A., Swanson, J. M., & Moyzis, R. K. (2004). The Genetic Architecture of Selection at the Human Dopamine Receptor D4 (DRD4) Gene Locus. *American Journal of Human Genetics*, *74*(5), 931–944.

Wang, F., Peng, K., Chechlacz, M., Humphreys, G. W., & Sui, J. (2017). The Neural Basis of Independence Versus Interdependence Orientations: A Voxel-Based Morphometric Analysis of Brain Volume. *Psychological Science*, *28*(4), 519–529. https://doi.org/10.1177/0956797616689079

Weaver, J. D., Huang, M.-H., Albert, M., Harris, T., Rowe, J. W., & Seeman, T. E. (2002). Interleukin-6 and risk of cognitive decline: MacArthur studies of successful aging. *Neurology*, *59*(3), 371–378. https://doi.org/10.1212/wnl.59.3.371

Weisz, J. R., Rothbaum, F. M., & Blackburn, T. C. (1984). Standing out and standing in: The psychology of control in America and Japan. *American Psychologist*, *39*(9), 955–969. https://doi.org/10.1037/0003-066X.39.9.955

Whitfield-Gabrieli, S., & Nieto-Castanon, A. (2012). Conn: A Functional Connectivity Toolbox for Correlated and Anticorrelated Brain Networks. *Brain Connectivity*, *2*(3), 125–141. https://doi.org/10.1089/brain.2012.0073

Williams, D. R., & Collins, C. (1995). US Socioeconomic and Racial Differences in Health: Patterns and Explanations. *Annual Review of Sociology*, *21*, 349–386.

Williams, D. R., & Collins, C. (2001). Racial Residential Segregation: A Fundamental Cause of Racial Disparities in Health. *Public Health Reports*, *116*, 13.

Williams, D. R., Lawrence, J. A., & Davis, B. A. (2019). Racism and Health: Evidence and Needed Research. *Annual Review of Public Health*, *40*(1), 105–125. https://doi.org/10.1146/annurev-publhealth-040218-043750

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, *32*(1), 20–47. https://doi.org/10.1007/s10865-008-9185-0

Williams, D. R., Neighbors, H. W., & Jackson, J. S. (2003). Racial/Ethnic Discrimination and Health: Findings From Community Studies. *American Journal of Public Health*, *93*(2), 200–208. https://doi.org/10.2105/AJPH.93.2.200

Wilson, W. J. (2012). *The Truly Disadvantaged: The Inner City, the Underclass, and Public Policy, Second Edition*. University of Chicago Press.

Wu, S., & Keysar, B. (2007). The Effect of Culture on Perspective Taking. *Psychological Science*, *18*(7), 600–606. https://doi.org/10.1111/j.1467-9280.2007.01946.x

Ye, M., Yang, T., Qing, P., Lei, X., Qiu, J., & Liu, G. (2015). Changes of Functional Brain Networks in Major Depressive Disorder: A Graph Theoretical Analysis of Resting-State fMRI. *PLoS ONE*, *10*(9). https://doi.org/10.1371/journal.pone.0133775

Young, L., Camprodon, J. A., Hauser, M., Pascual-Leone, A., & Saxe, R. (2010). Disruption of the right temporoparietal junction with transcranial magnetic stimulation reduces the role of beliefs in moral judgments. *Proceedings of the National Academy of Sciences*, *107*(15), 6753–6758. https://doi.org/10.1073/pnas.0914826107

Yu, Q., Abe, N., King, A., Yoon, C., Liberzon, I., & Kitayama, S. (2019). Cultural variation in the gray matter volume of the prefrontal cortex is moderated by the dopamine D4 receptor gene (DRD4). *Cerebral Cortex*, *29*(9), 3922–3931. https://doi.org/10.1093/cercor/bhy271

Zajacova, A., & Lawrence, E. M. (2018). The relationship between education and health: Reducing disparities through a contextual approach. *Annual Review of Public Health*, *39*, 273–289. https://doi.org/10.1146/annurev-publhealth-031816-044628

Zalesky, A., Fornito, A., Cocchi, L., Gollo, L. L., van den Heuvel, M. P., & Breakspear, M. (2016). Connectome sensitivity or specificity: Which is more important? *NeuroImage*, *142*, 407–420. https://doi.org/10.1016/j.neuroimage.2016.06.035

Zenk, S., Schulz, A., Israel, B., James, S., Bao, S., & Wilson, M. (2005). Neighborhood racial composition, neighborhood poverty, and the spatial accessibility of supermarkets in metropolitan Detroit. *American Journal of Public Health*. https://doi.org/10.2105/AJPH.2004.042150

Zhu, Y., Zhang, L., Fan, J., & Han, S. (2007). Neural basis of cultural influence on self-representation. *NeuroImage*, *34*(3), 1310–1316. https://doi.org/10.1016/j.neuroimage.2006.08.047