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Effect of Neighborhood Socioeconomic Disadvantage on Brain Functional Connectivity and Structural Properties in Trauma-Exposed Adults

Elisabeth Kathleen Webb
University of Wisconsin-Milwaukee

EFFECT OF NEIGHBORHOOD SOCIOECONOMIC DISADVANTAGE ON BRAIN FUNCTIONAL CONNECTIVITY AND STRUCTURAL PROPERTIES IN TRAUMA-EXPOSED ADULTS

by

Elisabeth Kathleen Webb

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ABSTRACT

EFFECTS OF NEIGHBORHOOD SOCIOECONOMIC DISADVANTAGE ON FUNCTIONAL AND STRUCTURAL PROPERTIES IN TRAUMA-EXPOSED ADULTS

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The University of Wisconsin-Milwaukee, 2021
Under the Supervision of Professor Christine L. Larson

Though there has been substantial progress towards understanding brain-behavior relationships and characterizing the neurobiology of psychiatric disorders, research has not translated as expected into novel prevention and treatment of mental health conditions. One limitation may be the emphasis on individual-level variables (e.g., income) and omission of relevant area-level factors (e.g., neighborhood socioeconomic disadvantage). Recently, attention has been directed towards identifying the biological mechanisms by which neighborhoods impact mental health. The chronic stress associated with living in a disadvantaged neighborhood promotes a cascade of maladaptive events, which in turn impact brain structure and functioning. The processes affected by chronic neighborhood stressors are likewise induced when an individual experiences an acute trauma. This provides a basis for the psychological consequences of trauma, such as post-traumatic stress disorder, to be worsened or maintained by an individual's neighborhood. More explicitly, where an individual lives may be intrinsically related to their recovery after a trauma.

In a sample of over two hundred traumatically injured participants, these projects sought to identify associations between neighborhood disadvantage and brain structure and function. Each project's analysis included a risk-resilience model, exploring

interactions between socioeconomic variables and resilience factors. In the first project, I demonstrated Area Deprivation Index (ADI) rankings, a measure of neighborhood socioeconomic disadvantage, were significantly related to white matter tract integrity. Ethnoracially (ethnically/racially) minoritized individuals disproportionately reside in disadvantaged neighborhoods, and a culturally relevant resilience factor, racial-ethnic identity, buffered against the effects of ADI.

In the second project, I investigated the effects of ADI on resting-state functional connectivity and structural volume of a region subserving emotion regulation processes, the anterior cingulate cortex (ACC). Using a seed-to-voxel analysis, I demonstrated lower income, but not ADI, was significantly associated with greater connectivity between the ACC and visual regions. In the final neuroethics project, I highlighted the necessity of research on area-level factors, the ethical implications of discoveries on neighborhood-mental health pathways, and the importance of devising informed policies. Ultimately, this dissertation provides additional support that sociopolitical factors represent important “missing pieces” of neuroscience research and that studies on these factors are essential in the path towards health equity.

“I am of the opinion that my life belongs to the whole community, and as long as I live, it is my privilege to do for it whatsoever I can.”

– *George Bernard Shaw*

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LIST OF ABBREVIATIONS

ACC	anterior cingulate cortex
ADI	area deprivation index (national ranking)
ATR	anterior thalamic radiations
CAB	cingulum angular bundles
CAPS-5	clinician-administered PTSD scale for DSM-5
CCG	cingulum-cingulate gyrus bundles
CST	corticospinal tract
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, 5th edition
DTI	diffusion tensor imaging
DWI	diffusion weighted images
FA	fractional anisotropy
FDR	false discovery rate
FMAJOR	forceps major
FMINOR	forceps minor
fMRI	functional magnetic resonance imaging
GLM	general linear model
HPA	hypothalamic-pituitary-adrenal (axis)
iSTAR	imaging study of trauma and resilience
MEIM	multigroup ethnic identity measure
MOS	medical outcome study social support survey
MMR	multivariate multiple regression

PCL-5	PTSD Checklist for DSM-5
PTSD	post-traumatic stress disorder
SEP	socioeconomic position
SLFP	superior longitudinal fasciculus parietal bundle
SLFT	superior longitudinal fasciculus temporal bundle
T1	time-point 1
T2	time-point 2
UNC	uncinate fasciculus

ACKNOWLEDGMENTS

This dissertation is dedicated to those who were declined metal health services or slipped through the cracks of the American healthcare system that was never designed to help them. In the weeks leading up to my dissertation defense, my 7th friend died by suicide, the 3rd with the same diagnosis as mine. At every stage, I've reflected about why I pursued this line of work. My current conclusion is that I do this work because I carry the stories of those who have struggled and fought, and these stories steer my scientific curiosity. Some people's stories are starkly different from my own, others have similar themes; regardless, I view my scholarship as a form of activism and storytelling. I try to conduct research that honors the loved ones we have lost and those still struggling. I've been able to do this work with support from mentors, friends, family who have kept my path illuminated, even during the darkest times.

No victory in academia (or in life I would argue) is the product of one person alone. I have been encouraged, taught, and loved by so many. In this sense, even my dissertation is not mine (although if you find a typo the responsibility rests with me). Although I would have liked to include everyone I've ever met in this section (and even those I haven't, including Lin-Manuel Miranda and Rihanna), for brevity, I'll highlight a select few.

First, I would like to acknowledge the participants who gave their time and trust. At its core, research is storytelling and I'm honored to have had the opportunity to tell the tale of people's resilience. I'm often overwhelmed by the richness and complexity of life, and although that no single study (no matter how many surveys) will ever fully capture a persons' *being*, I am fortunate to have had the opportunity to learn about the

many facets of people's lives. The attempt to understand the human experience - with the ultimate goal of improving wellbeing - is central to the Milwaukee Trauma Outcomes Project (MTOP). I am grateful to have been part of a team that continually sought improvement, discussed shortcomings, and created an environment where questioning was always encouraged. Thank you to the faculty members of MTOP - Lucas, Terri, and Chris - for creating an intellectual space to engage in collaborative and forward-thinking research.

I would have never found my path to research without the guidance of many mentors before graduate school. I was always fascinated by science, but also by English, art, and philosophy. Mentoring me was probably a bit like herding cats and yet each advisor was patient; truthful and generous with advice, and considerate of the adversity in my personal life. In my undergraduate training at Albion College, Dr. Mary Collar, Dr. Barbara Keyes, and Dr. Jeff Wilson embraced my love of knowledge and showed me the different paths I could follow to chase it. Jeff introduced me to neuroscience, teaching me the terms "axon" and "neurotransmitter", just weeks before releasing me into the world of research at the annual Society for Neuroscience meeting. Jeff's mentorship and impact on my career trajectory has been extensive; but it is his friendship that is perhaps most valued.

Even with a love of neuroscience, I was missing something (the elusive "something" that spurs doubt, and hours of rabbit-hole google searching). I needed meaning in my life; to invest everything into an idea that would continually excite and drive me. Through Jeff, I met dozens of talented rats and the mighty Dr. Cindy Fast (APOPO)! Cindy showed me how to be dedicated and devoted to research, but also the

struggles of being a woman in science faced with higher expectations and fewer recognitions. Her lessons on communication, research design, and grantsmanship, have been useful and relevant every day. Cindy fueled my love of behavioral neuroscience with late night chats about learning and memory experiments with hermit crabs, pigeons, and rats. To the entire APOPO team, particularly Dian, Jia Hui, Bakari, and Iyungu – thank you for making every moment, from chai time to lab meetings, joyful.

Chris' lab was a safe haven (quite literally). I arrived in Milwaukee soon after a close colleague had died by suicide, still grieving, and stumbling with shock. The love, acceptance, and care I felt from my lab was immediate and overwhelming. Ashley, Ken, Richard, Liz, Jacklynn (at the time a post-doc) and Carissa, thank you for welcoming me into the lab, sitting with me as I learned the foundations of neuroimaging, and engaging in the camaraderie that makes a good lab environment, great. A special thanks to Carissa, who read every word of my papers (including this one), agreed to (nearly all) my crazy ideas, pushed me to be better and do better, and even let me live in her house! I didn't burn out in graduate school because I respected and loved the people I worked with, and this was reciprocated.

Outside of school, my Nana and Daddad provide stability and encouragement. Every phone call for the last couple months included the question "what page are you on now?", and a "you can do it!" exclamation. The love my grandparents show me feels so rare, magical, and somehow infinite – and I'm better because of it. My world revolves around my brother and sister; our dreams and hopes have become shared, intertwining,

entities. Each sibling's accomplishment is a victory for the three of us and this dissertation is just another testament to that.

My last person (and favorite) to acknowledge is Jamie. When we decided to get married while I was still in undergrad, Jamie's one request was that I would continue with my education and pursue a doctoral degree. And here we are. Someone once told me that the biggest mistake they saw graduate students make was putting their life on hold; assuming that their life would really begin once school ended. With Jamie, life happened every single day and grad school happened around our very full life. Each paper I wrote, exam I took, class I taught, is defined in my mind by other (arguably more important) event: a trip to the lake, a taco night, a film festival, a birthday, a walk on the Oakleaf trail. Jamie has been a partner in navigating my career, my struggles, and my joy. When I speak of friends and family illuminating my path, it is his light that has been the brightest. To Jamie – I'm glad I could finally make your dream come true: a lifetime of making dinner reservations for Mr. and Dr. Webb!

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Dissertation Overview

The objective of this dissertation was to comprehensively analyze how neighborhood socioeconomic disadvantage affects neurobiology and to evaluate two potential mechanisms linking neighborhood disadvantage to post-trauma outcomes. The document is formatted in portfolio-style, such that each project was written to be a stand-alone manuscript with its own introduction, methods, results, and discussion. The following is a brief overview of the central theory imbued in this work, the specific projects, and the overall significance.

The socioeconomic position (SEP) of an individual, or a neighborhood, is defined by complex and malleable interactions between societal norms, policies, and systems of power. As researchers, we use quantifiable proxies of socioeconomic positions such as individual income or concentrated neighborhood socioeconomic disadvantage, to test associations between these constructs and health (Galobardes et al., 2006). Socioeconomic circumstances, whether at the individual, neighborhood, or national level, are inherently difficult to conceptualize and operationally define due to their multi-dimensional nature. In this document, I was specific with my use of language. For example, I used “socioeconomic circumstances” to reference both neighborhood-level and individual-level indicators. When a socioeconomic factor was specific to either the neighborhood or individual, it was indicated as such.

None of the cited socioeconomic indicators operate in isolation from each other, or in isolation from societal and political forces. For this reason, I situated all the proposed work in the epidemiological ecosocial theory of health, initially proposed by Nancy Krieger (1994; 2001), which links individual health outcomes to interactions

between systems of power (e.g., socio-political, and economic structures) *and* individual characteristics (e.g., biology, lived experiences, etc.). These pathways can be shaped by time (e.g., inter-generational, across the lifespan, etc.) and space (e.g., community factors, access to natural infrastructure, etc.; Krieger, 1994, 2001; Krieger et al., 2012). Critically, the ecosocial theory places emphasis on agency and accountability; unambiguously stating the role governments play in patterns of disease distribution. This is particularly applicable to this dissertation as neighborhoods have been radically and purposefully designed by federal and state laws (discussed in detail in Project 3).

In the United States, the distribution of neighborhood disadvantage within the population is not random. Exposure to neighborhood disadvantage (i.e., duration and magnitude) and the strength of the neighborhood-mental health association varies across people and places, with disparities directly attributed to structural racism[1]. The explicit racialization of local contexts (i.e., neighborhoods) has occurred through racist policies, such as the systematic underfunding of public resources (e.g., schools) in majority Black and Brown neighborhoods, the denial of housing loans based on skin color, and inequitable land use planning (e.g., placement of factories, highways, parks; Riley, 2018). The racialization of contexts, ethnoracial (i..e, racial/ethnic) differences in socioeconomic circumstances (e.g., Race x Class interactions), and individual experiences (e.g., racial differences in types of trauma exposure, experiences of discrimination), may all help explain mental health disparities (Berger & Sarnyai, 2015; Williams, 2018).

[1] I borrow Gee and Ford's (2011) definition, which identifies structural racism as "macro-level systems, social forces, institutions, ideologies, and processes that interact with one another to generate and reinforce inequalities among racial and ethnic groups."

As I review, scholars have documented that neighborhood socioeconomic disadvantage is associated with symptoms or diagnostic status of nearly all mental health conditions (see Diez Roux & Mair, 2010). I focused on post-trauma outcomes, specifically post-traumatic stress disorder (PTSD), for two key reasons. First, PTSD represents the only psychiatric condition for which the cause of the disorder, a traumatic event, is known. In the context of neighborhood research, this provides, to some degree, a temporal separation between risk factors and symptom development. Though this dissertation did not assess individuals prior to the traumatic event, participants were recruited in the early aftermath of a traumatic injury. The second rationale is that the neurobiological correlates of PTSD significantly overlap with the neurobiological features attributed to less advantaged socioeconomic circumstances (reviewed in Project 1 and 2). This presented an opportunity to explore whether neighborhood disadvantage, which may be uniquely associated with alterations to brain structure and function, could be related to a neural vulnerability to PTSD. Alternatively, previous work on neurobiology of socioeconomic circumstances may have been capturing the effects of acute trauma exposure, which individuals in less advantageous socioeconomic positions experience at disproportionately high rates (reviewed in Cubbin & Smith, 2002). Notably, all of the participants represented in this dissertation experienced a traumatic injury; a form of trauma with the highest incidence rates in the most disadvantaged neighborhoods (Loberg et al., 2018; Zarzaur et al., 2010).

I approached the dissertation topic with a multifaceted and multimodal analytic strategy. Each project tackled a distinct research question related to neighborhood factors and the brain. The empirical studies (Project 1 and 2) characterized the effects

of socioeconomic circumstances in both structural and functional neuroimaging data. In both projects, the first aim established a relationship between neighborhood disadvantage and neurobiology. In exploratory aims, I investigated whether the association between neighborhood disadvantage and neurobiology was moderated by resilience factors, including measures of social support and multigroup ethnic identity. Finally, I tested whether the relationship between neighborhood and the brain predicted non-remitting PTSD symptoms.

Project One interrogated whether neighborhood socioeconomic disadvantage could help explain differences in white matter tract integrity. Broadly, tract integrity, indexed by fractional anisotropy (FA), is conceptualized as a measure of how efficiently the brain processes information (Paus, 2010; Penke et al., 2012). Based on previous work I expected greater neighborhood disadvantage would be related to lesser integrity in the cingulum angular bundles and cingulum-cingulate gyrus bundles, two tracts with documented susceptibility to various forms of chronic stress and relationships to psychopathology (e.g., Kim et al., 2005; Rolls et al., 2019). Moreover, I anticipated a resilience factor, the multigroup ethnic identity measure, would significantly moderate the relationship between neighborhood disadvantage and white matter tract integrity, such that individuals living in more disadvantaged neighborhoods with greater affiliation to their ethnoracial group would have greater FA values.

In **Project Two,** I evaluated whether neighborhood disadvantage was related to resting-state functional connectivity of the anterior cingulate cortex (ACC) and if two resilience factors, (1) general social support and (2) affiliation to one's ethnoracial group would buffer against the effects of neighborhood disadvantage. The ACC plays a critical

role in emotion regulation and represents a “relay station” between cognitive and emotion processes (Stevens et al., 2011). As a region with strong anatomical and functional connections to sub-cortical regions underlying affective responding (e.g., amygdala), the ACC has been proposed as a top-down meditator of self-regulation in PTSD and a key correlate of resilience (Roeckner et al., 2021; Zweerings et al., 2018). Previous work has demonstrated PTSD symptoms are related to altered resting-state functional connectivity of the ACC (Kennis et al., 2015; Young et al., 2018). Despite evidence that neighborhood disadvantage is related to emotion dysregulation, the few studies probing the effects of neighborhood disadvantage on ACC connectivity have yielded mixed results (reviewed in detail in Project 2; c.f., Gard et al., 2021; Whittle et al., 2017). I hypothesized there would be a significant relationship between neighborhood disadvantage and ACC functional connectivity and that the two resilience factors would protect against the detrimental effects of both individual and neighborhood-level low SEP.

Although underlying drivers of health disparities are not frequently discussed in neuroscience research, I firmly dedicated **Project Three** to discussing the ethical implications of studies on neighborhood factors and how our understanding is hindered if the societal forces driving inequities, specifically structural racism, are not considered. In the final project, I situated research on the biological impact of neighborhoods within the historical policies which have shaped neighborhoods. I concluded by envisioning how this work can be used to inform policies and shape the future directions of psychological research. Improving post-trauma outcomes and narrowing health inequities will require committed, collaborative, and ingenious work. My goal is that this

dissertation helps cultivate future work and improve prevention and treatment of psychiatric disorders for *all* people.

Neighborhood Disadvantage Explains Differences in White Matter Tract Integrity and Racial-Ethnic Identity Buffers Negative Effects

Neighborhoods are comprised of distinct physical, social, and political aspects which shape individuals' behaviors, physical health, and psychological wellbeing (Diez Roux & Mair, 2010; Kind & Buckingham, 2018; Needham et al., 2014). Beyond the effects of individual factors, where a person resides plays an undeniable role in their mental health. For example, living in socioeconomically distressed or disadvantaged neighborhoods is associated with higher rates of depression (Alegría et al., 2014; Galea et al., 2007; Santiago et al., 2011), anxiety (Alegría et al., 2014; Casciano & Massey, 2012; Santiago et al., 2011), and post-traumatic stress disorder (PTSD; Johns et al., 2012). People who identify as a member of an ethnoracially (i.e., racially/ethnically) marginalized group disproportionally reside in disadvantaged neighborhoods (Williams et al., 2019). Ethnoracial segregation is a pervasive mechanism of structural racism and lays the foundation of differential exposure to neighborhood disadvantage (see Riley, 2018; Sewell, 2016; Williams et al., 2019). Consequently, neighborhood disadvantage is yet another racialized risk factor and driver of ethnoracial mental health disparities (Harnett & Ressler, 2021).

The Neurobiology of Neighborhood Disadvantage

There are a number of pathways by which neighborhood disadvantage affects mental health, including by directly influencing an individual's ability to access resources (e.g., healthy food, educational opportunities, natural infrastructure, healthcare; Camara Phyllis Jones et al., 2009; Diez Roux & Mair, 2010; Lurie & Dubowitz, 2007; Mennis et al., 2018; Nicole, 2018; Pun et al., 2018). In addition, living in more disadvantaged

neighborhoods is associated with greater exposure to chronic stressors (e.g., community violence, social disorganization) and thus heightened self-reported stress levels (Baglivio et al., 2017; Garo et al., 2018; Johns et al., 2012). Though studies have demonstrated that neighborhood disadvantage and related indicators bestow risk for poorer mental health outcomes, identifying specific pathways has been challenging as many biological systems appear to be affected.

A well-supported theory proposes the chronic stress produced by the conditions of neighborhood disadvantage "hijacks" the body's adaptive stress responses (McEwen & Gianaros, 2010; Richter-Levin & Sandi, 2021). More specifically, exposure to chronic stress elicits complex neuronal-neuroendocrine-immune responses that, over time, become maladaptive or toxic to the brain (McEwen, 2004; Richter-Levin & Sandi, 2021; Schulz et al., 2012). An important stress-related physiological process is the cascade of the hypothalamic-pituitary-adrenal (HPA) axis (Frodl & O'Keane, 2013; McEwen, 2000a, 2012; Richter-Levin & Sandi, 2021; Smith & Vale, 2006). The various stressful encounters associated with living in a disadvantaged neighborhood can activate the hypothalamus which promotes pituitary gland release of the adrenocorticotropic hormone via corticotropin releasing hormone (depicted in Figure 1.1). Downstream, the adrenal cortex releases glucocorticoids, including cortisol. In the absence of chronic exposure to stress, these allostatic processes, including negative feedback loops, are adaptive and ensure the appropriate response to acute stressors (e.g., heightened attention during test-taking; McEwen, 2000b); however, when the stressor is pervasive, erratic, and uncontrollable – such as the stressors associated with neighborhood

disadvantage (Finegood et al., 2017; Karb et al., 2012) – these processes are unable to appropriately regulate.

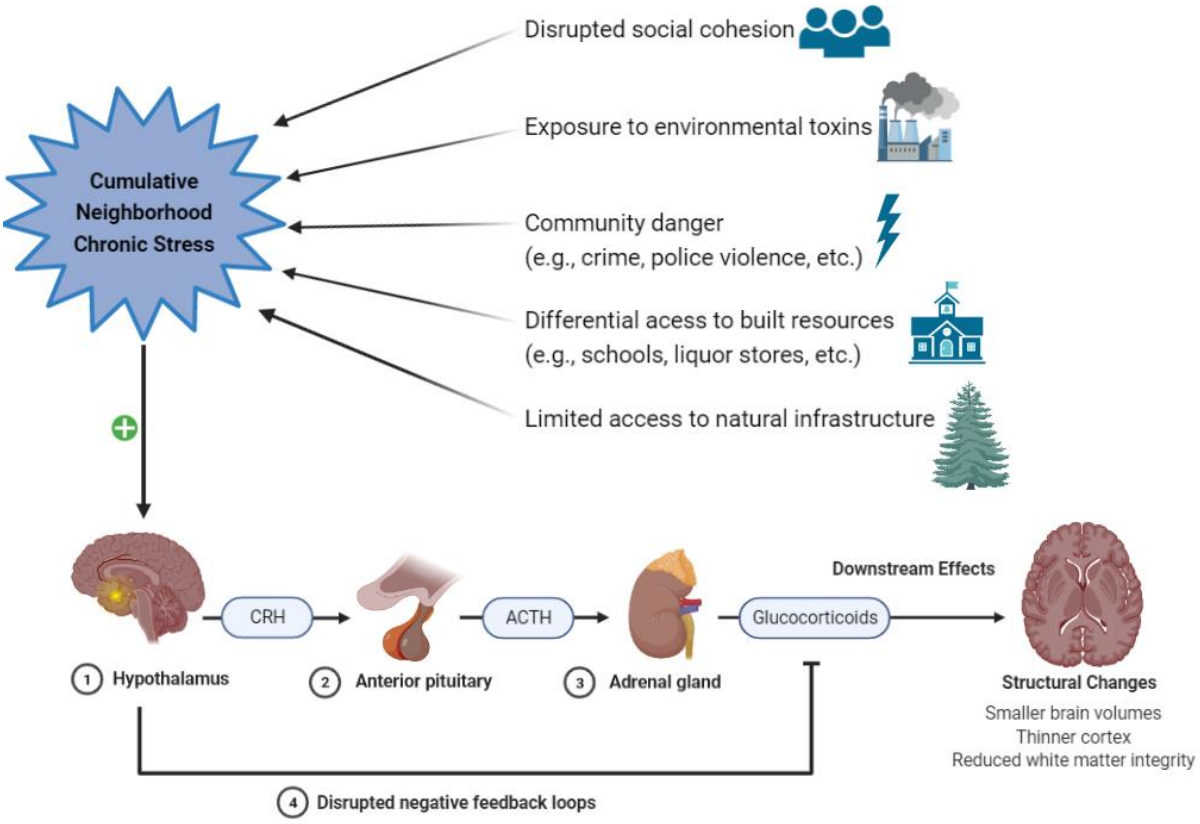

Figure 1.1. **A schematic depicting how neighborhood stressors elicit activation of the hypothalamic-pituitary-adrenal (HPA) axis.** Various stressors, associated with living in a socioeconomically disadvantaged neighborhood, **(1)** activate the hypothalamus to secrete corticotropin releasing hormone (CRH). Subsequently, the **(2)** anterior pituitary gland releases adrenocorticotropic hormone (ACTH) which acts on the **(3)** adrenal gland to produce glucocorticoids. **(4)** The negative feedback loop which would adaptively prevent persistent activation of this pathway is disrupted under conditions of chronic stress. The downstream effects of heightened HPA axis activity can

evoke structural changes, indexed by smaller brain volumes, thinner global cortex, and reduced white matter tract integrity. *Figure created with Biorender.*

Across preclinical and human studies, irregular cortisol levels, an index of HPA axis activity, are associated with altered brain structure (McEwen & Gianaros, 2010). As further evidence that neighborhood disadvantage tracks with neurobiological correlates of chronic stress, neighborhood disadvantage is associated with blunted cortisol reactivity to a stressor and elevated baseline cortisol levels (Barrington et al., 2014; Finegood et al., 2017). The majority of work has raised questions about socioeconomic circumstances – neighborhood-level or otherwise – and neurobiology in adolescent samples (for reviews see: Brito & Noble, 2014; Farah, 2017; Johnson et al., 2016; Noble et al., 2012). Still, the trends observed in adolescences appear to extend into adulthood. In adults, greater neighborhood disadvantage is associated with smaller brain structures (e.g., hippocampus, ventromedial prefrontal cortex; Hunt et al., 2020; Webb et al., 2021), total brain volume (Hunt et al., 2020a), and reduced cortical thickness (Gianaros et al., 2015; Hunt et al., 2020b).

Racial-Ethnic Identity as a Resilience Factor

In the literature on adult brain health and socioeconomic circumstances, resilience factors are glaringly absent, especially in the context of ethnoracial mental health disparities. The majority of the adult neuroscience literature offers only deficit models and applies a risk lens; the presence and power of resilience factors is under-acknowledged and under-studied (Yosso, 2005; c.f., James et al., 2012; Lee et al., 2021, 2021; Molesworth et al., 2015; Serra et al., 2015). Risk-models are useful as they

can help pinpoint the types of adversity, individual differences, and structural inequities that contribute to psychiatric diseases; however, resilience models offer an opportunity to identify which factors to invest in, not just eliminate. Given the intersections of race, ethnicity, and neighborhood disadvantage, the absence of resilience factors from germane studies is a noteworthy disservice to the marginalized groups who are economically exploited (Yosso, 2005).

As previously documented, the effects of neighborhood disadvantage (i.e., altered brain structure and function) may kindle neurobiological vulnerability for psychopathology (Harnett et al., 2019; Hunt et al., 2020; Hunt et al., 2020; Webb et al., 2021). Nevertheless, not everyone who lives in disadvantaged neighborhoods develop poor mental health, pointing to effects of protective factors. For example, in children, positive parenting buffers against negative effects of neighborhood disadvantage on brain function (Whittle et al., 2017). Resilience factors may have robust effects across ethnoracial groups or be especially salient for certain groups. Racial-ethnic identity, characterized as one's affiliation and sense of belonging to one's own ethnoracial group, is considered a predictor of psychological well-being (Lardier Jr. et al., n.d.; Nikulina et al., 2019). Black, Native, Hispanic, Latino, and Asian Americans endorse a greater sense of belonging and stronger commitment to their respective ethnoracial group compared to White counterparts (Goodstein & Ponterotto, 1997; Yancey et al., 2001). Higher scores on measures of racial-ethnic identity are associated with fewer symptoms of depression and anxiety (Walker et al., 2008; Williams et al., 2012). Yet, the association between culturally relevant resilience factors and brain health remains poorly understood. To our knowledge, no previous neuroimaging studies have

examined the impact of racial-ethnic identity on the brain. To address this gap, we examined the moderating role of racial-ethnic identity on the association between neighborhood disadvantage and white matter tract integrity.

White Matter Tract Integrity as an Index of Brain Health

White matter tracts are bundles of long-distance neuron axons. Broadly, tract integrity, indexed frequently by fractional anisotropy (FA), is considered a measure of how efficiently the brain processes information (Paus, 2010). FA quantifies the directional asymmetry of water diffusion at each voxel in the brain and is influenced by the extent of myelination, axonal integrity, and brain tissue fiber arrangements (De Erausquin & Alba-Ferrara, 2013). Importantly, these microstructural differences have been linked to behavioral differences. For example, greater global integrity is a predictor of better executive functioning performance (Kerchner et al., 2012; Penke et al., 2012; Xing et al., 2021). There are common white matter tracts that serve as critical "information highways" to facilitate communication between different parts of the brain contributing to cognitive, affective, and motor functions (Paus, 2010). Herein, we focused on 10 white matter tracts that have received considerable attention (eight tracts with left and right hemisphere and two tracts without hemisphere differentiation): corticospinal tract, inferior longitudinal fasciculus, superior longitudinal fasciculus-parietal bundle, superior longitudinal fasciculus-temporal bundle, uncinate fasciculus, anterior thalamic radiation, cingulum-cingulate gyrus bundle, cingulum-angular bundle, corpus callosum's forceps major, and corpus callosum's forceps minor (Yendiki et al., 2011).

In adults, the chronic stress related to lower individual socioeconomic position (SEP; i.e., education or income) is significantly related to alterations in white matter tract integrity, including in the cingulum-cingulate gyrus (CCG) and the cingulum angular bundles (CAB; Gianaros et al., 2013; Johnson et al., 2013; Noble et al., 2013; Shaked et al., 2019). The CCG runs alongside the anterior cingulate cortex, which underlies various emotion regulatory processes, including detecting and processing reward, safety, and fear cues (Rolls et al., 2019). The CCG projects cue-related information to the retrosplenial cortex, where it is integrated with information from the broader environmental context (Trask et al., 2021). Thus, this pathway provides bidirectional communication throughout part of the brain's learning and memory system (Bubb et al., 2018; Maddock, 1999; Robinson et al., 2018). The CAB connects the cingulate gyrus to the hippocampus, a critical region for contextual memory formation, offering yet another mechanism by which emotional information is transmitted across long distances (Averill et al., 2018; Ezzati et al., 2016; Nezamzadeh et al., 2010; Wu et al., 2016).

The handful of studies on neighborhood disadvantage and white matter tracts document effects similar to those of individual SEP. In adolescents, greater neighborhood disadvantage, but not community violence, is associated with reduced integrity in the CAB, uncinate fasciculus, and fornix (Bell et al., 2021). In adults, even after adjusting for income, education, gender, and age, neighborhood disadvantage was inversely associated with whole-brain FA (Gianaros et al., 2013). White matter tract abnormalities may represent vulnerabilities to certain psychopathologies or be further exacerbated by psychiatric conditions (e.g.,Bracht et al., 2015; Chen et al., 2016; Daniels et al., 2013). For example, studies have documented the extensive impact of

trauma exposure on white matter tracts (Akiki et al., 2017; Averill et al., 2018, 2018; Harnett et al., 2020, 2021; Kim et al., 2005). Although microstructural differences appear to vary by trauma type and timepoint, decreased integrity of both the CAB and CCG are associated with non-remitting PTSD. (Averill et al., 2018; Kim et al., 2005; c.f., Dennis et al., 2019). Notably, more extensive and severe trauma histories are associated with lower socioeconomic position (Breslau et al., 1998; Read et al., 2011). Given this overlap, previous individual SEP-white matter tract integrity findings may have inadvertently captured the effects of a trauma exposure, rather than isolating the unique contributions of individual SEP.

Current Study

Using a recently traumatically injured sample, the current project explored whether neighborhood disadvantage was associated with white matter tract integrity. We used a multivariate multiple regression model to simultaneously predict FA values in the 10 white matter tracts using neighborhood disadvantage, income, ethnoracial group, gender, age, and PTSD symptoms as predictors. We explored whether white matter abnormalities explained the relationship between neighborhood disadvantage and non-remitting PTSD symptoms (measured six-months post-injury).

In an exploratory aim, we took a risk-resilience approach, testing whether racial-ethnic identity (i.e., MEIM scores) moderated the relationship between neighborhood disadvantage and FA values. By assessing this Neighborhood Disadvantage x Racial-ethnic Identity interaction, we expected to expand existing work by demonstrating this resilience factor works at a neurobiological level.

Method and Materials

Participants

Between 2016-2019, 215 traumatically-injured adults were recruited from an Emergency Department in the United States Midwest and enrolled in the Imaging Study of Trauma and Resilience (iSTAR; Bird et al., 2021; Webb et al., 2021; Weis, Webb, Huggins, et al., 2021; Weis, Webb, Stevens, et al., 2021). Individuals were considered eligible if they had experienced a traumatic injury, were between the ages of 18 and 65 years old and could speak and understand English. Full inclusion and exclusion criteria are provided in Table 1.1. All participants provided written consent and were financially compensated for their time. The study protocol was approved by the Institutional Review Board at the Medical College of Wisconsin.

Table 1.1. Study Enrollment Criteria

Inclusion	Exclusion
Experienced a traumatic injury that led to ED visit	Moderate to severe traumatic brain injury (Glasgow Coma Scale[b] Score of < 13)
18-65 years of age	Suffered a spinal cord injury with neurological deficits
English-speaking	Substance use disorder
Ability to schedule a study appointment within two-weeks of trauma	Visit to ED was a result of suicide attempt or self-harm
A minimum score of 3 on the Predicting PTSD Questionnaire[a] (indicative of elevated risk of future PTSD)	Active psychosis, or history of psychotic or manic symptoms, or current prescription of antipsychotic medication
	On police hold following traumatic injury
	MRI incompatible (e.g., presence of ferromagnetic material in body, claustrophobic, pregnant, etc.)

Note: **ED**; emergency department; [a.] Rothbaum et al., 2014; [b.] Sternbach et al., 2000.

Procedure

Approximately two-weeks after experiencing a traumatic injury (timepoint one; T1), participants underwent two consecutive days of neuroimaging, including a diffusion-weighted imaging scan (acquired on day two). In addition, participants completed a battery of self-report measures and various behavioral tasks. Participants returned six-months post-injury (timepoint two; T2) and repeated select self-report measures, neuroimaging, and behavioral batteries. In addition, trained staff members delivered the Clinician-Administered PTSD Scale for DSM-5 (Weathers et al., 2018), to evaluate PTSD symptom severity and determine PTSD diagnostic status.

Measures

Neighborhood Socioeconomic Disadvantage

An Area Deprivation Index (ADI) ranking was derived from the home address participants provided at the two-week visit. ADI is a relative measure, meaning it captures how a neighborhood's socioeconomic position compares to all other neighborhoods in the United States during the specified period (Kind & Buckingham, 2018; Singh, 2003). A neighborhood is defined by the census geographical unit *block-group*. On average, between 600 to 3,000 individuals live in a block-group, defined primarily by visible natural and built boundaries (Census Bureau, 2011).

The index considers block-group estimates of 17 sociopolitical variables assessed in the 2011-2015 American Community Survey (administered by the Census Bureau). Using an established factor-weighting procedure, these variables were incorporated into the composite ranking (Hu et al., 2018; Hunt et al., 2020; Kind & Buckingham, 2018; Knighton et al., 2016; Singh, 2003). ADI ranges from 1 to 100,

where “1” indicates the most advantaged neighborhood and “100” represents the most disadvantaged neighborhoods.

After data collection, the ADI data was downloaded (February 2020; from https://www.neighborhoodatlas.medicine.wisc.edu/) and participants were geo-coded (i.e., their home address was matched to the correct census area designation code). Participants were excluded from this geocoding process if: they provided a post-office box as their primary residence, lived out of the state of Wisconsin, or the address was not associated with a block-group ID.

Individual Demographics

At T1, participants self-reported their race, ethnicity, gender, annual household income, and age. Possible responses for gender included: “male”, “female”, or “other”. A selection of “other” prompted an unrestricted text box where participants could type a response. Racial and ethnic categories aligned with those required by the National Institutes of Health (“American Indian/Alaska Native”, “Asian”, “Native Hawaiian or Other Pacific Islander”, “Black or African American”, “White”, “more than one race” or “unknown or not reported”). Ethnicity categories included: “Not Hispanic or Latino”, “Hispanic or Latino”, or “unknown/not reported”. Due to small sample sizes amongst various racial and ethnic groups, a binary grouping variable was created; “0” indicated the participant identified as a member of a racially and/or ethnically marginalized group and “1” designated that the participant identified as White. Moreover, scores on the multigroup ethnic identity measure were consistently higher in individuals from minoritized groups compared to White participants.

Annual household income was reported on a semi-continuous scale; “1” indicated the household earned between $0-10,000 annually. Every additional one-unit increment corresponded to an additional $10,000 in income. An “11” reflected an annual household income of over $100,000.

Multigroup Ethnic Identity Measure

The multi-ethnic identity measure (MEIM; Phinney, 1992) was administered at T1 to evaluate racial-ethnic identity. This 6-item questionnaire has two subscales examining the participant’s exploration (“I have spent time trying to find out more about my own ethnic group, such as its history, traditions, and customs”) and commitment (“I feel a strong attachment towards my own ethnic group”) to their ethnoracial group. Participants rated how much they agreed with each statement on a scale ranging from 1 (*strongly disagree)* to 5 (*strongly agree)*. Responses to all items were averaged to create a total score.

T1 Post-Traumatic Stress Symptoms

The PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015) was completed during T1. The PCL-5 is a 20-item questionnaire that evaluates the PTSD symptoms listed in the DSM-5 using a five-point Likert scale. Participants rated how much each of the symptoms had bothered them on a scale from 1 (*not at all*) to 5 (*extremely*) since the index trauma occurred. A total symptom severity score was created by summing the scores for each of the items (Blevins et al., 2015).

T2 Post-Traumatic Stress Disorder Symptoms

At T2, trained research staff members conducted the Clinician-Administered PTSD Scale for DSM-5 (CAPS; Weathers et al., 2018). The CAPS-5 is a 30-item semi-

structured interview during which the interviewer evaluates severity and frequency of PTSD symptoms over the past month. A total symptom severity score was calculated by summing the scores of 20 items which directly correspond to DSM-5 symptoms. CAPS-5 interviews were audio-recorded and 20% of all recordings were subjected to reliability checks by another staff member. There was excellent reliability across CAPS-5 administration within the study (interclass correlation coefficient = 0.96, with 95% confidence interval [0.93, 0.98]).

Diffusion Tensor Imaging Acquisition

All MRI images were collected using a 3.0 Tesla short-bore General Electric Signa Excite system (Waukesha, WI). First, high resolution T1-weighted structural images were acquired in a sagittal orientation (TR = 8.2ms; TE = 3.2ms; Field of View (FOV) = 24 cm; flip angle = 12°; voxel size = 1 x 0.9375 x 0.9375mm). Diffusion weighted images (DWI) were collected using an echoplanar pulse sequence and the following sequence: TR = 10s; TE = 77.99ms; b-value = 800s/mm^2; FOV = 25.6cm; flip angle = 12°; voxel size = 2 x 2 x 2mm.

Diffusion Weighted Image Processing

Structural T1 scans were processed in FreeSurfer (version 5.3; Fischl, 2004) and DWI were processed using TRACULA (Yendiki et al., 2011). Preprocessing of DWIs included image correction, registration to anatomical scans and tensor fitting. Head motion parameters were calculated during the preprocessing steps and carried as a nuisance covariate. Information on anatomical structures/boundaries (i.e., anatomical priors) was introduced during the default preprocessing steps and derived from an existing training set (Yendiki et al., 2011). TRACULA reconstructed white matter tracts

by generating probability distributions at every voxel using both the anatomical priors and a ball-and-stick diffusion tensor model. FA measures were then extracted from TRACULA. Path reconstruction was poor for a number of participants. For bilateral tracts, if one hemisphere was successfully reconstructed, then that hemisphere's FA value was used in place of the average. If neither hemisphere was reconstructed, then data was mean imputed: UNC (*n* = 6), CAB (*n* = 16), FMINOR (*n* = 11), FMAJOR (*n* = 30).

The degree of correlation between left and right FA values for each tract was moderate to high (correlation coefficients from 0.35 (CAB tracts) to 0.76 (SLFT tracts), Figure 1.2). For tracts with separate left and right hemisphere values, the values were averaged across hemispheres. The final FA values were then carried to the multivariate multiple regression analysis.

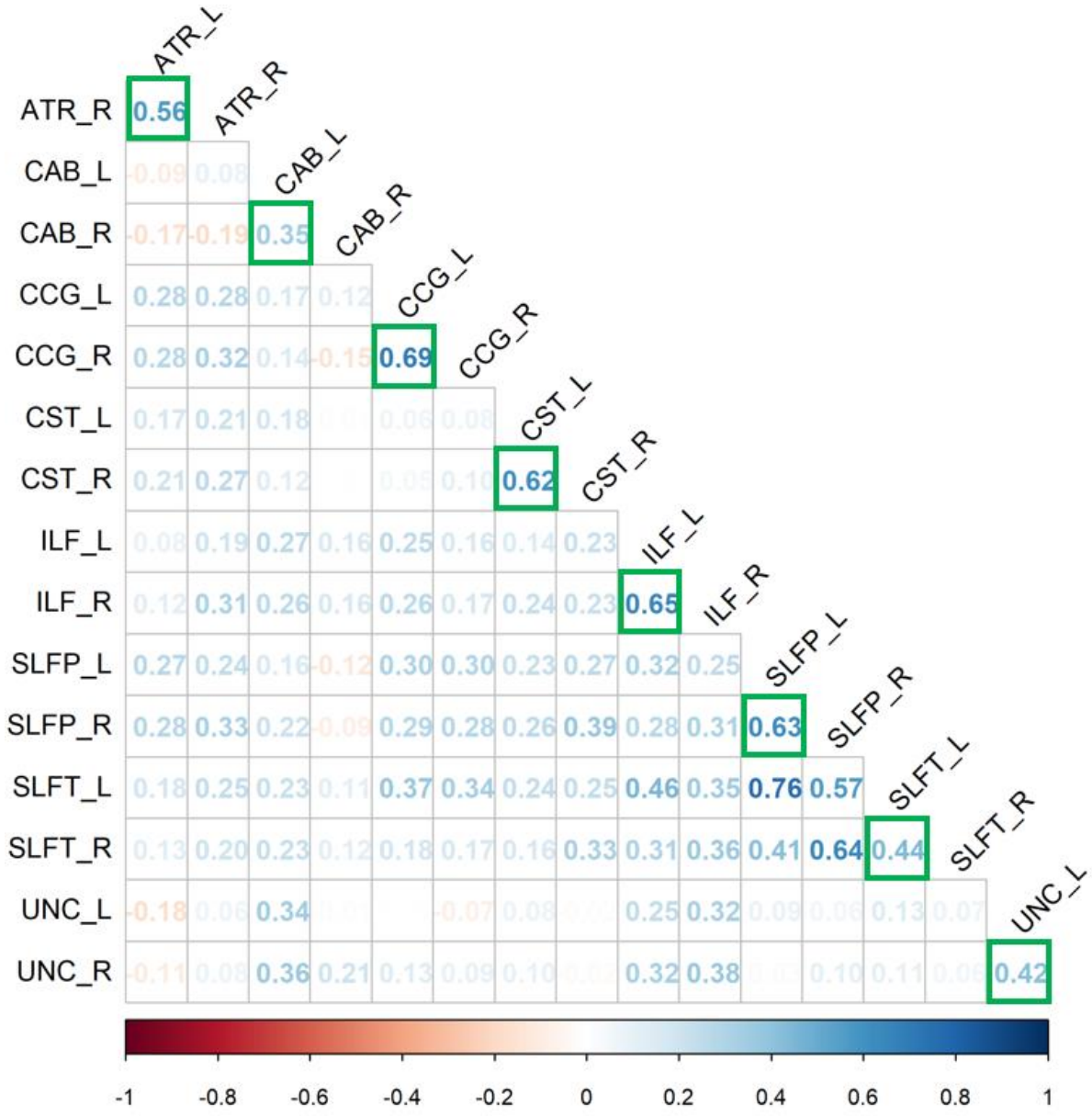

Figure 1.2. **Correlations between left and right white matter tract fractional anisotropy values.** Green squares highlight left and right correlations of the same tracts. *Abbreviations:* **ATR:** anterior thalamic radiations; **CAB:** cingulate-angular bundles; **CCG:** Cingulum-cingulate gyrus; **CST**: corticospinal tract; **SLFP:** superior longitudinal fasciculus parietal bundle; **SLFT**: superior

longitudinal fasciculus temporal bundle; **UNC**: uncinate fasciculus; **L:** left; **R:** right. *Note:* forceps major and minor are bilateral tracts and are not included.

Analysis Strategy

Of the 215 enrolled participants, 167 participants had usable scans (three participants had poor quality scans), nine could not be geocoded and ten were missing scores on the multigroup ethnic identity measure. Sample characteristics for participants (*N* = 148) can be found in Table 1.2.

All analyses were conducted in R (version 4.1.1; R Core Team, 2020; R markdown file with scripts is provided in Appendix 1). Continuous variables (e.g., FA values, age, income, ADI, PCL-5 scores, CAPS-5 scores) were grand-mean centered. Bivariate associations between continuous measures were assessed with Pearson's correlations. A multivariate multiple regression (MMR) analysis determined whether ADI was associated with white matter tract integrity over and above individual variables (Figure 1.3). In this model, ADI, PCL-5 scores, gender, age, ethnoracial group, and annual household income were all considered independent variables whereas FA values for each of the 10 white matter tracts served as simultaneous dependent variables. Ethnoracial group was included in the model to test whether societal racialization *beyond* the racialization of socioeconomic position, such as experiences of discrimination, may explain heterogeneity in white matter tract integrity. In this way, ethnoracial group was used as a reference proxy for sociopolitical position of the individual.

In the MMR, we first calculated the standardized regression weights for each of the independent variables for each dependent variable (these results are identical to

univariate multiple regression models). Then, we tested the MMR model to determine which independent variables significantly predicted the multiple dependent variables. To determine which values were significant, we examined the Pillai test statistics from a multivariate analysis of variance (MANOVA) of the MMR model.

To examine whether racial-ethnic identity moderated the relationship between neighborhood disadvantage and white matter tract integrity, we conducted 10 multiple regression models. In each model, the FA value of a specific tract was the dependent variable and covariates included, income, age, gender, PCL-5 scores, ADI, MEIM, and an ADI x MEIM interaction term. As the models evaluating ADI x MEIM interactions were exploratory, there was no correction for multiple comparisons. Significant interactions were probed by performing simple slopes analyses at the mean and one standard deviation below and above the mean MEIM scores.

Finally, we tested whether ADI explained relationships between FA values and CAPS-5 symptom severity. First, we consulted the Pearson's correlations between FA and CAPS-5. Any significant tracts were then taken to a mediation analysis to evaluate if ADI significantly mediated the relationship between FA and CAPS-5. In all analyses, $p < .05$ was considered significant.

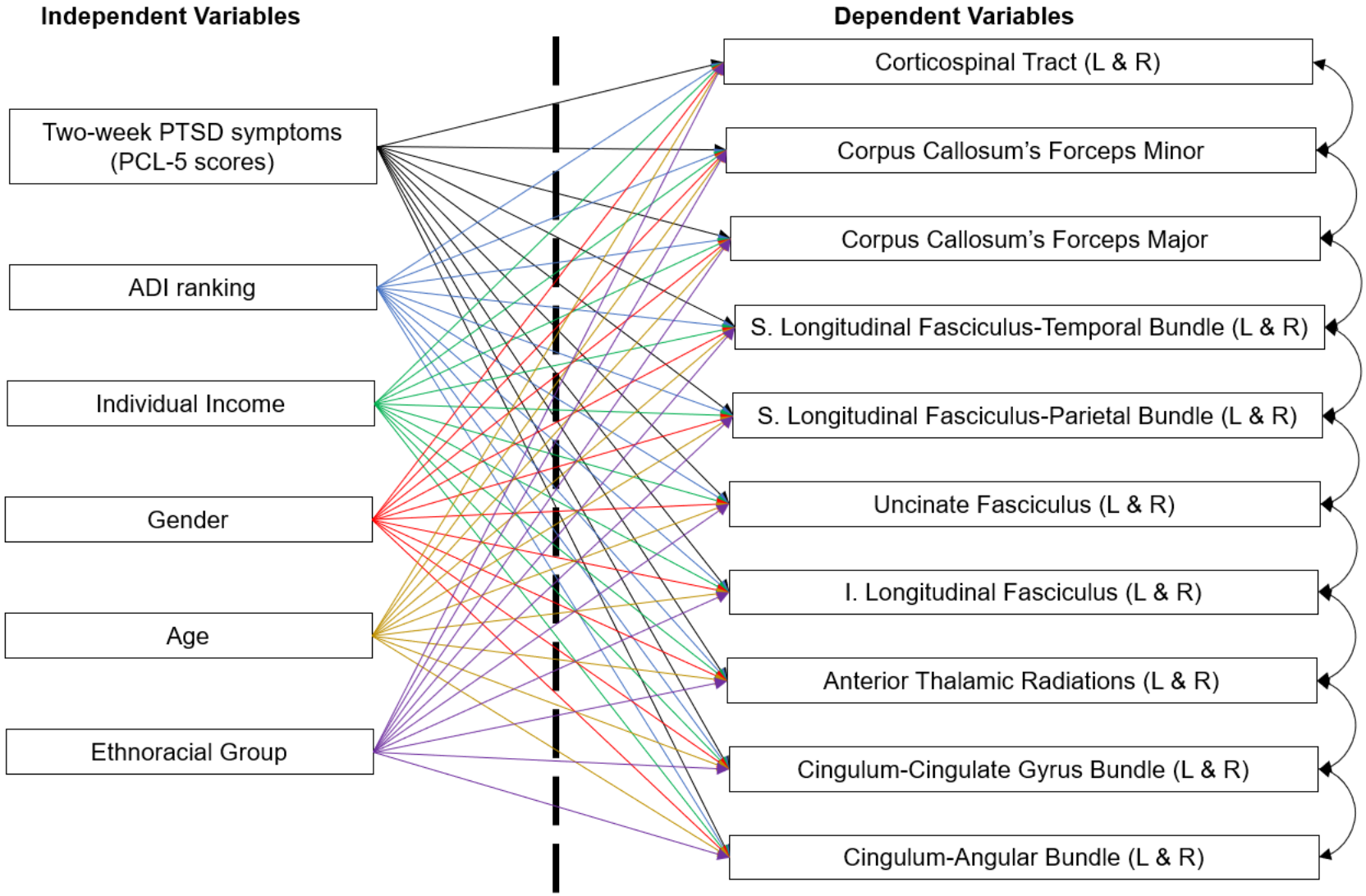

Figure 1.3. **Overview of the multivariate multiple regression model.** *Abbreviations:* **I**: inferior; **S:** superior; **L:** left; **R:** right. *Note:* hemispheres were averaged for tracts with left and right differentiation.

Table 1.2. Sample Characteristics (*N* = 148)

Variable	***Mean (SD) or %***
Age (years)	32.23 years (10.75)
Gender	
Female	55% (*n* = 81)
Individual Education	
Did not complete high school	8%
High school/GED	33%
Some post-secondary education/college	28%
Associate or bachelor's degree	26%
Master's degree, JD, MD, PhD	5%
Individual Income	
$0-10,000	20%
$10,000-20,000	16%
$20,000-30,000	15%
$30,000-40,000	8%
$40,000-50,000	8%
$50,000-60,000	6%
$60,000-70,000	6%
$70,000-80,000	7%
$80,000-90,000	<5%
$90-100,000	<5%
$100,000 and above	7%
Race	
African American and/or Black	60%
White	27%
Other racial identity*	13%
Ethnicity	
Hispanic or Latino	8%
Mechanism of Injury	
Motor vehicle crash	68%
Physical assault	16%
Other	14%
T1 PTSD Symptoms (PCL-5)	25.43 (16.64)
T2 PTSD symptoms (CAPS-5) **	12.00 (10.28)
Multigroup Ethnic Identity Measure (MEIM)	2.77 (0.85)
Area Deprivation Index	68.89 (21.83)

Abbreviations: **CAPS-5**: Clinician-Administered PTSD Scale for DSM-5; **PCL-5:** PTSD Checklist for DSM-5, **T1:** timepoint 1 (two-weeks post-injury; **T2:** timepoint 2 (six-months post-injury). *Note:* ***** due to small sample sizes, additional self-reported racial identities have been combined. ** due to loss-to-follow-up, only 132 participants had six-month PTSD symptoms.

Results

Correlations

All bivariate associations between study measures are presented in Figure 1.4. Age was significantly related to PCL-5 scores ($r(146) = -0.23$, $p = .006$), forceps minor FA (FMINOR; $r(146) = -0.17$, $p = .040$), superior longitudinal fasciculus-parietal bundle FA (SLFP; $r(146) = -0.17$, $p = .038$), and inferior longitudinal fasciculus FA (ILF; $r(146) = -0.18$, $p = .034$). Six-month PTSD symptoms (CAPS-5 scores; $n = 132$) were associated with PCL-5 scores ($r(130) = 0.29$, $p < .001$), income ($r(130) = -0.21$, $p = .015$), and cingulum-cingulate gyrus FA (CCG; $r(130) = -0.17$, $p = .046$). PCL-5 scores were positively correlated with FA of the anterior thalamic radiations (ATR; $r(146) = 0.17$, $p = .044$), SLFP ($r(146) = 0.23$, $p = .005$), CCG ($r(146) = 0.17$, $p = .038$), and superior longitudinal fasciculus-temporal bundle (SLFT; ($r(146) = 0.21$, $p = .012$). Greater income was associated with greater neighborhood disadvantage ($r(146) = -0.43$, $p < .001$) and higher FA in the CST ($r(146) = 0.20$, $p = .013$) and forceps major (FMAJOR; $r(146) = 0.18$, $p = .028$). Higher ADI rankings were significantly related to lower FA values in the CST ($r(146) = -0.17$, $p = .043$) and FMAJOR, $r(146) = -0.23$, $p < .001$.

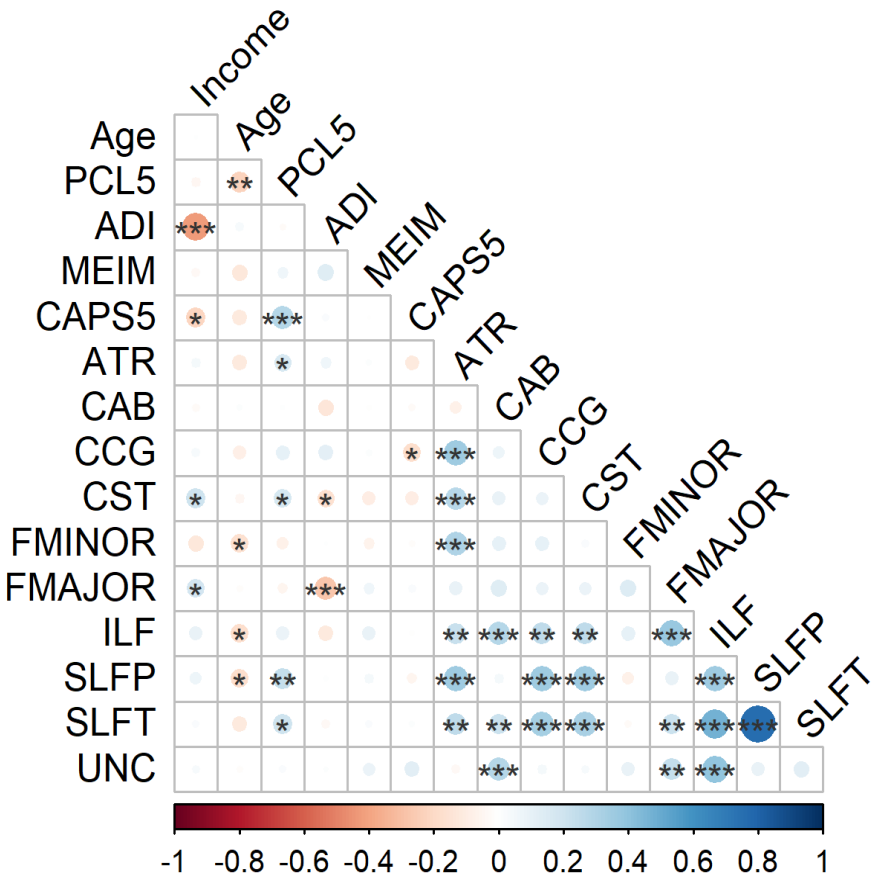

Figure 1.4. **Correlations between study measures and white matter tract fractional anisotropy values.** *Abbreviations:* **ADI:** Area Deprivation Index (National Ranking); **ATR:** anterior thalamic radiations; **CAB:** cingulate-angular bundles; **CAPS-5:** Clinician Administered PTSD Scale for DSM-5 symptom severity; **CCG:** Cingulum-cingulate gyrus; **CST**: corticospinal tract; **FMAJOR:** forceps major; **FMINOR:** forceps minor; **MEIM:** Multigroup Ethnic Identity Measure; **PCL-5:** PTSD Checklist for DSM-5; **SLFP:** superior longitudinal fasciculus parietal bundle; **SLFT**: superior longitudinal fasciculus temporal bundle; **UNC**: uncinate fasciculus. *Note:* ******* $p < .001$, ****** $p < .01$, ***** $p < .05$.

Differences in Study Measures by Ethnoracial Group

White participants reported significantly higher annual income and lived in more advantaged neighborhoods (*n* = 40; mean (*M*) ADI = 51.6, standard deviation (*SD*) = 22.68; *M* income = 6.63, *SD* = 3.54; Figure 1.5A) compared to participants from ethnoracially marginalized groups (*n* = 108; *M* ADI = 75.28, *SD* = 17.73; $t(146) = 6.67$, $p < .001$; *M* income = 3.62, *SD* = 2.59; $t(146) = -5.66$, $p < .001$). Ethnoracially minoritized participants scored significantly higher on the MEIM (*n* = 108; *M* = 2.90, *SD* = 0.83) than White participants (*n* = 40; *M* = 2.41, *SD* = 0.80; $t(146) = 3.22$, $p = .002$; Figure 1.5B).

Finally, there was no significant difference between groups in baseline PTSD (White participants (*n* = 40) *M* PCL-5 = 25.40, *SD* = 18.80; ethnoracially minoritized participants (*n* = 108) *M* PCL-5 = 25.44, *SD* = 18.85; $t(146) = 0.01$, $p = .989$) or six-month PTSD symptoms (White participants (*n* = 36) *M* CAPS-5 = 11.42, *SD* = 11.42; ethnoracially minoritized participants (*n* = 96) *M* CAPS-5 = 12.01, *SD* = 9.88; $t(130) < 0.01$, $p = .996$).

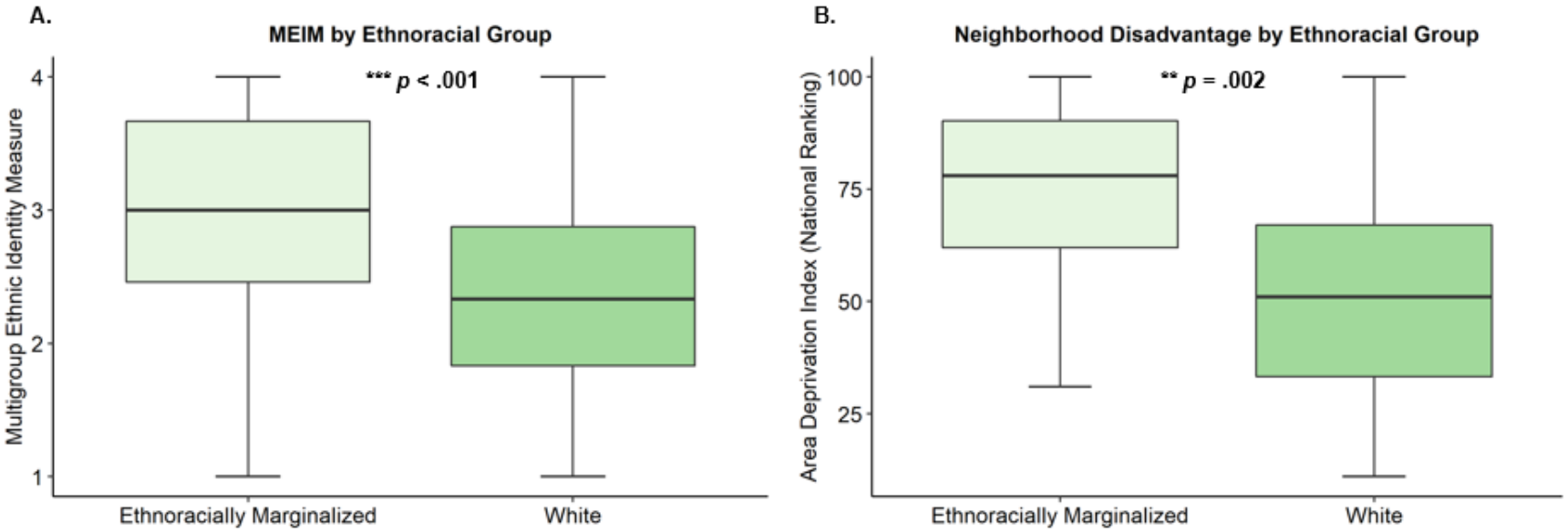

Figure 1.5. **MEIM and neighborhood disadvantage significantly differed by ethnoracial group.** White participants **(A)** lived in more advantaged neighborhoods (ADI mean (*M*) = 51.6, standard deviation (*SD*) = 22.68; *n* = 40) compared to participants from ethnoracially marginalized groups (*n* = 108; ADI *M* = 75.28, *SD* = 17.73). **(B)** Participants from minoritized groups had significantly higher scores on the MEIM (*M* = 2.90, *SD* = 0.83) compared to White participants (*M* = 2.41, *SD* = 0.80).

Neighborhood Disadvantage is Associated with White Matter Tract Integrity

As part of the MMR model, univariate regression models were fitted (Table 1.3). Older age was associated with less integrity in FMINOR (B = -0.0007, $t(147) = -2.06$, $p = .042$; full model: $F(6,141) = 2.20$, $p = .046$, $R^2_{adj} = 0.05$). Neighborhood disadvantage was significantly associated with lower FA in FMAJOR (B = -0.0007, $t(147) = -2.85$, $p = .005$; full model: $F(6,141) = 2.42$, $p = .030$, $R^2_{adj} = 0.05$) and CAB, B = -0.0007, $t(147) = -2.85$, $p = .005$; full model: $F(6,141) = 0.90$, $p = .497$, $R^2_{adj} < 0.01$). Greater baseline PTSD symptoms was significantly associated with higher FA in SLFP (B = 0.0002, $t(147) = 2.19$, $p = .030$; full model: $F(6,141) = 3.41$, $p = .003$, $R^2_{adj} = 0.09$) and CST (B = 0.0002, $t(147) = 2.16$, $p = .032$; full model: $F(6,141) = 2.41$, $p = .030$, $R^2_{adj} = 0.05$). White participants had significantly higher FA values in SLFT (B = 0.0129, $t(147) = 2.77$, $p = .006$; ; full model: $F(6,141) = 2.55$, $p = .022$, $R^2_{adj} = 0.06$) and SLFP, B = 0.0137, $t(147) = 2.61$, $p = .010$) .

The results of the MMR analysis indicated only neighborhood disadvantage was significantly associated with the FA values *(*Pillai's trace = 0.13*,* approximate $F(10,132) = 1.91$, $p = .049$). Ethnoracial group *(*Pillai's trace = 0.11*,* approximate $F(10,132) = 1.92$, $p = .108$), income *(*Pillai's trace = 0.09*,* approximate $F(10,132) = 1.31$, $p = .231$), PCL-5 *(*Pillai's trace = 0.09*,* approximate $F(10,132) = 1.35$, $p = .213$), gender *(*Pillai's trace = 0.10*,* approximate $F(10,132) = 1.46$, $p = .160$), and age *(*Pillai's trace = 0.09*,* approximate $F(10,132) = 1.26$, $p = .258$) were not significantly associated with FA.

Table 1.3: Univariate Results from MMR Analysis

Tract	*Variable*	*B*	*t-statistic*	*p*
Forceps minor; $F(6,141) = 2.20$, ***p* = .046***, $R^2_{adj} = 0.05$				
	Intercept	0.4631	80.80	< .001 ***
	Ethnoracial group	-0.0045	-0.49	.623
	Income	-0.0022	-1.76	.081
	ADI	-0.0002	-0.96	.340
	Gender	-0.0133	-1.89	.062
	Age	**-0.0007**	**-2.06**	**.042***
	PCL-5	-0.0002	-1.10	.274
Forceps major; $F(6,141) = 2.42$, ***p* = .030***, $R^2_{adj} = 0.05$				
	Intercept	0.5676	95.84	< .001 ***
	Ethnoracial group	-0.0044	-0.47	.637
	Income	0.0010	0.79	.429
	ADI	**-0.0005**	**-2.85**	**.005**
	Gender	-0.0073	-1.00	.317
	Age	<-0.0001	-0.10	.925
	PCL-5	-0.001	-0.50	.615
Uncinate fasciculus tract; $F(6,141) = 0.26$, $p = .954$, $R^2_{adj} = -0.03$				
	Intercept	0.3795	74.12	< .001 ***
	Ethnoracial group	0.0040	-0.48	.629
	Income	<-0.0001	0.50	.618
	ADI	<0.0001	0.05	.964
	Gender	0.0062	-0.99	.323
	Age	<-0.0001	-0.03	.974
	PCL-5	<0.0001	0.45	.651
Anterior thalamic radiations; $F(6,141) = 2.02$, $p = .067$, $R^2_{adj} = 0.04$				
	Intercept	0.4253	120.83	< .001 ***
	Ethnoracial group	0.0041	0.74	.459
	Income	0.0005	0.68	.496
	ADI	0.0001	1.28	.203
	Gender	**-0.0088**	**-2.05**	**.043***
	Age	< 0.0002	-0.76	.45
	PCL-5	**0.0003**	**2.18**	**.031***
Superior longitudinal fasciculus-temporal bundle; $F(6,141) = 2.55$, ***p* = .022***, $R^2_{adj} = 0.06$				
	Intercept	0.4015	136.72	<.001*
	Ethnoracial group	**0.0129**	**2.77**	**.006**
	Income	-0.0003	-0.46	.643
	ADI	<0.0001	0.80	.424
	Gender	0.0010	0.27	.790
	Age	-0.0002	-0.97	.332
	PCL-5	**0.0002**	**2.19**	**.030***
Superior longitudinal fasciculus-parietal bundle; $F(6,141) = 3.41$, ***p* = .003***, $R^2_{adj} = 0.09$				
	Intercept	0.3993	121.01	<.001*

Ethnoracial group	**0.0137**	**2.61**	**.010***
Income	0.0004	0.61	.544
ADI	0.0002	1.75	.083
Gender	0.0045	1.10	.272
Age	-0.0003	-1.78	.077
PCL-5	**0.0003**	**2.30**	**.023***
Inferior longitudinal fasciculus; $F(6,141) = 1.60$, $p = .150$, $R^2_{adj} = 0.02$			
Intercept	0.4582	100.53	<.001**
Ethnoracial group	0.0072	1.00	.318
Income	0.0003	0.28	.778
ADI	0.0001	-0.53	.597
Gender	-0.0065	-1.12	.245
Age	0.0004	-1.76	.080
PCL-5	0.0001	0.85	.397
Corticospinal tract; $F(6,141) = 2.41$, $\mathbf{p = .030^*}$, $R^2_{adj} = 0.05$			
Intercept	00.4281	165.72	<.001*
Ethnoracial group	0.0051	1.34	.182
Income	0.0008	1.44	.152
ADI	<-0.0001	-0.47	.639
Gender	-0.0014	-0.43	.670
Age	<-0.0001	-0.01	.992
PCL-5	**0.0002**	**2.16**	**.032***
Cingulum-angular bundles; $F(6,141) = 0.90$, $p = .497$, $R^2_{adj} < 0.01$			
Intercept	0.3352	65.14	<.001*
Ethnoracial group	-0.0016	-0.20	.842
Income	-0.0013	-1.19	.236
ADI	**-0.0003**	**-2.00**	**.047***
Gender	-0.0072	-1.14	.258
Age	0.0001	0.40	.688
PCL-5	<0.0001	0.25	.802
Cingulum-cingulate gyrus; $F(6,141) = 1.88$, $p = .088$, $R^2_{adj} = 0.03$			
Intercept	0.5212	97.24	<.001*
Ethnoracial group	-0.0103	-1.21	.230
Income	0.0016	1.34	.183
ADI	0.0002	1.11	.271
Gender	-0.0128	-1.95	.053
Age	-0.0002	-0.57	.572
PCL-5	0.0003	1.54	.125

Abbreviations: **ADI:** Area Deprivation Index (National Ranking); **PCL-5:** PTSD Checklist for DSM-5. *Note:* * $p < .05$.

Racial-Ethnic Identity Moderates the Relationship Between Neighborhood Disadvantage and Tract Integrity

Results of the 10 separate multiple regressions models which examined whether MEIM scores moderated the relationship between neighborhood disadvantage and white matter tract integrity can be found in Table 1.4. There was a significant interaction between ADI and MEIM on FA in the SLFP tract (Figure 1.6; B = 0.0003, $t(147) = 2.30$, $p = .023$; full model: $F(7,140) = 2.67$, $p = .013$, $R^2_{adj} = 0.07$). Simple slope analyses revealed that individuals with stronger racial-ethnic identity (+1 SD MEIM) had a positive relationship between ADI and SLFP tract integrity (B = 0.0003, $t(147) = 2.15$, $p = .033$).

The ADI x MEIM interaction term approached significance in the SLFT (B = 0.0002, $t(147) = 1.91$, $p = .058$; full model: $F(7,140) = 1.57$, $p = .148$, $R^2_{adj} = 0.03$) and CCG tracts (B = 0.0003, $t(147) = 1.80$, $p = .074$; full model: $F(7,140) = 1.88$, $p = .078$, $R^2_{adj} = 0.04$). As in the MMR univariate analysis, neighborhood disadvantage was associated with less integrity in the FMAJOR (B = -0.0010, $t(147) = -3.07$, $p = .003$, full model: $F(7,140) = 2.37$, $p = .026$, $R^2_{adj} = 0.06$). Older age was significantly related to less integrity in FMINOR (B = -0.0007, $t(147) = -2.09$, $p = .038$; full model: $F(7,140) = 2.35$, $p = .027$, $R^2_{adj} = 0.06$). Higher PCL-5 scores were significantly associated with greater integrity in the CST (B = 0.0002, $t(147) = 2.02$, $p = .045$; full model: $F(7,140) = 2.10$, $p = .048$, $R^2_{adj} = 0.05$).

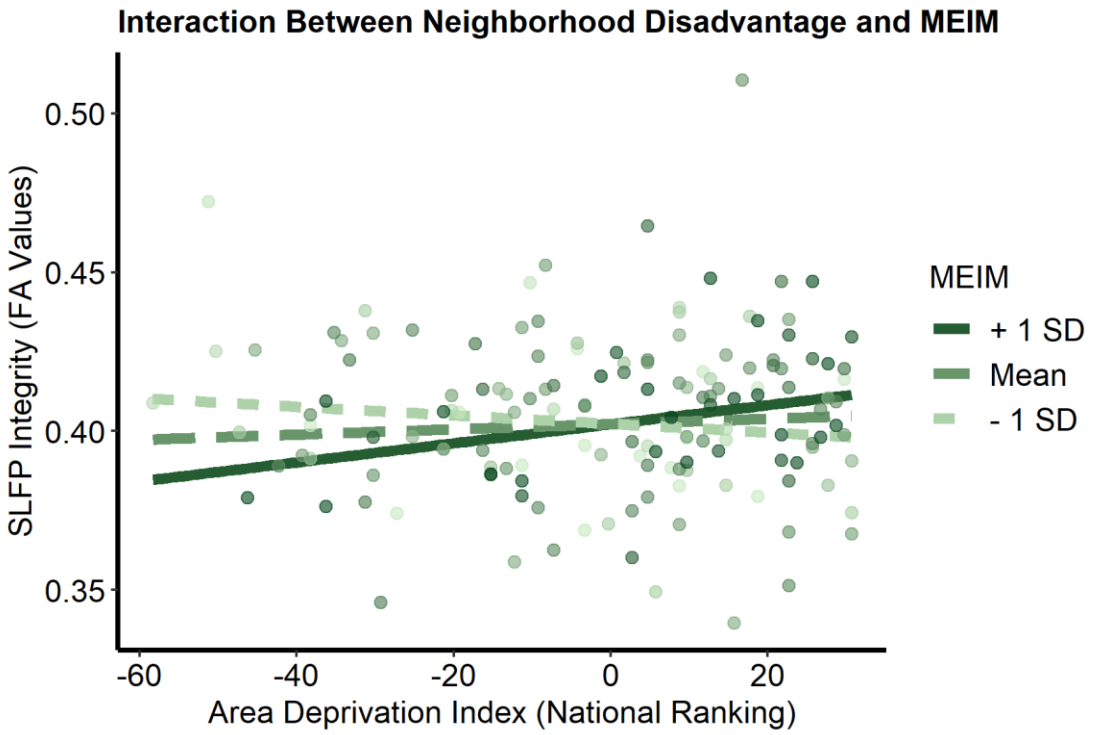

Figure 1.6. **Racial-ethnic identity moderates the relationship between neighborhood disadvantage and SLFP tract integrity.** Participants with higher MEIM scores residing in more disadvantaged neighborhoods, had significantly higher FA in the SLFP tract. *Abbreviations*: **MEIM:** Multigroup Ethnic Identity Measure; **SLFP:** superior longitudinal fasciculus parietal bundle. *Note:* ADI rankings are grand mean centered. MEIM is a continuous variable grouped by +1 standard deviation (SD), mean, and -1 SD for visualization purposes.

Table 1.4: Multiple Regressions with ADI x MEIM Interaction

Tract	*Variable*	*B*	*t-statistic*	*p*
Forceps minor; $F(7,140) = 2.35$, ***p* = .027***, $R^2_{adj} = 0.06$				
	Intercept	0.4718	61.62	<.001***
	MEIM	-0.0031	-0.77	.446
	ADI	-0.0001	-0.70	.487
	Gender	-0.0130	-1.85	.066
	Income	-0.0021	-1.73	.087
	Age	**-0.0007**	**-2.09**	**.038***
	PCL-5	-0.0002	-0.83	.410
	ADI x MEIM	-0.0003	-1.61	.109
Forceps major; $F(7,140) = 2.37$, ***p* = .026***, $R^2_{adj} = 0.06$				
	Intercept	0.5633	70.93	<.001***
	MEIM	0.0061	1.44	.153
	ADI	**-0.0010**	**-3.07**	**.003****
	Gender	-0.0081	-1.12	.265
	Income	-0.0009	0.68	.498
	Age	<0.0001	0.11	.916
	PCL-5	-0.0001	-0.47	.637
	ADI x MEIM	-0.0001	-0.41	.683
Uncinate fasciculus tract; $F(7,140) = 0.39$, $p = .907$, $R^2_{adj} = -0.03$				
	Intercept	0.3770	54.75	<.001***
	MEIM	0.0038	1.02	.309
	ADI	<0.0001	0.09	.929
	Gender	-0.0068	-1.08	.284
	Income	0.0005	0.42	.676
	Age	<0.0001	0.12	.903
	PCL-5	0.0001	0.52	.604
	ADI x MEIM	-0.0001	-0.64	.523
Anterior thalamic radiations; $F(7,140) = 1.69$, $p = .117$, $R^2_{adj} = 0.03$				
	Intercept	0.4236	89.02	<.001***
	MEIM	-0.0002	-0.09	.930
	ADI	0.0001	1.07	.285
	Gender	**-0.0089**	**-2.03**	**.044***
	Income	0.0006	0.84	.404
	Age	-0.0002	-0.77	.444
	PCL-5	**0.0003**	**2.07**	**.040***
	ADI x MEIM	0.0001	0.56	.575
Superior longitudinal fasciculus-temporal bundle; $F(7,140) = 1.57$, $p = .148$, $R^2_{adj} = 0.03$				
	Intercept	0.4044	100.59	<.001***
	MEIM	-0.0001	-0.05	.961
	ADI	<-0.0001	-0.25	.807
	Gender	0.0008	0.23	.820
	Income	<0.0001	0.05	.963
	Age	-0.0002	-0.96	.338

PCL-5	0.0002	1.87	.064
ADI x MEIM	0.0002	1.91	.058
Superior longitudinal fasciculus-parietal bundle; $F(7,140) = 2.67$, ***p* = .013*,** $R^2_{adj} = 0.07$			
Intercept	0.3991	89.11	<.001***
MEIM	-0.0001	-0.03	.980
ADI	0.0001	0.80	.423
Gender	0.0044	1.07	.288
Income	0.0007	1.05	.295
Age	-0.0003	-1.78	.077
PCL-5	0.0002	1.92	.057
ADI x MEIM	**0.0003**	**2.30**	**.023***
Inferior longitudinal fasciculus; $F(7,140) = 1.57$, $p = .147$, $R^2_{adj} = 0.03$			
Intercept	0.4583	74.84	<.001***
MEIM	0.0038	1.16	.247
ADI	-0.0002	-1.15	.253
Gender	-0.0071	-1.27	.206
Income	0.0004	0.40	.689
Age	-0.0004	-1.61	.111
PCL-5	0.0001	0.68	.501
ADI x MEIM	0.0001	0.96	.337
Corticospinal tract; $F(7,140) = 2.10$, ***p* = .048*,** $R^2_{adj} = 0.05$			
Intercept	-0.4249	121.93	<.001***
MEIM	-0.0021	-1.12	.264
ADI	-0.0001	-0.86	.389
Gender	-0.0011	-0.35	.728
Income	0.0010	1.77	.078
Age	<-0.0001	-0.17	.868
PCL-5	**0.0002**	**2.02**	**.045***
ADI x MEIM	0.0001	0.95	.343
Cingulum-angular bundles; $F(7,140) = 0.77$, $p = .612$**,** $R^2_{adj} = -0.01$			
Intercept	0.3411	49.09	<.001
MEIM	0.0010	0.28	.781
ADI	**-0.0004**	**-2.09**	**.039***
Gender	-0.0073	-1.15	.254
Income	-0.0014	-1.29	.201
Age	0.0001	0.43	.668
PCL-5	<0.0001	0.24	.809
ADI x MEIM	<-0.0001	-0.30	.976
Cingulum-cingulate gyrus; $F(7,140) = 1.88$, $p = .078$**,** $R^2_{adj} = 0.04$			
Intercept	0.5132	71.35	<.001***
MEIM	-0.0006	-0.16	.875
ADI	0.0003	1.66	.099
Gender	-0.0122	-1.86	.066
Income	0.0009	0.83	.409
Age	-0.0002	-0.71	.477

PCL-5	0.0002	1.23	.221
ADI x MEIM	0.0003	1.80	.074

ADI: Area Deprivation Index (National Ranking); **PCL-5:** PTSD Checklist for DSM-5; * $p < .05$.

Neighborhood Disadvantage Does Not Explain Link Between White Matter Tract Integrity and Non-remitting PTSD Symptoms

Results of the Pearson's correlation revealed CAPS-5 scores (n = 132) were predicted by lesser integrity in the CCG, $r(130) = -0.17$, $p = .046$. Neighborhood disadvantage was not significantly associated with CCG integrity ($r(146) = .11$, $p = .172$) or CAPS-5 , $r(130) = 0.02$, $p = .805$. Recent work using the same sample (Weis et al., 2021) demonstrated that after controlling for relevant covariates (e.g., PCL-5 scores) CCG FA values do not predict PTSD symptoms. For this reason, no mediation analysis was performed.

Discussion

In a sample of recently traumatically injured adults, neighborhood disadvantage was associated with white matter tract integrity, even after accounting for income, gender, age, race and ethnicity, and baseline PTSD symptoms. Using an MMR model, we demonstrated ADI significantly predicted FA values in white matter tracts. Although univariate regressions revealed significant associations between FA values and gender, age, PCL-5 scores, and race and ethnicity, these variables were not significant in the MMR model. These findings align with the current literature on socioeconomic circumstances and brain morphology, replicating previous studies linking less integrity to neighborhood disadvantage exposure (Bell et al., 2021; Gianaros et al., 2013). We found initial evidence that racial-ethnic identity buffers against the effects of

neighborhood disadvantage on the superior longitudinal-parietal bundle (SLFP). For individuals who lived in more disadvantaged neighborhoods, higher MEIM scores were related to greater tract integrity. Surprisingly, although we hypothesized that neighborhood disadvantage would help explain relationships between white matter tract integrity and non-remitting PTSD symptoms, we found no such association.

The significant effect of neighborhood disadvantage in the MMR model was driven by two tracts: CAB and FMAJOR. The relationship between ADI and CAB FA corroborates Bell and colleagues (2021) finding that greater neighborhood disadvantage is significantly related to less integrity in CAB (see also Gianaros et al., 2013). This fronto-limbic bundle has received considerable attention in studies on emotion and is clearly clinical relevant, with studies implicating less CAB integrity across psychiatric conditions, including PTSD and depression (Bubb et al., 2018; Harnett et al., 2020; Roeckner et al., 2021). The behavioral effects related to less CAB integrity appear widespread, with studies suggesting lower FA is related to poor performance across neurocognitive domains, including working memory and decision making (Averill et al., 2018). Still, the most consistent functional link is between greater integrity and emotion regulation (Bubb et al., 2018), offering a neurobiological pathway linking neighborhood disadvantage to indices of emotion dysregulation (Barch et al., 2020; Cooley et al., 2019; Hackman et al., 2019; Sun et al., 2020).

Greater neighborhood disadvantage was also related to lower FA in the posterior FMAJOR, which broadly supports interhemispheric communication and integration. Although FMINOR is primarily considered the critical interhemispheric bundle for affective information, as it connects the two frontal lobes (Versace et al., 2015),

FMAJOR assists with visual processing of affective stimuli. Evidence of this role is found in clinical studies; individuals with PTSD who experienced a natural disaster showed greater FA in FMAJOR, perhaps related to the greater cognitive demands on visual processing underlying hyper-vigilance and mental imagery during flashbacks (Li et al., 2016). In general, people living in urban areas typically encounter more complex visual (and auditory) stimuli which demand more attentional focus (Mennis et al., 2018). Using the same sample, Webb and colleagues (Project 2), demonstrated that lower socioeconomic position (i.e., income) was significantly associated with greater connectivity between the anterior cingulate cortex and visual regions (e.g., primary visual cortex), suggesting lower socioeconomic position is related to a greater demand on visual processing resources. Less structural integrity may promote an overcompensation in functional connectivity, although this theory counters previous work suggesting greater FA is associated with greater connectivity and activation in related structures (but see Marstaller et al., 2015). Future multi-model imaging studies may reveal a clearer picture of how neighborhood disadvantage relates to structure and function.

To our knowledge, no previous studies have examined any neural correlates of racial-ethnic identity. A strong connection to and identification with one's own ethnoracial group may correspond to increases in various protective elements (Lardier Jr. et al., 2021). For example, greater racial-ethnic identity can represent strong social networks and reflect feelings of solidarity, especially when confronted with discrimination. For ethnoracially marginalized individuals, racial-ethnic identity is a critical component of self-concept and is correlated with higher levels self-esteem,

another robust resilience factor (Phinney et al., 1997; Pyant & Yanico, 1991). Given the plethora of evidence demonstrating racial-ethnic identity plays a key role in the daily functioning and health of minoritized people, the lack of emphasis on this culturally relevant resilience factor in neuroscience is unacceptable. The typical deficit-only models have been harmful to ethnoracially minoritized groups because, without careful consideration, these models are capable of: placing blame on the individual, exonerating oppressive structures, and minimizing unique individual and cultural strengths (Davis, 2019).

In our exploratory aim, we observed that racial-ethnic identity does indeed buffer against deleterious effects of neighborhood disadvantage on integrity of the SLFP (corresponds to SLF III). This tract is a large component of the superior longitudinal fasciculus linking frontal (dorsolateral prefrontal cortex) and parietal regions (angular and supramarginal gyrus; Madhavan et al., 2014; Yendiki et al., 2011). SLFP is involved in cognitive control and recruited to process affective information (Madhavan et al., 2014). Less integrity of the SLFP is also linked to psychopathology, including major depressive disorder and PTSD (Daniels et al., 2013; Lai & Wu, 2014; Na et al., 2018). In general, emotion regulation strategies recruit frontoparietal networks (Li et al., 2021). Thus, greater SLFP integrity is critical for effective deployment of emotion regulation strategies, an ability essential to resilience. Empirical work aligns with this hypothesized mechanism: Parkinson and Wheatley (2014) demonstrated greater FA in the SLFP was related to a measure of empathy, a necessary competency for social interactions and a resilience indicator. Although additional support is required, our preliminary investigation

suggests racial-ethnic identity is protective, with neurobiological effects comparable to other resilience factors.

For individuals living in more advantaged neighborhoods, racial-ethnic identity appeared to be related to less SLFP integrity. Given the racialization of neighborhood disadvantage, this side of the interaction should be interpreted cautiously. That is, this result should *not* be interpreted as high racial-ethnic identity in advantaged neighborhoods is harmful, rather that very few participants from ethnoracially minoritized groups (*M* ADI = 75.29, *SD* = 17.73; range: 31 - 100) live in low ADI neighborhoods. White participants disproportionately reside in more advantaged neighborhoods (*M* ADI = 51.60, *SD* = 22.68; range: 11 - 100). White participants also scored significantly lower on MEIM compared to participants from ethnoracially marginalized groups, ultimately confounding race, ethnicity, MIEM, and neighborhood disadvantage. Because there are few participants living in more advantaged neighborhood who reported higher levels of racial-ethnic identity, the estimation of this side of the model is based on fewer observations. A relevant concept is racial residential segregation, an upstream determinant that has maintained ethnoracial disparities and slowed action to address neighborhood disadvantage (Bennett, 2011; Turner et al., n.d.). Although we did not include an index of residential segregation, we consider our findings to reflect the realities of structural racism and encourage future neuroscience work to better explore the complexities of race, ethnicity, and neighborhood factors.

Finally, we did not detect significant associations between neighborhood disadvantage, FA, and six-months post-injury PTSD symptoms (CAPS-5). Using the

same sample, Weis and colleagues (2021) showed white matter tract integrity did not predict PTSD symptoms. In the current study we found a significant correlation between CCG FA and CAPS-5. However, after adjusting for relevant covariates this relationship was only marginally significant (Weis et al., 2021). Explanation as to why our results do not align with other published CCG FA – PTSD associations (see meta-analysis: Ju et al., 2020), may lie in our sample characteristics. The majority of participants had subthreshold PTSD symptoms at baseline (PCL-5 *M* = 25.43; provisional diagnosis cut-off = 31; Blevins et al., 2015) and approximately 18% met criteria for PTSD at six-months (Weis et al., 2021). Our sample was largely resilient in the aftermath of a traumatic injury and rates of PTSD were significantly lower compared to other studies. Still, our findings underscore the importance of studying resilience. Our sample was largely disadvantaged and relatedly, included a greater representation of individuals from ethnoracially marginalized groups. These are factors which the literature might suggest are related to *greater* risk for PTSD after trauma (Shalev et al., 2019), and yet we observe low overall rates of symptom severity. Resilience factors are clearly at play and understanding the relationship between risk and resilience factors on neurobiology will undoubtedly help improve trauma outcomes.

Several limitations temper the generalizability of our findings. First, our sample was comprised of individuals from various ethnoracial identities, however due to small sample sizes across different groups, we grouped participants as either White or as a member of an ethnoracially minoritized group. Although MEIM scores are relatively stable across ethnoracially marginalized groups, this lack of nuance diminished our ability to observe potential differences. In fact, this approach minimizes the struggle and

strengths that are specific within different ethnoracial groups. Large-scale population-based neuroscience has a unique position to better describe the neural intersections between neighborhood factors, race, and ethnicity. These larger studies may also provide more geographical diversity and contextual data. ADI only captures socioeconomic aspects of the neighborhood. As previously mentioned, we did not evaluate the extent of residential segregation or other neighborhood components that may influence people's mental health (e.g., exposure to community violence, neighborhood cohesion, greenspace, walkability, etc.). Future directions include more comprehensive assessment of neighborhood factors in studies with clinical samples, principally in those attempting to predict future mental health symptoms.

Conclusion

Identifying the mechanisms by which neighborhoods impact mental health has emerged as a new field of neuroscience. Across modalities, neighborhood disadvantage is significantly associated with alterations to brain function and structure. We add to this literature by showing that neighborhood factors are significantly associated with less integrity in white matter tracts, even after accounting for PTSD symptoms from a recent trauma. Although replication of this work is useful in documenting sensitive periods, affected regions, and behavioral consequences, a risk-resilience approach offers an opportunity to identify protective factors, showcasing people's resilience in the face of adversity. Here, we found individuals living in disadvantaged neighborhoods who had a strong sense of belonging to their racial and/or ethnic group had greater integrity in the SLFP. Given the existing ethnoracial mental health disparities, studying culturally relevant protective factors as they relate to risk factors driven by structural racism (e.g.,

neighborhood disadvantage, discrimination, etc.) is needed. This is an exciting direction for neuroscience; work in this area may spur interventions that uplift culturally relevant protective characteristics and yield new insight into the heterogeneity of brain structure and function.

Interactions Between Socioeconomic Circumstances and Protective Factors on Anterior Cingulate Cortex Resting-State Functional Connectivity

The percentage of Americans living in socioeconomically distressed or disadvantaged neighborhoods has been on the rise since 2000 (National Equity Atlas, 2021). Though the public health repercussions of this upward trend are still evolving, research has demonstrated disadvantaged neighborhoods are associated with a higher prevalence of psychiatric conditions (Sundquist et al., 2015). Exposure (i.e., duration and magnitude) to neighborhood disadvantage and the strength of neighborhood-mental health associations varies across people and places, with disparities directly attributed to structural racism. The explicit racialization of local contexts (e.g., neighborhoods) has occurred through racist policies, such as the systematic underfunding of public resources (e.g., schools) in majority Black and Brown neighborhoods, the denial of housing loans based on skin color, and inequitable land use planning (e.g., placement of factories, highways, and parks; Riley, 2018). Context racialization, coupled with ethnoracial differences in socioeconomic circumstances (i.e., Race x Class interactions) and individual experiences (e.g., differences in types of trauma, experiences of discrimination), ties structural racism to ethnoracial mental health disparities (Berger & Sarnyai, 2015; Harnett & Ressler, 2021; Williams, 2018).

Elucidating the neurobiological effects of neighborhood disadvantage - while considering interactions between neighborhoods and minoritized group status - may offer insight into how myriad sources of oppression impact brain health and which protective factors buffer against harm. There is an urgent need to understand resilience in the context of both health disparities and socioeconomic inequities as this may lead

to additional investment (financial and/or cultural) in these factors or incite political action to address inequities. We sought to characterize how socioeconomic circumstances and resilience factors relate to a known neural correlate of emotion regulation, the anterior cingulate cortex (ACC).

Emotion (Dys)regulation as a Link Between Neighborhoods and Mental Health

Emotional dysregulation, a universal hallmark of both internalizing and externalizing disorders, is a compelling mediator between neighborhoods and mental health. Individual-level adversity, such as childhood maltreatment, are well known to hinder emotion regulation (e.g., Bradley et al., 2011; Nickerson et al., 2015); however, neighborhood-level variables also alter emotion regulation. General neighborhood distress, frequently operationalized by greater exposure to violence and socioeconomic disadvantage (a proxy for limited access to material and social resources), is associated with difficulties regulating emotion (Barch et al., 2020; Cooley et al., 2019; Hackman et al., 2019; Sun et al., 2020). Importantly, these relationships are not fully explained by individual-level variables, including metrics of individual socioeconomic position (i.e., income or education).

Neighborhood disadvantage has been linked to alterations in the neural correlates of emotion regulation (Bell et al., 2021; Farah, 2017; Finegood et al., 2017; Gianaros et al., 2015; Hackman & Farah, 2009); however, the majority of these studies have been conducted in healthy/asymptomatic participants and adolescents. Yet effective emotion regulation is especially critical in the face of an acute traumatic or stressful event, when regulatory strategies are triggered and ultimately tested (Raio et al., 2013). In the current study, we investigated the link between a brain region

subserving emotion regulation and neighborhood disadvantage in trauma exposed adults. The unique sample provided an opportunity to test neurobiological mechanisms that may explain why people residing in more disadvantaged neighborhoods have an increased risk of developing psychiatric conditions, including post-traumatic stress disorder (PTSD; e.g., Cheng & Mallinckrodt, 2015; Dinwiddie et al., 2013; López et al., 2017; Spoont et al., 2020). This work is also fundamental in the path to health equity, as people from minoritized groups are more likely to reside in disadvantaged neighborhoods (Berger & Sarnyai, 2015; Williams, 2018).

PTSD is a severe and devastating psychological consequence of experiencing a traumatic event (Kessler et al., 2017). Up to 30% of individuals who experience a traumatic injury will develop persistent PTSD symptoms (Shih et al., 2010), which include heightened arousal and reactivity, avoidance of trauma reminders or related-stimuli, intrusive thoughts and feelings, and negative cognition and mood. Notably, PTSD is the only psychiatric condition where the onset of the disorder, a trauma, is identifiable. The Diagnostic and Statistical Manual of Mental Disorders, (DSM-5; American Psychiatric Association, 2013) constrains diagnosis of PTSD, until symptoms have been present for at least one-month post-trauma. As distress and acute symptoms in the early aftermath of a trauma is common, and even adaptive. Together, these diagnostic prerequisites position PTSD as a “model” psychiatric condition for the study of risk and resilience prediction factors.

Though a number of pre-trauma (e.g., age, gender, trauma history), peri-trauma (e.g., trauma type, peri-traumatic dissociation), and post-trauma (e.g., social support, coping strategies) features influence PTSD development and trajectory, no single risk or

resilience factor completely predicts who will go on to develop PTSD (e.g., Kiely et al., 2006; Schultebraucks et al., 2020; Shalev et al., 2019; Wshah et al., 2019). Evidence links emotion dysregulation to post-trauma trajectories, such that neural and behavioral indices of emotion regulation predict resilience and indices of emotion dysregulation predict non-remitting PTSD symptoms (e.g., Christ et al., 2021; Cisler et al., 2016; Fitzgerald et al., 2018; Harnett et al., 2021; Nickerson et al., 2015; Roeckner et al., 2021). Thus, pre-trauma emotion dysregulation is a risk factor for PTSD. This supports a theory that living in a disadvantaged neighborhood may influence trauma-related psychopathology via its link to emotion dysregulation and impact on relevant neural substrates. Among the neural correlates of emotion regulation, the ACC has emerged as a particularly important structure.

The Anterior Cingulate Cortex's Role in Psychological Resilience

The cingulate cortex is an extraordinarily diverse structure, divided into at least six functionally and structurally distinct subregions (Jin et al., 2018). A major distinction is between the anterior cingulate cortex (ACC), considered a node in the salience network and the posterior cingulate cortex, a node of the default mode network. The precise function of the ACC has been debated (Etkin et al., 2011; Stevens et al., 2011). Nevertheless empirical work suggests all the ACC subregions, including the dorsal and ventral-rostral subregions, contribute to emotion regulation through bidirectional communication with other affective regions (Egner et al., 2008; Etkin et al., 2006, 2010, 2011). The amygdala and anterior insula perform initial processing of salient stimuli whereas the ACC facilitates the corresponding cognitive and behavioral responses (Geng et al., 2016). In this role, the ACC is heavily involved in the "management" of

emotional information. Notably, ACC activation is associated with deployment of self-regulatory strategies, such as reappraisal and distraction (e.g., Bryant et al., 2020; Giuliani et al., 2011; Ochsner et al., 2002).

Aberrant ACC connectivity and activation is linked to measures of emotion dysregulation, further underscoring its role in emotion conflict and implementation of resolution strategies (reviewed in Bush et al., 2000). In individuals with PTSD, atypical activity of the ACC during resting-state fMRI and affective tasks is consistently associated with symptom severity (Kennis et al., 2015; Koch et al., 2016; Offringa et al., 2013; Shin et al., 2001). For example, greater connectivity between ACC and anterior insula can differentiate individuals with PTSD from trauma exposed controls (Chen et al., 2019; Zhang et al., 2016).

Akin to the neural features of PTSD, neighborhood disadvantage is associated with aberrant functional connectivity and activation of regions underlying emotion regulation (e.g., Assari, 2020; Harnett et al., 2019; Gard et al., 2019, 2018; Ramphal et al., 2020; Webb et al., 2021). We previously found greater neighborhood disadvantage was associated with greater functional connectivity between the anterior insula and ventrolateral prefrontal cortex, two regions which interact during the evaluation and (prefrontal-initiated) suppression of emotions (Webb et al., 2021). A handful of studies have detailed effects of socioeconomic circumstances (neighborhood or individual) on the ACC function. Adolescents residing in more disadvantaged neighborhoods show decreased efficiency and fewer connections between salience network nodes, including the ACC (Gellci et al., 2019). Task-based studies strengthen the theory that socioeconomic circumstances may impact emotion dysregulation via effects on the

ACC. For example, children raised in families with lower socioeconomic positions, show disrupted ACC activity and connectivity during reward tasks (Gianaros et al., 2011; Palacios-Barrios et al., 2021).

The ACC's role in deploying emotion regulation strategies makes an argument for examining how this region could be a neural correlate of resilience, contributing to effective emotion regulation in the face of adversity. In the context of PTSD, resilience refers to an individual's resistance to the negative psychological effects of trauma. Although resilience has historically been conceptualized as an individual state or difference, more recent conceptualizations call for envisioning resilience as a reflection of the "quality of the environment and its capacity to facilitate growth" as well as a construct that is sensitive to cultural differences (Ungar, 2013). Anterior cingulate cortex functional connectivity has been noted as a sensitive measure for stress resilience (Shao et al., 2018). For example, resting-state connectivity of the ACC with precentral/postcentral gyrus and left middle frontal gyrus is associated with higher levels of social support, a measure that bestows resilience in the context of PTSD (Chen et al., 2019). Taking advantage of social support available could be an adaptive behavioral response to stress, facilitated to some degree by the ACC. However, no studies to date have examined whether the relationship between neighborhood disadvantage and ACC connectivity changes based on resilience factors, especially those reflecting cultural values.

Current Study

We recruited over two-hundred participants who experienced a traumatic injury from an urban Emergency Department in a Level I Trauma Center. To shed light on the

neural correlates of socioeconomic circumstances, we evaluated whether neighborhood disadvantage and individual income were significantly associated with ACC resting-state functional connectivity even after adjusting for each other and relevant covariates (e.g., PTSD symptoms).

As a region assumed to be a key correlate of resilience, our study probed whether the effect of socioeconomic circumstances (both income and neighborhood disadvantage) on ACC connectivity varied depending on individual resilience markers. To meet this aim, we used two well-documented resilience variables, a general social support measure (the Medical Outcome Study Social Support Survey; MOS) and a social support measure more sensitive to cultural differences (i.e., the Multi-Ethnic Identity Measure; MEIM). The MEIM captures one's sense of belonging to their own ethnoracial group (Phinney, 1992; Phinney et al., 1997). Members of ethnoracially marginalized groups report significantly higher affiliation and commitment to their respective group compared to White people (e.g., Bracey et al., 2004; Brown et al., 2014; Williams et al., 2012). Higher scores on the MEIM are associated with more positive health outcomes and general well-being (Phinney et al., 1997; Roberts et al., 1999). The sociopolitical positions of ethnoracially minoritized individuals may alter the functioning of both income and racial-ethnic identity on the brain; therefore, we performed exploratory analyses to determine if the interactions between resilience factors and socioeconomic circumstances on neurobiology were consistent across ethnoracial groups (i.e., three-way interactions). Finally, disrupted ACC connectivity may represent neurobiological vulnerabilities and be associated with non-remitting

PTSD symptoms. Therefore, we evaluated whether ACC connectivity values (i.e., significant clusters from earlier analyses) were predictive of future PTSD symptoms.

Method and Materials

Participants

The participants in Project 2 were recruited as part of the iSTAR project (detailed in Project 1). Nine hundred and sixty-nine traumatically injured individuals were approached in an Emergency Department in the United States Midwest. Individuals were considered eligible if they: had experienced a traumatic injury, were between the ages of 18 and 65 years old, could speak and understand English, and could schedule a research visit within 30 days of the injury. Participants were excluded if they scored 13 or higher on the Glasgow Coma Scale (Sternbach, 2000), had a spinal cord injury with neurological deficits, or were diagnosed with any neurological condition affecting brain structure or function. Additional exclusion criteria included: a self-inflicted traumatic injury, severe vision or hearing impairments, history of psychotic or manic symptoms, current antipsychotic medication use, substance abuse, on a police hold to be released to jail, and/or any contraindications for MRI scanning including metal objects or fragments in the body, claustrophobia, and pregnancy or planned pregnancy within the next 6 months.

Two-hundred and fifteen individuals met eligibility criteria and were enrolled in the Imaging Study of Trauma and Resilience (iSTAR; Bird et al., 2021; Webb et al., 2021; Weis, Webb, Huggins, et al., 2021; Weis, Webb, Stevens, et al., 2021). The study protocol was approved by the Institutional Review Board at the Medical College of

Wisconsin. All participants provided written informed consent and were financially compensated for their time.

Procedure

The iSTAR procedure in Project 2 is identical to the procedure described in Project 1.

Measures

Neighborhood Socioeconomic Disadvantage

A neighborhood was defined by the census geographical unit block-group. To quantify neighborhood socioeconomic disadvantage, Area Deprivation Index (ADI) rankings were derived from participant's home addresses. ADI is an established measure of neighborhood disadvantage which uses 17 variables (e.g., housing quality, income, etc.) from the 2011-2015 American Community Survey (administered by the Census Bureau; Hu et al., 2018; Hunt et al., 2020; Kind & Buckingham, 2018; Knighton et al., 2016; Singh, 2003). ADI ranges from 1 to 100, where "1" indicates the most advantaged neighborhood and "100" represents the most disadvantaged neighborhoods.

Individual Demographics

At T1, participants self-reported their race, ethnicity, gender, annual household income, and age. Race was asked in compliance with the NIH revised standards which included the following categories: "American Indian or Alaska Native", "Asian", "Black or African American", "Native Hawaiian or Other Pacific Islander", or "White". Two categories on ethnicity were presented: "Hispanic or Latino" and "Not Hispanic or Latino". Due to small sample sizes across racial and ethnic groups, a binary variable

was created. “0” indicated the participant identified as a member of a racially and/or ethnically marginalized group and “1” designated that the participant identified as White.

Participants were asked to indicate whether their gender was “male”, “female”, or other. A selection of the “other” category was followed up with a text box in which participants could type. Annual household income was reported on a semi-continuous scale that ranged from “1” (the household earned between $0-10,000 annually) to “11” (reflected an annual household income of over $100,000).

T1 Post-Traumatic Stress Symptoms

At T1, participants completed the PTSD Checklist for DSM-5 (PCL-5; Blevins et al., 2015). This 20-item questionnaire was used to assess acute post-trauma PTSD symptoms, corresponding to those listed in the DSM-5. Using a five-point Likert scale, participants rated how much each of the symptoms bothered them: from 1 (*not at all*) to 5 (*extremely*). A total symptom severity score was created by summing the scores for all items.

General Social Support

At T1 participants completed the 19-item Medical Outcomes Study Social Support Survey (MOS; Sherbourne & Stewart, 1991). Using a 5-point Likert scale, participants indicate how much of the time each type of support is available to them, from 1 (*none of the time)* to 5 (*all the time).* Four sub-scales of social support were included: emotional/information (“someone you can count on to listen to you when you need to talk”), tangible (“someone to help you if you were confined to bed”), affectionate (“someone who shows you love and affection”), and positive social interactions (“someone to have a good time with”). A final score was created by averaging all items.

Multi-Ethnic Identity Measure

To evaluate the participant's affiliation to their ethnoracial group, the multi-ethnic identity measure was administered at T1 (Phinney, 1992). This 6-item questionnaire has two subscales examining both exploration ("I have spent time trying to find out more about my own ethnic group, such as its history, traditions, and customs") and commitment ("I feel a strong attachment towards my own ethnic group") to an ethnoracial group. Participants rated how much they agreed with each statement on a scale ranging from 1 (*strongly disagree)* to 5 (*strongly agree)*. Responses to all items were averaged to create a total score.

T2 Post-Traumatic Stress Disorder Symptoms

Six-month PTSD symptom severity and diagnostic status was evaluated at T2. Trained research staff members conducted the 30-item semi-structured Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2018). During the interview, the researcher queried the index trauma and evaluated severity and frequency of PTSD symptoms. A total symptom severity score was calculated by summing the scores of 20 items directly corresponding to DSM-5 symptoms. CAPS-5 interviews were audio-recorded and 20% of all recordings were subjected to reliability checks by another staff member. There was excellent reliability across CAPS-5 administration within the study (interclass correlation coefficient = 0.96, with 95% confidence interval [0.93, 0.98]).

Imaging Acquisition

MRI images were collected using a 3.0 Tesla short-bore General Electric Signa Excite system (Waukesha, WI) with a 32-channel head-coil. High resolution T1-

weighted anatomical images were acquired in a sagittal orientation (repetition time [TR] = 8.2ms; echo time [TE] = 3.2ms; Field of View (FOV) = 24 cm; flip angle = 12°; voxel size = 1 x 0.9375 x 0.9375mm). During an 8-minute resting-state scan, participants were asked to keep their eyes open and focus on a fixation cross. 240 volumes were obtained using the following parameters: TR = 2s; TE = 25ms; FOV = 22.4mm; flip angle = 77°; matrix = 64 x 64; slice thickness: 3.5mm.

Imaging Processing

Resting-State Functional Connectivity Preprocessing

Resting-state images were preprocessed in CONN (version 20). The first three volumes were discarded to allow for magnetic field stabilization, and the remaining volumes underwent motion-correction using a six-parameter linear transformation. Volumes were normalized to the Montreal Neurological Institute (MNI 152) template and blurred with a 4-mm full-width-at-half maximum kernel. To reduce signal-to-noise ratio, a temporal bandpass was applied (0.01-0.1 Hz). Head motion parameters and white matter and cerebrospinal fluid signal were included as nuisance covariates in first-level analyses. Volumes with more than 0.3mm frame-wise displacement were scrubbed and participants with more than 20% of the volumes scrubbed were excluded. The region of interest ACC seed was created in FreeSurfer (version 5.3; Fischl, 2004; http://surfer.nmr.mgh.harvard.edu/) by combining the caudal anterior cingulate and rostral anterior cingulate regions (structural labels in default FreeSurfer parcellation; Figure 2.1).

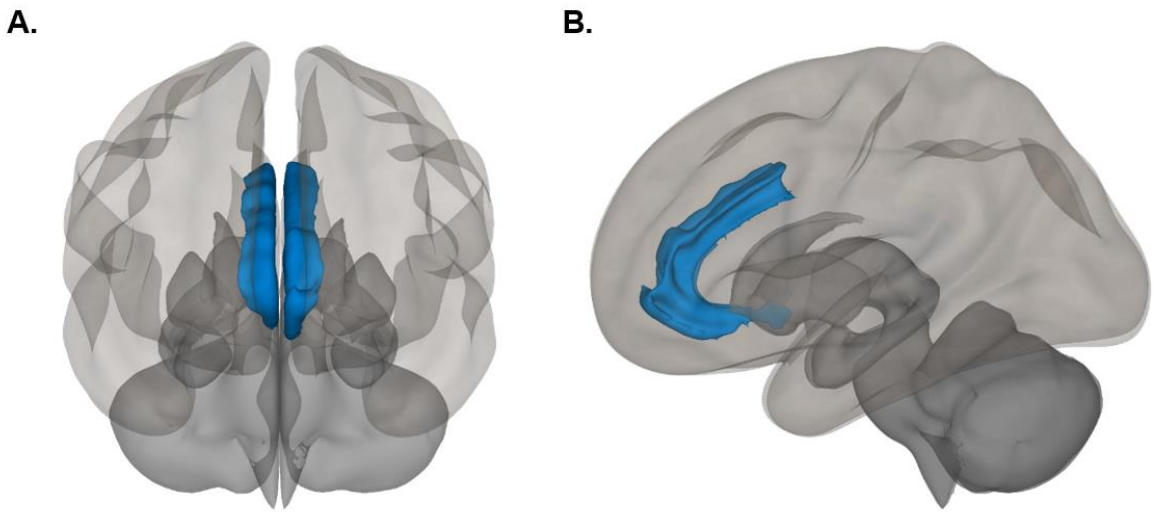

Figure 2.1. **(A) Anterior and (B) left hemisphere view of anterior cingulate cortex region of interest.** The seed region was created by combining the caudal anterior and rostral anterior cingulate regions from FreeSurfer.

Analysis Strategy

Of the 215 enrolled participants, 120 had useable resting-state scans (*n* = 7 were excluded due to motion; *n* = 2 excluded due to poor data quality). Six participants could not be successfully geocoded. Missing datapoints on the MOS were grand-mean imputed (*n* = 9) and missing data on the MEIM (*n* = 8) were group-mean imputed. Sample characteristics for the final 115 participants are provided in Table 1.1.

Resting-State Functional Connectivity Analysis

Seed-to-voxel analyses were conducted; in which the mean blood-oxygen-level-dependent (BOLD) signal from the ACC was correlated with that of all other voxels in the brain. Group-level general linear models (GLMs) were tested directly in CONN. Connectivity statistics were considered significant at $p < .05$, with a height threshold of p

< .001 uncorrected and a cluster-size threshold of an adjusted $p < .05$ false discovery rate (FDR).

We examined the unique effects of neighborhood disadvantage and income on resting-state connectivity patterns of the ACC after adjusting for the following covariates: PCL-5 scores (assessed at T1), gender, and age (Analysis I). We then tested interactions between socioeconomic variables and resilience variables (Analysis II). In separate GLMs, we probed whether the effect of ADI or Income on ACC connectivity varied by level of general social support (ADI x MOS and Income x MOS) or racial-ethnic identity (ADI x MEIM and Income x MEIM). Potential three-way interactions between resilience factors, socioeconomic circumstances, and ethnoracial group, were further examined in post-hoc GLMs in R (version 4.1.1; R Core Team, 2020; R markdown file with scripts is provided in Appendix 2). Significant interactions were probed by performing simple slopes analyses of MOS or MEIM at -1 standard deviation, mean, and +1 standard deviation.

We were interested in evaluating whether the significant effects of socioeconomic circumstances on ACC connectivity would help explain who develops PTSD. Using Pearson's correlations, we examined whether the ACC functional connectivity values (i.e., significant clusters from Analysis I and II) significantly predicted future PTSD symptoms (CAPS-5 scores; Analysis III).

Table 2.1. Sample Characteristics (*N* = 115)

Variable	***Mean (SD) or %***
Age (years)	32.23 years (10.75)

Gender	
Female	52% (*n* = 60)
Individual Income	
$0-10,000	20%
$10,000-20,000	12%
$20,000-30,000	16%
$30,000-40,000	9%
$40,000-50,000	10%
$50,000-60,000	6%
$60,000-70,000	8%
$70,000-80,000	8%
$80,000-90,000	<5%
$90,000-100,000	<5%
$100,000 and above	6%
Race and Ethnicity	
African American and/or Black	57%
White	30%
Hispanic or Latino	8%
Other racial/ethnic identity*	13%
Mechanism of Injury	
Motor vehicle crash	68%
Physical assault	16%
Other	14%
T1 PTSD Symptoms (PCL-5)	26.29 (16.87)
T2 PTSD symptoms (CAPS-5; n = 105)	11.65 (11.04)
MEIM	2.72 (0.83)
MOS	3.77 (0.99)
Area Deprivation Index	67.57 (21.78)

Abbreviations: **CAPS-5**: Clinician-Administered PTSD Scale for DSM-5; **MEIM:** Multigroup Ethnic Identity Measure; **MOS**: Medical Outcome Study Social Support Survey; **PCL-5:** PTSD Checklist for DSM-5; **T1:** timepoint 1 (two-weeks post-injury); **T2:** timepoint 2 (six-months post-injury); *Note*: ***** due to small sample sizes, additional self-reported racial identities have been combined.

Results

Bivariate Correlations

Relationships between continuous study measures are presented in Figure 2.2. Higher income was significantly associated with lower ADI rankings (i.e., more advantaged neighborhoods; $r(113) = -0.49$, $p < .001$), higher MOS scores ($r(113) = 0.20$, $p = .028$), and lower CAPS-5 symptom severity (n = 105; $r(103) = -0.23$, $p = .031$). Women reported greater levels of social support, ($r(113) = 0.24$, $p = .010$). MOS and MEIM scores were not related to PCL-5 (MOS: $r(113) = -0.07$, $p = .482$; MEIM: $r(113) = 0.01$, $p = .891$) or CAPS-5 symptom severity (n = 105; MOS: $r(103) = -0.07$, $p = .492$; MEIM: $r(103) < 0.01$, $p = .972$).

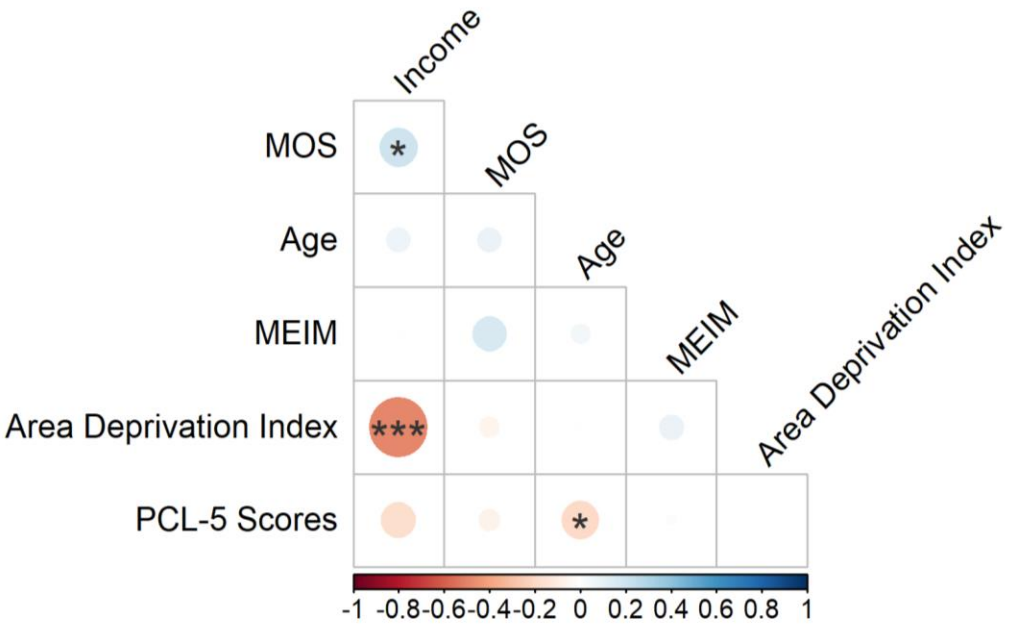

Figure 2.2. **Correlations between study measures (N = 115).** *Abbreviations:* **MEIM:** Multigroup Ethnic Identity Measure; **MOS:** Medical Outcome Study Social Support; **PCL-5:** PTSD Checklist for DSM-5; *** $p < .001$, ** $p < .01$, * $p < .05$.

Ethnoracial Differences in Study Measures

Participants (n = 80) identifying as a member of an ethnoracially minoritized group had significantly higher scores on the MEIM (M = 2.87, SD = 0.80) than White participants (n = 35; M = 2.39, SD = 0.82; $t(113) = 2.94$, $p = .004$; Figure 2.3). There was no significant difference between ethnoracial groups on the MOS, $t(113) = -0.41$, $p = .683$. Similar to Project 1, participants who identified as a member of a ethnoracially marginalized group lived in more disadvantaged neighborhoods (Figure 2.4A; M ADI = 74.36, SD = 16.99) and reported lower annual household income (approximately $10,000-20,000; M = 2.90, SD = 0.83) compared to White participants (Figure 2.4B; M ADI = 52.06, SD = 23.74; $t(113) = 5.71$, $p < .001$; approximately $50,000-60,000; M income = 6.37, SD = 3.55; $t(113) = -4.52$, $p < .001$).

Finally, there was no significant difference between groups on PTSD symptoms at baseline (White participants: M PCL-5 = 27.97, SD = 18.40; ethnoracially minoritized participants: M PCL-5 = 25.55, SD = 16.22; $t(113) = -0.71$, $p = .481$) or follow-up (White participants: M CAPS-5 = 11.15, SD = 10.83; ethnoracially minoritized participants: M CAPS-5 = 12.78, SD = 11.59; $t(103) = -0.70$, $p = .489$).

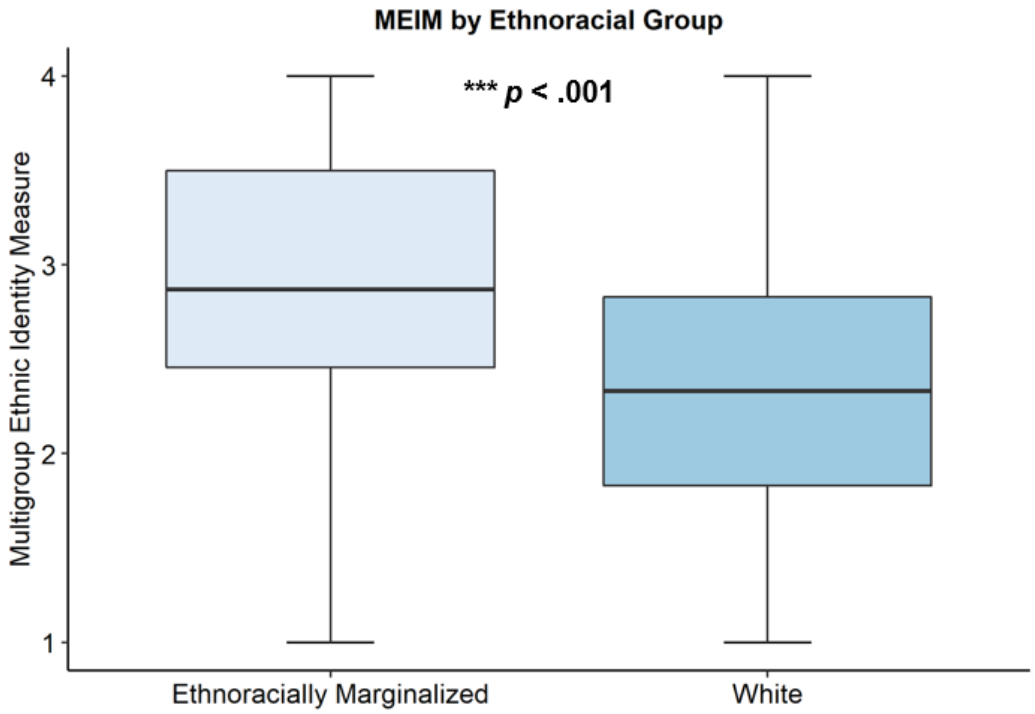

Figure 2.3. **Members of ethnoracially minoritized groups had significantly greater racial-ethnic identity.** Ethnoracially minoritized participants (n = 80) had significantly higher scores on the MEIM (MEIM M = 2.90, SD = 0.83) compared to White participants (n = 35; MEIM M = 2.41, SD = 0.80; $t(113) = 2.94$, p = .004).

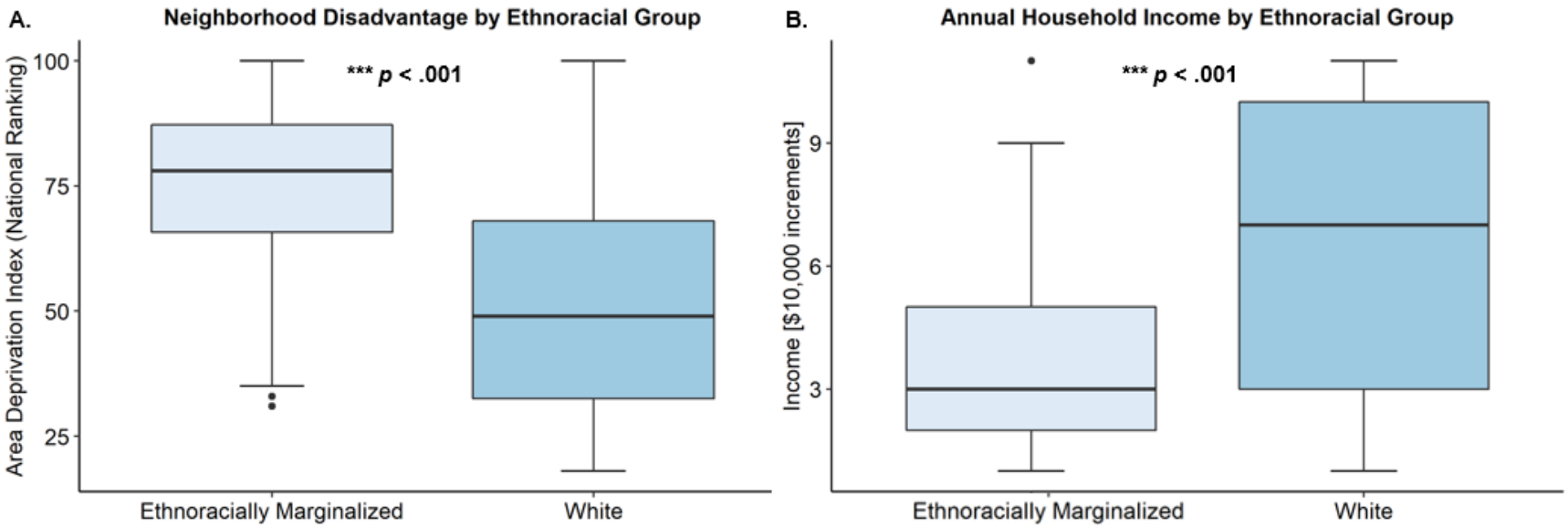

Figure 2.4. **There were significant ethnoracial differences in socioeconomic circumstances.** White participants **(A)** lived in more advantaged neighborhoods (*n* = 40; *M* = 51.6, standard deviation (*SD*) = 22.68) compared to participants from ethnoracially marginalized groups (*n* = 108; *M* = 75.28, *SD* = 17.73; *t*(113) = 5.71, *p* < .001). **(B)** Participants from minoritized groups reported significantly lower annual household income (*M* = 2.90, *SD* = 0.83) compared to White participants *(M* income = 6.37, *SD* = 3.55; *t*(113) = -4.52, *p* < .001.

Analysis I: Main Effects of Socioeconomic Circumstances on ACC Connectivity

After controlling for income, PCL-5, gender, and age, there was no significant main effect of ADI on ACC resting-state functional connectivity. Greater income was associated with less connectivity between the ACC and precuneus (MNI coordinates *x*: -06, *y*: -76, *z*: 54; cluster size *k* = 145; *p*FDR = .002; Figure 2.5A and 2.5B), cerebellum (Crus 1; MNI coordinates x: -14, y: -78, z: -22; cluster size *k* = 97; *p*FDR = .012), and the primary visual cortex (MNI coordinates *x*: 20, *y*: -80, *z*: 06; cluster size *k* = 83; *p*FDR = .002; Figure 2.5C and 2.5D).

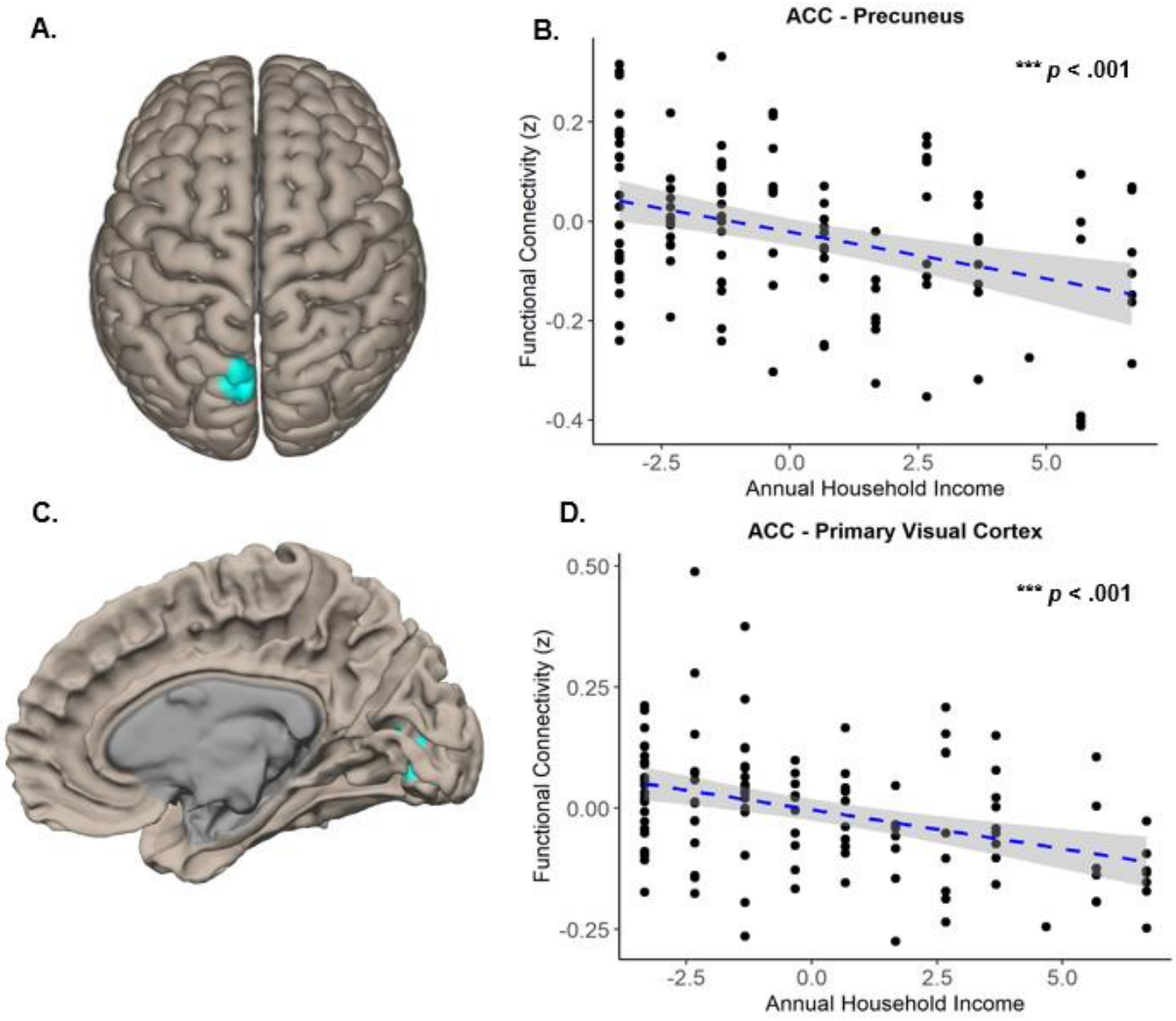

Figure 2.5. **Annual household income was significantly associated with less connectivity (Fischer's *z*-scores) between the anterior cingulate cortex (ACC) and visual regions.** Participants who reported higher annual household income showed significantly less ACC connectivity with the precuneus (**A and B;** MNI coordinates *x*: -06, *y*: -76, *z*: 54; cluster size *k* = 145; *p*FDR = .002) and primary visual cortex (**C and D;** MNI coordinates *x*: 20, *y*: -80, *z*: 06; cluster size *k* = 83; *p*FDR = .002).

Analysis II: Interactions with Resilience Factors

There was a significant interaction between ADI and MOS, such that, for individuals residing in more disadvantaged neighborhoods, greater social support was associated with greater connectivity between the ACC and putamen (Figure 2.6; MNI coordinates *x*: -26, *y*: -12, *z*: -08; cluster size *k* = 119; *p*FDR = .004) and the inferior frontal gyrus (IFG; Figure 2.7; MNI coordinates *x*: 60, *y*: 18, *z*: 24; cluster size *k* = 82; *p*FDR = .016). Results of simple slopes analyses probing the ADI x MOS interaction are provided in Table 2.2. There was no significant interaction between income and MOS.

Table 2.2. Simple slope analyses of associations between neighborhood disadvantage and ACC connectivity by levels of social support

Region	**Low MOS**			**Average MOS**			**High MOS**		
	B	*t*	*p*	*B*	*t*	*p*	*B*	*t*	*p*
Putamen	**-0.003**	**-3.16**	**.002**	<.001	0.25	.800	**0.003**	**3.87**	**<.001**
IFG	**-0.002**	**-2.28**	**.024**	**0.001**	**2.02**	**.046**	**0.005**	**5.54**	**<.001**

Abbreviations: **IFG:** Inferior frontal gyrus; **MOS:** Medical Outcome Study Social Support Survey. *Note*: low = -1 SD, high = +1 SD; **bold:** $p < .05$.

There was also a significant Income x MEIM interaction on ACC functional connectivity (results of simple slopes analyses provided in Table 2.3). Individuals reporting higher income with stronger racial-ethnic identity had greater connectivity between the ACC and right and left occipital cortices (right occipital cortex: MNI coordinates *x*: 16, *y*: -100, *z*: 02; cluster size *k* = 88; *p*FDR = .013; Figure 2.8A; left occipital cortex: MNI coordinates *x*: -20, *y*: -94, *z*: 08; cluster size *k* = 170; *p*FDR < .001; Figure 2.8B).

Table 2.3. Simple slope analyses of associations between income and ACC connectivity by levels of racial-ethnic identity

Group	Region	Low MEIM			Average MEIM			High MEIM		
		B	*t*	*p*	*B*	*t*	*p*	*B*	*t*	*p*
Ethnoracially Marginalized	Occipital Cortex (L)	**-0.028**	**-5.17**	**<.001**	**-0.011**	**-2.48**	**.015**	0.010	1.23	.222
	Occipital Cortex (R)	**-0.022**	**-3.76**	**<.001**	-0.004	-0.92	.358	**0.014**	**2.25**	**.027**
	Hippocampus	**0.032**	**4.26**	**<.001**	0.004	0.84	.400	**-0.02**	**-3.92**	**<.001**
White	Hippocampus	0.004	0.63	.527	-0.008	-1.37	.172	**-0.02**	**-2.15**	**.034**

Abbreviations: **MEIM:** Multigroup Ethnic Identity Measure. *Note*: low = -1 SD, high = +1 SD; **bold:** $p < .05$.

Individuals with greater racial-ethnic identity who reported higher income showed greater connectivity between the ACC and hippocampus (Figure 2.9A; MNI coordinates *x*: 26, *y*: -36, *z*: 00; cluster size *k* = 76; *p*FDR = .018). Follow-up tests revealed a significant three-way interaction between ethnoracial group, MEIM, and income on ACC – hippocampus connectivity. Although both groups displayed similar trends (Figure 2.9B; Table 2.3), ethnoracially minoritized participants displayed a more robust connectivity effect (MEIM x Income x Ethnoracial Group term: $\beta = 0.019$, $t(113) = 2.28$, $p = .025$; full model: $F(11,103) = 4.72$, $p < .001$, $R^2_{adj} = 0.26$). No other three-way interactions were significant and there was no significant interaction between ADI and MEIM.

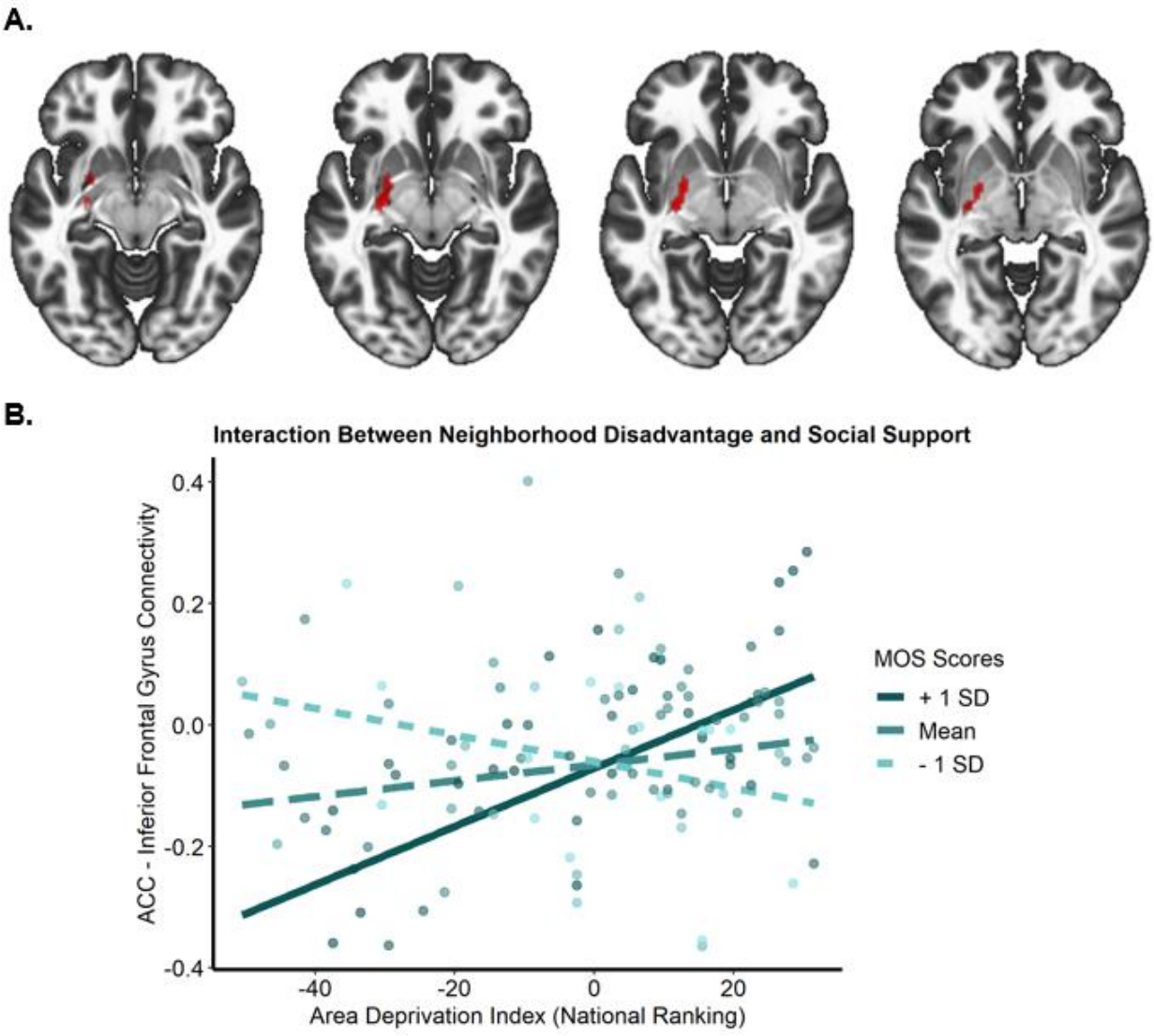

Figure 2.6. **Individuals living in more disadvantaged neighborhoods who reported greater social support available showed greater connectivity (Fischer's *z*-scores) between the anterior cingulate cortex (ACC) and putamen (MNI coordinates *x*: -26, *y*: -12, *z*: -08; cluster size *k* = 119; *p*FDR = .004).**

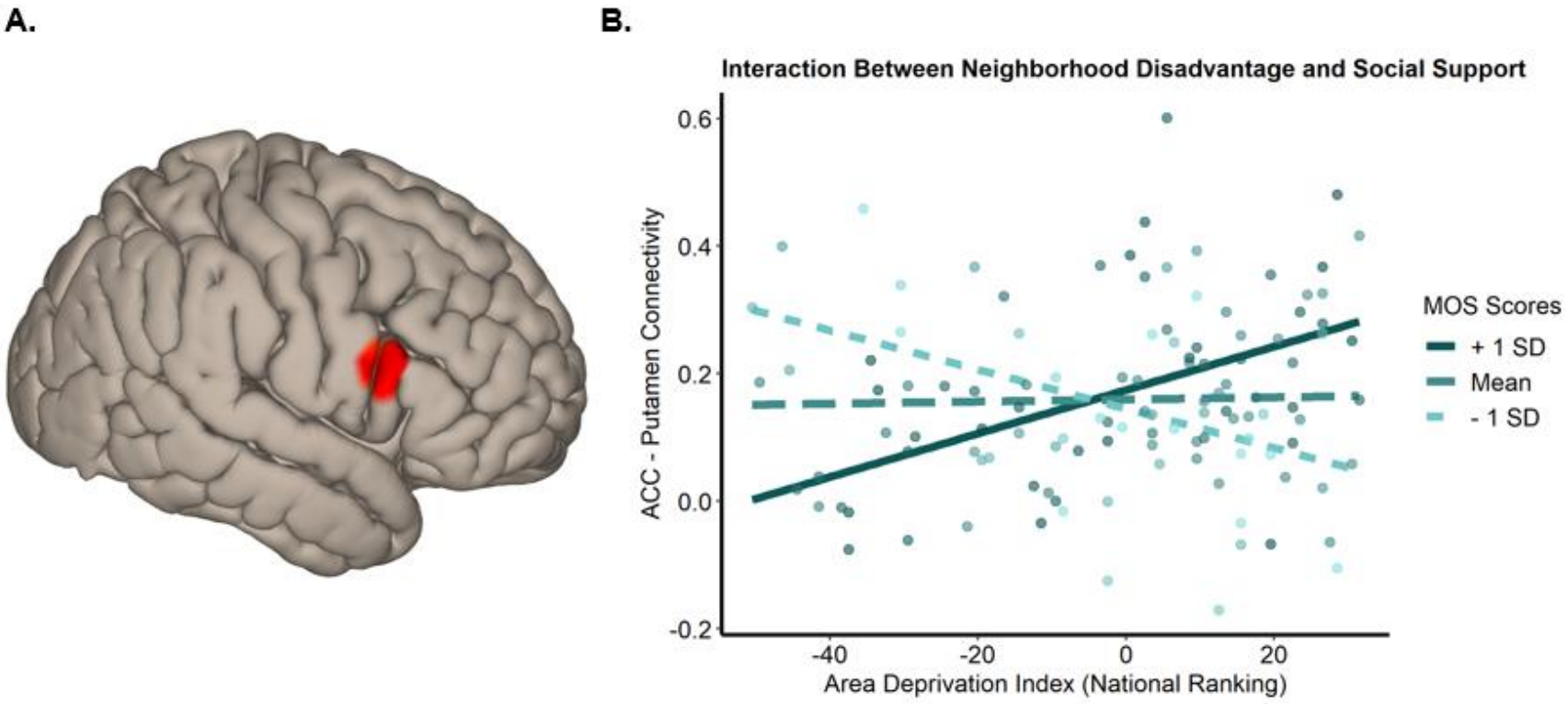

Figure 2.7. **Individuals living in more disadvantaged neighborhoods who reported greater social support available showed greater connectivity (Fischer's *z*-scores) between the anterior cingulate cortex (ACC) and inferior frontal gyrus (MNI coordinates *x*: 60, *y*: 18, *z*: 24; cluster size *k* = 82; *p*FDR = .016).**

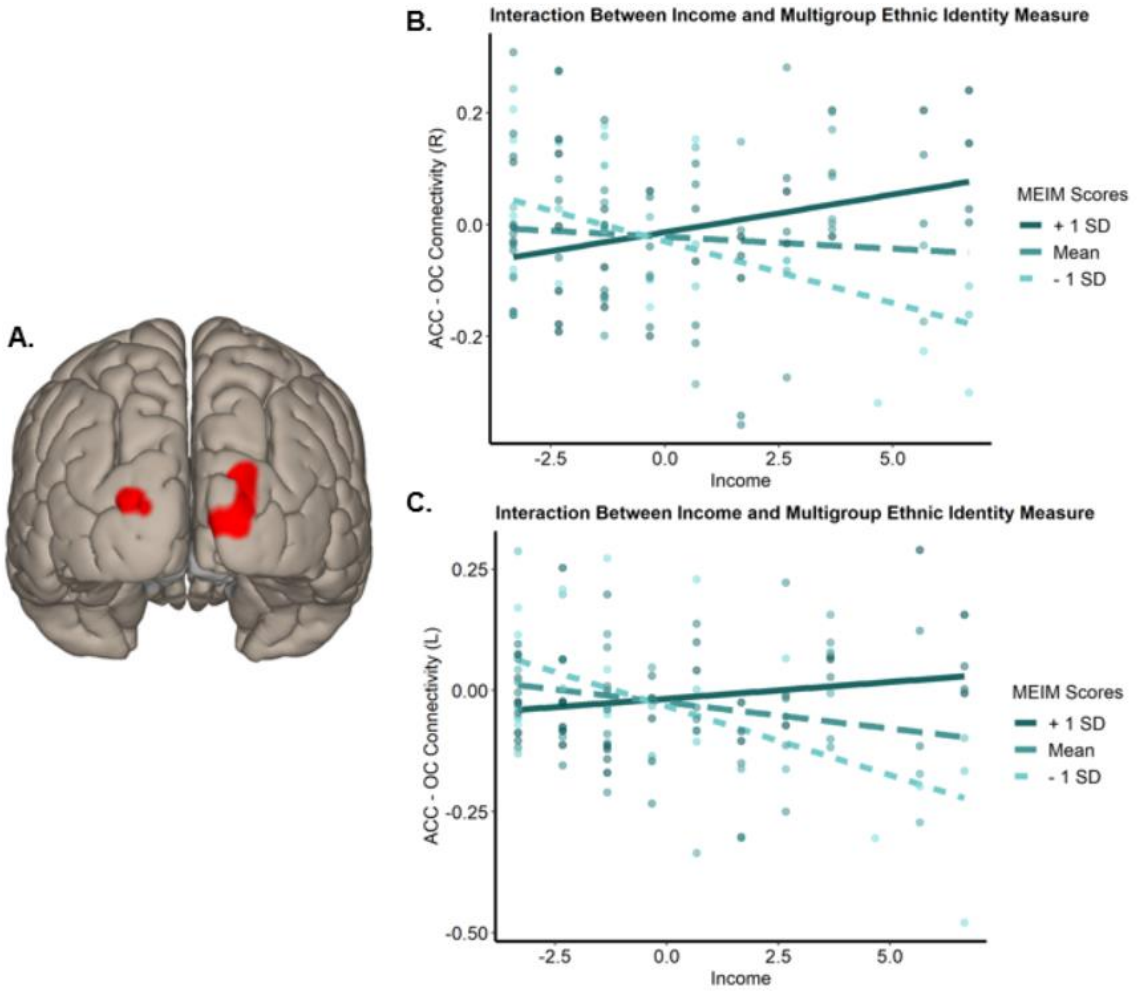

Figure 2.8. **Individuals living in more disadvantaged neighborhoods with stronger racial-ethnic identity showed greater connectivity (Fischer's *z*-scores) between the anterior cingulate cortex (ACC) and (B) right (MNI coordinates *x*: 16, *y*: -100, *z*: 02; cluster size *k* = 88; *p*FDR = .013) and (C) left occipital cortices (MNI coordinates *x*: -20, *y*: -94, *z*: 08; cluster size *k* = 170; *p*FDR < .001)**

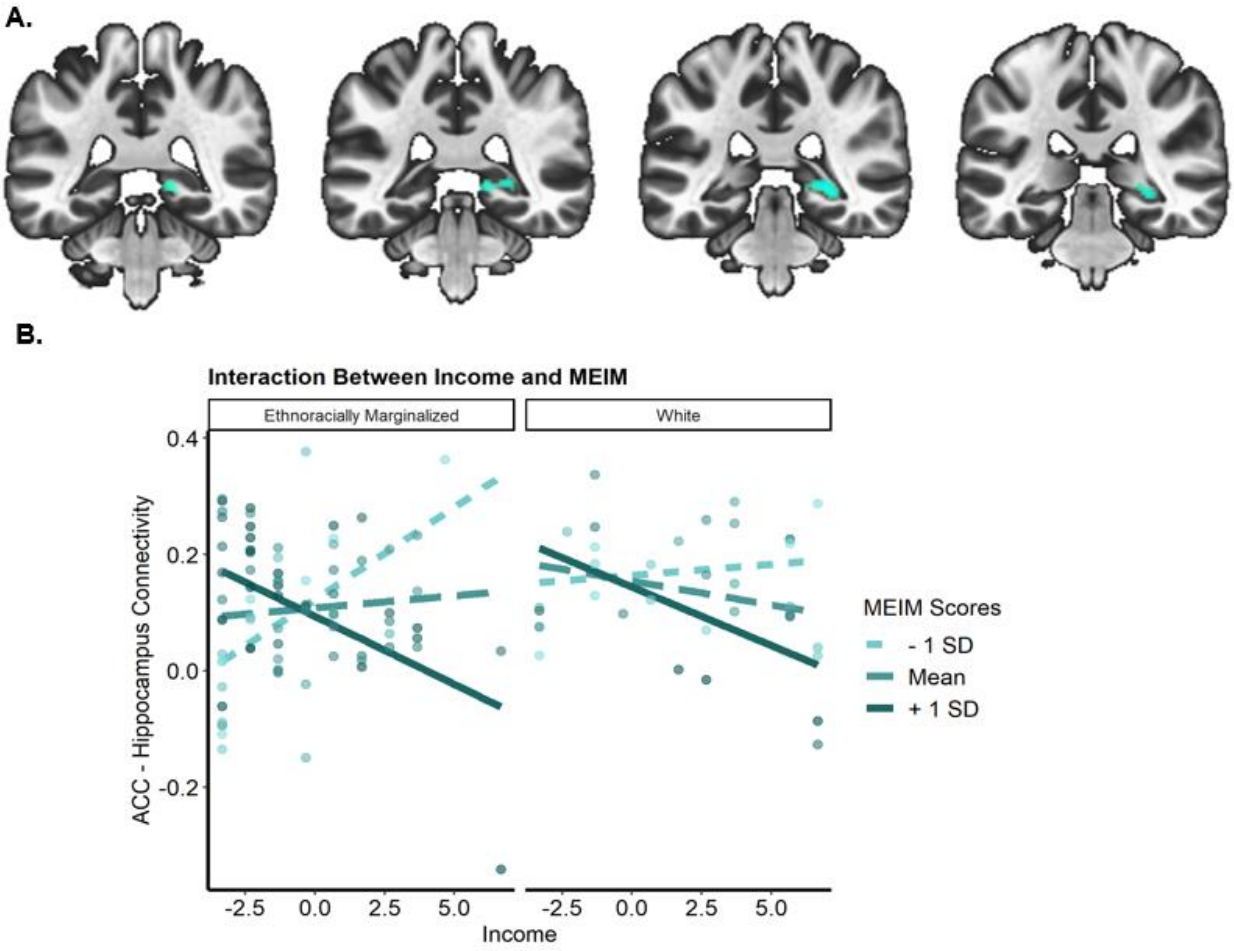

Figure 2.9. **Individuals with stronger racial-ethnic identity who reported higher annual household income displayed less connectivity (Fischer's *z*-scores) between the ACC and hippocampus (MNI coordinates: *x*: 26, *y*: -36, *z*: 00; cluster size *k* = 76; *p*FDR = .018).** Post-hoc tests revealed this pattern was more robust in ethnoracially minoritized participants (p = .025).

Analysis III: ACC functional connectivity values and future PTSD symptoms

Bivariate relationships between ACC functional connectivity values (derived from the significant analyses described above) and CAPS-5 scores are depicted in Figure 2.10. None of the ACC connectivity values predicted future PTSD symptoms.

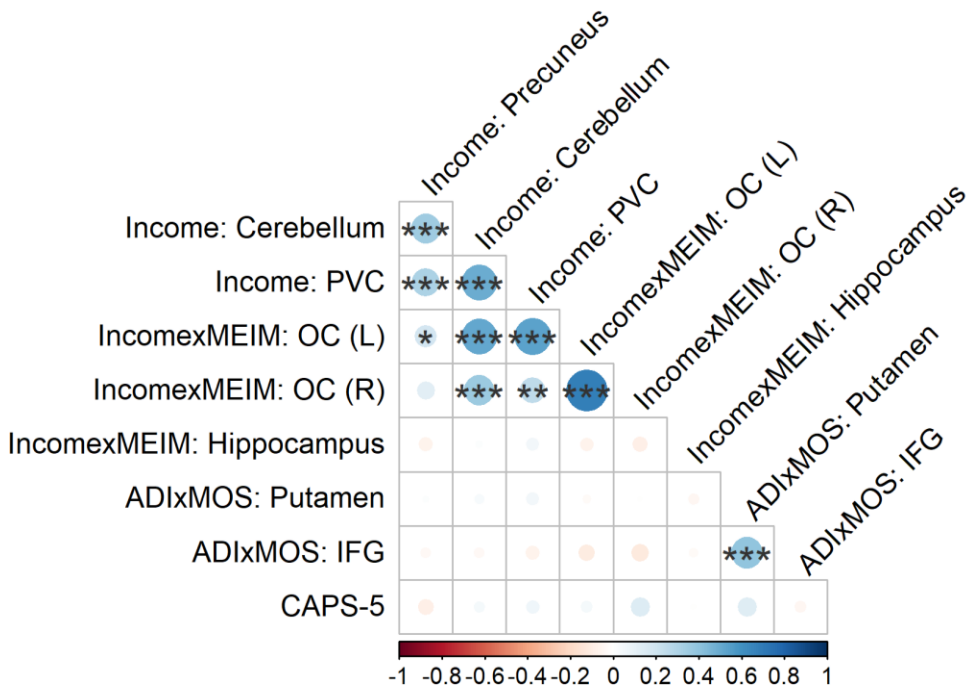

Figure 2.10. **There were no significant associations between ACC connectivity values (Fischer's *z*-scores) and six-month PTSD symptoms (CAPS-5 total symptom severity scores).** *Abbreviations:* **ACC:** anterior cingulate cortex; **CAPS-5:** clinician-administered PTSD Scale for DSM-5; **IFG:** Inferior frontal gyrus; **PVC:** primary visual cortex; **OC:** occipital cortex. ******* $p < .001$, ****** $p < .01$, ***** $p < .05$.

Discussion

Using a well-characterized sample of over a hundred participants who recently experienced a traumatic injury, we found significant effects of socioeconomic circumstances on functional connectivity of the ACC, a proposed neural correlate of resilience. There was no significant relationship between neighborhood disadvantage and ACC connectivity after adjusting for income, gender, and age. Higher annual household income was related to less connectivity between the ACC and visual processing regions, including the precuneus, visuomotor region of the cerebellum, and primary visual cortex. The ACC is implicated in resilience (Roeckner et al., 2021) and we found that socioeconomic variables interacted with resilience factors on ACC connectivity.

For individuals living in more disadvantaged neighborhoods, higher levels of general social support were associated with greater connectivity between the ACC and putamen as well as the inferior frontal gyrus. Given the ethnoracial inequities in socioeconomic circumstances (Phelan & Link, 2015; Williams & Collins, 1995), we explored whether racial-ethnic identity would also act as a buffering factor. Although MEIM did not buffer the effects of neighborhood disadvantage, there was a significant Income X MEIM interaction. Individuals reporting higher income with stronger racial-ethnic identity had greater connectivity between the ACC and occipital cortices and less ACC – hippocampus and connectivity. Though the pattern of the interaction for ACC – hippocampus was similar between White and minoritized participants, the trend was more robust for members of ethnoracially marginalized groups. Studies have suggested altered ACC connectivity represents neurobiological vulnerability and is associated with

non-remitting PTSD symptoms (Roeckner et al., 2021; Zweerings et al., 2018); however, there were no significant associations between ACC connectivity values from significant clusters and six-month PTSD symptoms.

Preclinical and human studies report the ACC has a critical role in visual processing. The ACC influences activation in sensory regions and alterations to ACC connectivity can reflect heightened attention to external cues (Crottaz-Herbette & Menon, 2006; Zimmer et al., 2010). In general, it is adaptive - rather than pathological - to attend to visually novel and/or meaningful stimuli, especially when potential threats are present (Shechner & Bar-Haim, 2016). Differences in connectivity between the ACC and visual regions at rest may reflect environmentally driven adaptations. For example, urban scenes exhibit rich visual content and preferentially recruit both visual regions and the ACC (Grassini et al., 2019).

Participants in the current study recently experienced a traumatic injury, and the psychological effects of acute trauma also influence ACC function (Roeckner et al., 2021; Zweerings et al., 2018). Greater connectivity between the ACC and visual processing regions is related to hypervigilance symptoms (Brooks et al., 2012; Bryant et al., 2005; Stevens et al., 2011). Still, lower SEP and other contextual factors appear to heighten threat-vigilance (Hostinar et al., 2017; Phan et al., 2020). Phan and colleagues (2020) demonstrated exposure to community violence, which may be captured indirectly in ADI rankings (Mears & Bhati, 2006), was related to greater threat-related vigilance. Although we did not observe a significant relationship between connectivity values and six-month PTSD symptoms, lower income did predict CAPS-5 scores. We theorize that greater pre-trauma connectivity between the ACC and visual regions may represent a

neural predisposition to hypervigilance symptoms, helping explain why individuals in lower socioeconomic positions are more likely to develop PTSD (Schumm et al., 2006; Webb et al., *under review*). However, the majority of participants had sub-threshold PTSD symptoms at baseline and six-months which made it challenging to test any mechanisms contributing to PTSD development. Future directions include replicating this analysis in a sample of participants with chronic PTSD and probing symptom clusters, specifically hypervigilance.

Of the existing body of work characterizing the neural underpinnings of resilience factors, studies on social support are the most prominent (Bhanji & Delgado, 2014; Eisenberger, 2013; Inagaki, 2018). Social support promotes positive health outcomes by reducing neurobiological stress-related mechanisms and activating reward circuitry (Inagaki, 2018; Taylor, 2011). The neural correlates of giving and receiving social support overlap substantially with the neural circuits of reward (which includes the ACC; Bhanji & Delgado, 2014; Eisenberger, 2013; Hyde et al., 2011; Inagaki, 2018). The striatum in particular, comprised of the caudate nucleus, nucleus accumbens, and putamen, is implicated in social behavior and reward-learning across different species (Báez-Mendoza & Schultz, 2013; Bhanji & Delgado, 2014). Striatal activation is one mechanism by which general social support is posited to influence affect and subsequent mental health (Bhanji & Delgado, 2014).

Here, we provided additional evidence of the relationship between the striatum and social support, showing that social support is associated with greater connectivity between the ACC and striatum for individuals living in more disadvantaged neighborhoods. Various studies have examined the buffering effects of social support

on health, with some explicitly probing associations and interactions with socioeconomic circumstances (e.g., John-Henderson et al., 2015; Wight et al., 2006). For example, John-Henderson and colleagues (2015) demonstrated social support reduced immune system reactivity in adults reporting low SEP in childhood. Social support is associated with reduced internalizing and externalizing symptoms; with effects strongest for individuals living in more disadvantaged neighborhoods (Wight et al., 2006).

Social support also moderated the effects of neighborhood disadvantage on the IFG. The IFG is critical for executing reappraisal strategies, highlighting a role in "affective inhibition" (Hampshire et al., 2010). IFG activity is also modulated by reward sensitivity, underlying reward-related behaviors (Fuentes-Claramonte et al., 2016). For individuals living in more disadvantaged neighborhoods, social support was related to greater connectivity between the ACC and IFG, suggesting a neural predisposition to more effective deployment of emotion regulation strategies.

Notably, the connectivity patterns associated with ADI x MOS and those associated with Income x MEIM did not overlap. Whereas ADI x MOS was related to altered connectivity between the ACC and reward-related regions (e.g., striatum), the interaction between Income x MEIM was tied to regions recruited during threat (e.g., occipital cortex and hippocampus). To our knowledge, no previous studies have examined the effect of racial-ethnic identity on the brain, therefore our interpretations are motivated by non-neuroimaging work and are speculative in nature. Individuals in higher SEPs with stronger racial-ethnic identity had greater connectivity between the ACC and occipital cortices.

One interpretation draws from the multidimensional model of racial identity (Sellers et al., 1998). In our sample (and the general United States population), White individuals reported significantly higher income than individuals from ethnoracially minoritized groups. Regarding employment, marginalized groups are under-represented in middle and senior roles, causing individuals to be a minority (statistically speaking) with fewer peers that have shared characteristics (e.g., social, cultural, economic and/or religious; Bloch et al., 2021; Hudson et al., 2012). The multidimensional model of racial identity proposes that racial salience (Sellers et al., 1998), a construct intertwined with racial-ethnic identity, is context-dependent. Applying this definition, racial salience may be high for individuals in a workplace where they are a minority (Sellers et al., 1998). High levels of racial salience are coupled with more experiences of discrimination (Sanchez & Awad, 2016), and greater exposure to discrimination recruits threat-related neural systems (Berger & Sarnyai, 2015; Clark et al., 2018; Fani et al., 2021; Webb et al., *in press*). This is further supported by the significant links between visual vigilance and activation of visual regions and ACC (Kastner-Dorn et al., 2018; Maren et al., 2013).

Although speculative, we hypothesize that greater ACC – occipital cortex connectivity in individuals reporting higher income with greater racial-ethnic identity, reflects a propensity to utilize “vigilant coping” and experience higher levels of racial salience (Himmelstein et al., 2015). Still, our sample recently experienced a traumatic injury and although we controlled for psychological symptoms related to the index trauma, we cannot account for all aspects of the injury. The OCC responds to more than threat-related stimuli; in this context, greater connectivity may reflect rich visual

environments, rather than vigilance (Grassini et al., 2019). Indeed, globally integrated resting-state networks, including sensory systems is widely considered a marker of efficient neural processing. Therefore, we encourage future work to explore neurobiological correlates of racial salience and MEIM in non-trauma exposed samples.

Greater racial-ethnic identity was associated with less connectivity between the ACC and hippocampus in those reporting higher income. The ACC modulates sub-cortical representation of threat-related stimuli and coordinates behavioral responses to threat, whereas the hippocampus underlies consolidation of fear memories (Battaglia et al., 2011; Bissière et al., 2008; Descalzi et al., 2012; Etkin et al., 2011; Maren et al., 2013; Maren & Holt, 2000). Altered connectivity between the ACC and hippocampus is associated with non-remitting PTSD symptoms, with the majority of work indicating lower engagement of threat-related regions signifies resilience (see review Roeckner et al., 2021). Less connectivity between these regions even in in the acute aftermath of a traumatic event may represent the additional protective benefit of strong racial-ethnic identity, on top of the advantage of higher SEP.

To our knowledge, this is the first study to characterize relationships between socioeconomic circumstances and resilience factors on resting-state connectivity patterns in trauma exposed individuals. Our study was well-designed to evaluate risk and resilience predictors of PTSD, and we included multi-level (e.g., individual income and neighborhood disadvantage) variables in a risk-resilience model. By focusing on one region of interest and two resilience factors, we were able to compare the distinct connectivity patterns associated with social support and racial-ethnic identity. Still, the scope of the ACC, and the absence of task data limits the interpretation of our findings.

Resting-state connectivity analyses, especially with large heterogenous regions (like the ACC) do not provide precise explanations into brain-behavior relationships. Another limitation of the current study was the inability to tease apart differences between ethnoracial groups (i.e., subgroup analyses), instead assigning individuals as either White or ethnoracially minoritized. Future work should continue to explore how different resilience factors' function, with particular attention to including participants from ethnoracially minoritized groups. The majority of neuroscience research is conducted with White participants (Dotson & Duarte, 2020); but there is little evidence to suggest that resilience and risk factors are identical across ethnoracial groups (Brondolo, 2015; Castro & Murray, 2010; Clauss-Ehlers, 2008; Lee, 2005; Yosso, 2005). In fact, scholars have called for more culturally relevant studies of resilience which should consider the differences in the demands, resources, and experiences of various ethnoracial groups (Brondolo, 2015; Castro & Murray, 2010; Clauss-Ehlers, 2008; Lee, 2005).

Conclusion

By studying the nexus between the brain, individual experiences, and the environment, we can better understand how societal structures, alongside an individual's underlying biology and characteristics, impact health status. Here, we added to the growing body of literature demonstrating that socioeconomic circumstances have a significant effect on brain functioning (e.g., Betancourt et al., 2016; Brito & Noble, 2014; Farah, 2017; Gard et al., 2021; Hanson et al., 2011; Hunt et al., 2020; Lotze et al., 2020; Palacios-Barrios et al., 2021; Rakesh et al., 2021; Webb et al., 2021; Whittle et al., 2017). Socioeconomic circumstances are a risk factor for poor post-trauma outcomes, and we replicated that income significantly predicts six-month PTSD

symptoms (Herrera-Escobar et al., 2019; Webb et al., *under review*). The racialization of individual socioeconomic position and neighborhood disadvantage represents intersecting forms of oppression (Williams et al., 2016). As such, neuroscientists examining the effects of socioeconomic circumstances on neurobiology have an obligation to test associations between ethnoracial identity and socioeconomic circumstances. At a neurobiological level, we provided evidence that both social support and racial-ethnic identity buffer against the deleterious effects of neighborhood disadvantage and income, respectively. Ultimately, these results provide additional evidence that risk-resilience models, including sociopoltical factors, are essential to research examining post-trauma outcomes.

Project III Statement of Intent.

Though not typically part of a neuroscience doctoral dissertation, I have opted to reflect on the empirical work connecting neighborhood factors and the brain through a neuroethics commentary. In doing so, I encouraged myself and others to consider the cultural, ethical, societal, and legal implications of conducting studies on the neurobiological impact of neighborhoods and how these findings could be used to inform policies and future directions of neuroscience research.

Radicalizing Studies on Neurobiology of Socioeconomic Circumstances: A Call for Social Justice-Oriented Neuroscience

"Radical simply means 'grasping things at the root'."

- Angela Davis.

Despite national economic growth in the United States, the number of socioeconomically disadvantaged neighborhoods has continued to rise over the past 40 years (Benzow & Fikri, 2020). In 2018, an estimated 24 million people lived in high-poverty neighborhoods, defined by an average poverty rate of 30 percent or higher (Benzow & Fikri, 2020). Though the lasting economic impact of the coronavirus pandemic is still uncertain, initial evidence suggests deepening economic hardships, particularly for low-income adults and Black and Latinx communities (Parker et al., 2020). Individual socioeconomic factors (e.g., education and income) have received considerable attention as robust predictors of mental and physical health (Adler et al., 1994; Marmot & Wilkinson, 2005; Prus, 2007; Reiss, 2013; Smith, 2004). However, throughout the lifespan, where a person lives and contextual factors beyond the individual (e.g., neighborhood-level socioeconomic disadvantage) also impacts their health (Clarke et al., 2014; Diez Roux, 2001; Diez Roux & Mair, 2010; Kind & Buckingham, 2018; Minh et al., 2017; Riley, 2018; Sewell, 2015, 2016). Widening

socioeconomic inequities[2], at both the neighborhood and individual level, therefore signal a public health crisis.

Mental health research has centered on how adversity, including lower individual socioeconomic position (SEP), becomes biologically embedded (e.g., Keinan et al., 2012; Mcewen, 2004; McEwen, 2012; McLaughlin & Sheridan, 2016; Turner & Lloyd, 1995). With evidence from physiology, genomics, and neuroimaging, the evolution of our knowledge regarding the impact of socioeconomic circumstances on the brain, body, and mental health, has been remarkable (reviewed in Brito & Noble, 2014; Farah, 2017, 2018; Gianaros & Hackman, 2013; Hackman & Farah, 2009; Johnson et al., 2016). Despite considerable empirical evidence demonstrating the biological burden of these factors, the development of evidence-based interventions to address these types of adversity has been laborious and with few victories. We propose this impasse is because most mental health research, particularly studies with neuroimaging, do not systematically include these adversities in study designs or situate findings within existing social inequities (Camara Phyllis Jones et al., 2009; Gee & Ford, 2011; Harnett, 2020; Harnett & Ressler, 2021; Payne-Sturges et al., 2021; Riley, 2018; Sewell, 2015; Yearby, 2020). Historically, mental health research braved the topic of social inequities (e.g., social class) but, in the 1980's, a shift towards biological perspectives meant the focus was relinquished (Muntaner et al., 2000). More explicitly, while research on physical health has increasingly built upon studying social inequities and disease (Krieger, 1994, 2011), "mainstream" mental health research has embraced

[2] **Inequity** is defined here as differences (e.g., between ethnoracial groups, between socioeconomic positions, etc.,) which are unjust, unfair, and avoidable. The differences referred to in this article are injustices caused by structural racism.

predominantly biological mechanisms of disease, leaving little room for reflection that the context in which individuals operate matters (reviewed in Muntaner et al., 2000 and Krieger 1994).

In a similar vein, the association between mental health and neighborhood factors (e.g., neighborhood socioeconomic disadvantage) has received even less attention than associations with individual-level variables (e.g., income or education Farah, 2017, 2018). This may reflect study design limitations; there is simplicity in collecting individual-level measures directly from the participant, and also the benefit of evading the expenses associated with larger sample sizes, which are often required to observe significant effects of neighborhood factors. Another explanation is that neuroscience research has been implicitly biased towards using a “Freedom” model of health, which suggests people are solely responsible for their health and related behaviors (i.e., individual- oriented theories of disease causation Dougherty, 1993; Krieger, 2001, 2011, 2011; Muntaner et al., 2000). Though many issues arise when defaulting to the Freedom model, perhaps most insidious is that it complements the “deserving poor” argument or “boot-strap” ideology, which alleges people are in specific socioeconomic positions because of individual differences in ambition or talent. This stance is not reflected in data; in fact, upward mobility (rising to a higher socioeconomic position) rates in the United States have continued to decline over the past 10 years, and both race and place (i.e., regional differences) remain the strongest predictors of mobility (Connor & Storper, 2020). The “Freedom” model - and those akin to it - disregards the longstanding inequities in opportunity in the United States and, when

applied (consciously or not) to neuroscience research, exonerates the oppressive structures which maintain the inequities.

Here, we focus on studies of neighborhood socioeconomic disadvantage and neurobiology because research in this area inherently emphasizes *place* and *context* rather than the *individual*. This work, albeit limited, marks a fervent shift in human neuroscience literature towards recognizing that the sociopolitical context[3] affects how individuals navigate within social groups, interpret stimuli and events, even brain structure and function. This shift also signifies a scientific acknowledgment that people do not live in isolation and that societal inequities exist. Few scientists (if any) would endorse the contrary, but by excluding these variables and disregarding societal influences, the resulting scientific products lack this context. By including socioeconomic variables (individual and neighborhood-level) in human neuroscience experiments, researchers acknowledge that some of the variability in individual differences - whether in biological functioning, cognitive and affective task performance, or clinical symptoms – is attributable to the sociopolitical stratification in society (Chen et al., 2006; Chen & Miller, 2013; Gianaros & Hackman, 2013; Harnett, 2020). Although we chose to focus on neighborhood disadvantage, the issues presented in this article are shared between neurobiological studies on individual socioeconomic circumstances (e.g., education, income).

Studies on the effects of neighborhood disadvantage on neurobiology are at the forefront and intersection of public health, neuroscience, and sociology, and in this

[3] The authors use **sociopolitical**, rather than socioenvironmental, to emphasize the role of political systems in defining social hierarchies and creating societal stratification.

paper, we leverage knowledge across these disciplines. After briefly reviewing evidence that neighborhood disadvantage is associated with poor mental health outcomes, with relationships to both brain function and structure, we argue that neuroscience work would be strengthened by positioning research questions and findings within sociological and historical perspectives. We call for future studies to name structural racism, define neighborhood disadvantage as an institutionalized form of racial inequity, and interpret how racism is captured in methods and manifests in results (Riley, 2018; Sewell, 2015, 2016). Finally, we describe areas and steps for improvement, including acknowledging historical and current inequities, reporting meaningful disaggregated data, and funding community-based participatory research. These recommendations are based in the belief that neuroscience could more critically address mental health disparities if a radical framework, which considers the root causes of inequities, was applied.

Neural Correlates of Neighborhood Socioeconomic Disadvantage

Neighborhood socioeconomic disadvantage measures (e.g., poverty rate, composite measures such as area deprivation index, etc; Coulton et al., 2002; Kind & Buckingham, 2018; Nicotera, 2007; Riley, 2018; Singh, 2003; Tomlinson et al., 2020), established with a geographical ID and through a process of geo-coding (Fan et al., 2021), can predict myriad mental health symptoms, even above individual measures of socioeconomic circumstances. Greater neighborhood disadvantage is associated with higher stress levels (Aneshensel, 2009; Barrington et al., 2014; Chattarji et al., 2015; Hackman et al., 2012; Snedker & Herting, 2016; Steptoe & Feldman, 2001) and symptoms of depression (Blair et al., 2014), anxiety (Casciano & Massey, 2012; Vine et

al., 2012), and PTSD (Douglas et al., 2021; Gapen et al., 2011; Hall Brown & Mellman, 2014). Biological correlates of neighborhood disadvantage include altered cortisol reactivity (with directions of relationship depending on task and collection method; Barrington et al., 2014; Finegood et al., 2017; Karb et al., 2012; Zilioli et al., 2017), accelerated aging (e.g., Lawrence et al., 2020; Lei et al., 2018, 2019; Olden et al., 2015), and immune system dysregulation (e.g., Kepper et al., 2016; Neergheen et al., 2019; Roberts et al., 2020). In nearly all proposed mechanistic models, neighborhood disadvantage is conceptualized as chronic stress[4] and therefore hypothesized to influence mental health via stress-responding pathways (e.g., immune system dysregulation, persistent hypothalamic-pituitary-adrenal axis activation Farah, 2017; Gianaros & Hackman, 2013; Hackman & Farah, 2009; McEwen, 2012b; McEwen & Gianaros, 2010). Despite differences between various neighborhood factors and exposures, neighborhood socioeconomic disadvantage is used as an imprecise, yet practical, proxy of neighborhood chronic stress.

The impact of neighborhood disadvantage on neurobiology continues to grow as an exciting line of research, with the majority of work conducted in adolescents. A

[4] In this context, the term **chronic stress** is used broadly to encompass exposures to factors (e.g., environmental toxins, community violence, police brutality, etc.) and unstable access to necessary resources (e.g., education, food, transportation, etc.; Kim et al., 2018; McEwen & Gianaros, 2010). For example, residing in socioeconomically advantaged neighborhoods generally corresponds with greater access to favorable built and natural infrastructure, such as schools, banks, and recreational parks, and less exposure to harmful environmental toxins (e.g., proximity to factories, lead piping; Aneshensel, 2009; Diez Roux, 2001; Diez Roux & Mair, 2010). Quality of schools, access to transportation, and food availability, are among the many daily activities and resources shaped by geographical designations (Diez Roux & Mair, 2010; Durfey et al., 2019; Farah, 2017; Kind & Buckingham, 2018).

comprehensive review of this literature is outside the scope of this commentary and has been recently conducted by Rakesh and colleagues (2021). A review on associations between socioeconomic circumstances and the *adult* brain has not been conducted to our knowledge, likely because this research is scarce. Studies on gene-environment interactions suggest that even through adulthood, the environmental context, including neighborhood disadvantage, continues to alter our biology (Lawrence et al., 2020). Thus, we argue that studies on neighborhood disadvantage in adulthood are just as valuable for our understanding of adult mental health outcomes and transgenerational effects and call for future work to consider asking these questions in adult samples. Still, we highlight key findings (primarily from adolescent samples given the limited adult work) suggesting that neighborhood disadvantage is associated with widespread alterations in brain structure and function.

Perhaps most well-documented, in both adults and adolescents, is the significant effects of neighborhood disadvantage on prefrontal thickness and hippocampal volumes (Brito & Noble, 2014; Hunt et al., 2020; Taylor et al., 2020; Vargas et al., 2020; Webb et al., 2021; Whittle et al., 2017; Wrigglesworth et al., 2019). Several studies have also found neighborhood disadvantage is associated with lower total surface area and subcortical volume (Hackman et al., 2021; Hunt et al., 2020). Additional evidence indicating widespread structural effects, arises from diffusion tensor imaging studies: greater neighborhood disadvantage is associated with lesser integrity in various tracts (Bell et al., 2021; Gianaros et al., 2013; Webb et al, *Project 1*). In identifying the neurobiological mechanisms linking neighborhood disadvantage to mental health, these structural changes are compelling targets; thinner prefrontal cortex and smaller

hippocampus are all risk factors for PTSD, depression, and anxiety (Roeckner et al., 2021).

Even after accounting for individual SEP, neighborhood disadvantage has been linked to delayed structural and functional neurodevelopmental trajectories (e.g., Gard et al., 2021; Rakesh et al., 2021; Ramphal et al., 2020; Tooley et al., 2020). Rakesh and colleagues (2021) convincingly teased apart the distinct and shared effects of neighborhood disadvantage and household SEP, demonstrating interactive effects between the two different measures on resting-state networks and further highlighting that individual SEP does not fully account for neighborhood effects. Task-based neuroimaging indicates neighborhood disadvantage helps explains individual differences in affective and cognitive domains (Weis et al., *under review*). For example, Tomlinson and colleagues demonstrated neighborhood disadvantage was related to neural and behavioral correlates of response inhibition (i.e., cognitive domain). In adolescents, neighborhood disadvantage was associated with greater amygdala reactivity to ambiguous neutral faces (Gard et al., 2018) and, in adults, neighborhood disadvantage was related to diminished amygdala threat-related activity (Harnett et al., 2017; i.e., affective domains).

These findings, albeit limited, point to environmentally driven adaptations, suggesting living in a disadvantaged neighborhood elicits adaptive neural processes which may place abnormal demands on cognitive resources (Harnett et al., 2017, 2020; Webb et al., 2021). These resources may already be jeopardized by the structural changes evoked by living in a chronically stressful environment. Together, modifications

to brain structure and function may create susceptibility to psychopathology, connecting neighborhood disadvantage to poor mental health outcomes.

Although more empirical work is needed, this theory helps explain why individuals residing in more disadvantaged neighborhoods report more symptoms of depression, anxiety, and PTSD (Blair et al., 2014; Casciano & Massey, 2012; Douglas et al., 2021; Gapen et al., 2011; Hall Brown & Mellman, 2014; Vine et al., 2012). Although the mechanisms by which neighborhood disadvantage impacts brain structure and function may be fundamentally the same across people, not everyone is exposed to this factor at the same rates. Individuals from racially and ethnically minoritized groups are disproportionally exposed to neighborhood disadvantage. Ethnoracial inequities in mental health are multifaceted and may stem from differential access and utilization of health care services, individual-level SEP, and differences in exposure to stressors (e.g., experiences of racial discrimination, differential exposure to types of trauma, etc.; Bird et al., 2021; Carter et al., 2017; Gee & Ford, 2011; Harnett, 2020; Harnett & Ressler, 2021; Williams, 2018; Williams & Cooper, 2019). Although neighborhood disadvantage alone may not fully explain ethnoracial mental health inequities, it is certainly a contributing factor.

In all the aforementioned work, researchers were faced with decisions concerning the intersections between race, ethnicity, SEP, and neighborhood disadvantage. Despite strong theoretical support that ethnoracial inequities and socioeconomic inequities are related but not equivalent (Williams, 1999), the ability to statistically tease apart these effects - or even interpret results within this context - is challenging. Others (e.g., Nuru-Jeter et al., 2018) have provided recommendations on

how to statistically approach measures of ethnoracial and socioeconomic inequities. At best it is methodologically negligent and at worse, ethically harmful to ignore the overlapping patterns of ethnoracial and socioeconomic disparities in studies on neurobiology and related factors[5] (Nuru-Jeter et al., 2018; Williams, 2018; Williams & Mohammed, 2013).

Naming Structural Racism as the Root Cause

Socioeconomic inequities influence health independent of race and ethnicity, however, both individual and neighborhood socioeconomic indicators are ethnoracialized (i.e., stratified by race and ethnicity; Nuru-Jeter et al., 2018; Williams, 1999; Williams et al., 2019; Williams & Mohammed, 2013). In this way, the socioeconomic inequities discussed in studies on neighborhood disadvantage and neurobiology are undergirding and intersecting with other forms of oppression, particularly structural racism[6] (Sewell, 2015). In fact, all of the canonically defined social determinants of health (e.g., economic stability, education access and quality, etc.) take form and hold power through structural racism (Harnett, 2020; Nuru-Jeter et al., 2018; Riley, 2018; Sewell, 2015, 2016; Yearby, 2020). Certain exposures, such as neighborhood disadvantage, exist as a risk factor because of structural racism (Riley, 2017). Empirical evidence underscores this: Black Americans in middle SEPs are still

[5] The majority of work on socioeconomic circumstances and the brain has been based in the United States and therefore this paper discusses this research within the American sociopolitical context. However, the authors encourage researchers outside of the United States to consider how global, national, and regional structures of oppression, including racism, are impacting their neuroscience studies.

[6] **Structural racism** is defined here as "macro-level systems, social forces, institutions, ideologies, and processes that interact with one another to generate and reinforce inequalities among racial and ethnic groups" (Gee and Ford, 2011)

more likely to live in disadvantaged neighborhoods compared to White Americans in *lower* SEPs (Turner and Greene, 2021).

Recent years have seen resounding calls in public health for structural racism to be named as the root cause of ethnoracial health disparities and related racialized socioeconomic inequities (Bailey et al., 2017; Ford & Airhihenbuwa, 2010; Gee & Ford, 2011; Hardeman et al., 2018; Harnett & Ressler, 2021; Yearby, 2020; Yosso, 2005). Yet human neuroscience research has been reluctant to confront structural racism; rarely even naming the oppressive structure in introductions or discussions. To resound a question raised by Sewell (2016): "why not then spell out the connections between health disparities and institutional (in)actions rooted in racism?" The addition of historical and sociological perspectives and the explicit naming of structural racism does not hinder or diminish neuroscience, rather these perspectives complement, advance, and aptly challenge the current state of the research.

Situating Studies Within Historical and Contemporary Contexts

Differential exposure to neighborhood disadvantage is maintained by historical and current ethnoracial residential segregation. Historic redlining is perhaps the most well-known practice contributing to residential segregation (McClure et al., 2019). Discriminatory law [7] from the 1930's until 1968 (when redline mapping was made illegal), allowed the government-led Home Owners' Loan Corporation to create maps for lending institutions (Connolly et al., 2018; Hillier, 2003; Massey & Denton, 1993; McClure et al., 2019; Sewell, 2015). These maps were used to prevent people of color

[7] **Law** refers to the mechanisms of legal systems, including the political processes, policies, and legal practices such as enforcement (see also Yearby 2020).

from residing in specific neighborhoods by limiting bank credit and altering real-estate practices (Massey & Denton, 1993). The resulting shift in the entire homebuying process ultimately forced people to buy houses in less “desirable” (redlined) neighborhoods (Massey & Denton, 1993). In addition, these policies and practices resulted in expansive divestment in redlined neighborhoods and disproportionate investment in predominately White neighborhoods. Redlining may have historic roots, but the legacy in redlined neighborhoods manifests in the lasting neighborhood disadvantage and ultimately the residents’ mental and physical health (Massey & Denton, 1993; Park & Quercia, 2020; Sewell, 2015; Williams et al., 2019).

Current housing law and practices are also culpable, people of color are still disproportionately denied fair mortgage loans (Hanifa, 2021) and Black and Latinx communities continue to be under-valued and under-funded (Park & Quercia, 2020). Withholding certain types of investment (e.g., under-funding of schools) while also misallocating funds to non-community approved budgets (e.g., policing) maintains neighborhood socioeconomic disadvantage. Historic and current racist policies and practices force(d) people of color, particularly Black Americans, to disproportionally reside in neighborhood’s experiencing socioeconomic disadvantage. Thus, neighborhood advantage is a protective factor that can be - and has been - bestowed upon White people by law. Even the terms “neighborhood advantage” or “neighborhood disadvantage” fundamentally align with language used (particularly in theories of Black feminism and intersectionality) to discuss structural racism; White people unfairly benefit from these structural advantages and all other people are harmed.

Recommendations for Radicalizing Human Neuroscience

In our work as neuroscientists, we must recognize that people live within environmental contexts shaped by sociopolitical stratification. When we study neighborhood disadvantage, we are studying an exposure that is relevant to mental health because of its connection to structural racism (Riley, 2018; Sewell, 2015). In essence, this commentary is a call for the radicalization of human neuroscience work – a necessary paradigm shift that grasps at the roots of the issue rather than dodging them. By remaining silent (i.e., not acknowledging structural racism) in our introductions and discussions, we fail to hold the institutions protecting structural racism responsible. When we name structural racism, we direct attention to the laws, processes, and practices which produce and maintain health disparities (Sewell, 2015, 2016). This offers an incredible opportunity to connect research findings directly to policies (e.g., non-discriminatory housing laws), instead of relying on blanket statements about ending socioeconomic inequities.

In general, few studies examining neighborhood disadvantage have methodologically confronted ethnoracial *and* socioeconomic inequities (c.f., Douglas et al., 2021; Harnett et al., 2019; Taylor et al., 2020 - in supplemental analyses) – though many call for future work with larger samples to explore these intersections (e.g., Hunt et al., 2020; Rakesh et al., 2021; Sripada et al., 2021; Webb et al., 2021). Providing descriptive information on between group differences on socioeconomic measures would contextualize the sample and position inequities at the forefront. Even outside of the work on neighborhood disadvantage, reporting of complete sociodemographic variables is not commonplace. Race and ethnicity are still not frequently reported, despite being “required” by many journals. It seems obvious, but ethnoracial differences

in study measures can only be observed and interpreted if the data are presented. Therefore, we echo calls to report demographic data that is meaningfully and appropriately disaggregated (i.e., based on historical and structural inequities; e.g., Kauh et al., 2021).

Neuroscience research on neighborhood factors has largely focused on risk modelling, evaluating variables believed to worsen mental health. Institutionalized forms of racial inequities, including neighborhood disadvantage and community violence, are risk factors dominating the emerging field (e.g., Borg et al., 2021; Butler et al., 2018; Gellci et al., 2019; Rakesh et al., 2021; Reda et al., 2021; Saxbe et al., 2018; Webb et al., 2021; Wrigglesworth et al., 2019). Discussions backed by critical race theory[8] being held in other fields including education, law, and psychology, should inform neuroscience work moving forward (e.g., Gillborn & Ladson-Billings, 2009; Giraldo et al., 2017; Yosso, 2005). A key tenant of critical race theory is that deficit-only perspectives, which minimize the strengths of ethnically and racially minoritized groups/individuals, are harmful (Giraldo et al., 2017; Yosso, 2005). Theoretically, risk-only models are incomplete; and practically, they further stigmatize marginalized populations.

There is ample room for resilience modeling (also known as strength-based approach) in studies on socioeconomic circumstances and neurobiology. In the field of neuroscience, exploring the effects of individual, familial, and community factors that are known to mitigate risk of poor mental health outcomes, such as social

[8] **Critical race theory** is a framework used to analyze the historical and contemporary forms of structural racism. The term was coined by Kimberlé Crenshaw.

support/engagement, neighborhood cohesion, and racial-ethnic identity, is still novel and could be extraordinarily beneficial (e.g., Bracey et al., 2004; Burt et al., 2021; Dassopoulos & Monnat, 2011; Forsyth & Carter, 2012; Gapen et al., 2011; Johns et al., 2012; Karb et al., 2012; Lardier Jr. et al., 2021; Neblett Jr et al., 2012; Neergheen et al., 2019). Studies on potential resilience factors are even more interesting than replication of deleterious effects of neighborhood disadvantage because the findings may reveal possible targets for financial and cultural investment. For example, studies documenting the beneficial effects of greenspace on brain structure and mental health (Besser et al., 2021; Engemann et al., 2019; Mennis et al., 2018; South et al., 2018) underscore funding calls for vacant-lot greening and park development.

The final recommendation is perhaps the most radical. Human neuroscience has relied primarily on “top-down” scientific processes. In this approach, the power (i.e., decision-making, funding, control over dissemination process, etc.) rests entirely with the study team. Although those researched provide data, they are not consulted to ensure the research question(s) or outcomes align with their experiential knowledge or the community’s needs. Even with best intentions, this Western knowledge production pipeline is inequitable because the power is not equally distributed between the researchers and the researched (Minkler & Wallerstein, 2003; Wallerstein & Duran, 2010). Community-based participatory research (CBPR) is a different approach to knowledge production which involves various stakeholders (i.e., community members and academic partners) collaborating throughout the research process (Minkler & Wallerstein, 2003; Wallerstein & Duran, 2010). At its core, CPBR hopes to build health equity by practicing equity (Minkler & Wallerstein, 2003; Wallerstein & Duran, 2010).

Psychology has started to answer the calls for community-driven research and human neuroscience should follow (Arredondo, 2021; Collins et al., 2018; Wallerstein, 2021; Wallerstein & Duran, 2010, 2017). A first step for research teams is for members to reflect on how their own positionality[9] manifest in their work and in interactions with fellow team members and participants (Muhammad et al., 2015). Just as we cannot isolate participants from the sociopolitical environment, we cannot ignore the intrinsic influences of society on research practices or hide behind a façade of self-proclaimed objectivity (Muhammad et al., 2015). CPBR entails community-building (which takes time) as well as sharing wealth and final products (which requires funding and time; Collins et al., 2018; Wallerstein, 2021; Wallerstein & Duran, 2010, 2017). To make progress in neuroscience CPBR, funding agencies like the National Institutes of Health, must be receptive to funding studies that are likely longer and more expensive. These organizations must also value including community members on research teams, even if these members do not have traditional (i.e., Western knowledge production) research training or traditional indicators of research contributions. As researchers, we can advocate for more funding opportunities while also introducing CBPR practices into existing studies (e.g., collaborating with an established community organization during data analysis and dissemination).

Conclusions

[9] **Positionality** refers to a person's sociopolitical identity (e.g., gender identity, sexual identity, race, ethnicity, socioeconomic position, religion, etc.) and lived experiences, all of which shape their position in society. Ultimately, this position influences how a person interacts with and perceives the world.

The call to address health disparities and build health equity must be met with a radical response. As the field of human neuroscience continues to identify biological mechanisms underlying disease, it must cautiously avoid biological reductionism. We encourage all to remain vigilant about discussions of neurobiological effects of sociopolitical variables using only biological terms, and without actually naming oppressive structures (e.g., racism, sexism). In the context of studies on neighborhood disadvantage, defining neighborhood disadvantage as an institutionalized form of racial inequity (Sewell, 2016), is the initial move towards the root. Additional steps include more thorough reporting of demographics which requires comprehensive evaluations of ethnoracial and socioeconomic inequities (i.e., asking participants about race and ethnicity, geocoding addresses to derive neighborhood disadvantage). Ultimately, however, more radical changes such as challenging Western knowledge production, embracing CBPR, and reforming funding agencies' priorities, will fully grasp the roots.

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Appendix A. Code for Project I

Overview

This dissertation was completed Fall 2021 by Kate Webb. It uses data from the Imaging Study of Trauma and Resilience (iSTAR; PI: Dr. Christine Larson). iSTAR recruited participants in the acute aftermath of a traumatic injury and conducted follow-up assessments across a two-year period. The purpose of the study was to evaluate potential predictor of PTSD, including neural biomarkers. The SPSS files with raw data can be found on the iSTAR Box.

Project 1 Introduction, N = 148

In the first project, I explored how different factors influenced global white matter tract integrity using a multivariate multiple regression approach.

In an exploratory aim, the protective effect of commitment to and willingness to explore one's own racial and/or ethnic group was evaluated. To address this aim, we examined each tract separately and tested an ADI x MEIM interaction.

Data Codebook

Brain Measures

FA - Fractional Anisotropy, an index of white matter tract integrity.
UNC - Uncinate Fasciculus Tract
CST - Corticospinal Tract
CAB - Cingulum-Angular Bundles
ARC - Arcuate Fasciculus Tract
CCG - Cingulum-Cingulate Gyrus
ILF - Inferior Longitudinal Fasciculus
SLFT - Superior Longitudinal Fasciculus
FMINOR - Forceps Minor
FMAJOR - Forceps Major

Self-report Measures

PCL-5 - PTSD Checklist for DSM-5; baseline total PTSD symptom severity measured at T1.
ADI - Area Deprivation Index; a measure of a neighborhoods socioeconomic position.
MEIM - Multigroup Ethnic Identity Measure; probes racial-ethnic identity and assesses elements of commitment to and exploration of one's own racial-ethnic group.
Income - Income; measured on a semi-continuous scale where "1" represents annual household income of $0-10,000.
Gender - Gender; dummy-coded where "1" represents women and "0" indicates men.
Ethnoracial_group - Grouping variable to examine racial and ethnic differences; dummy-coded so that "0" represents individuals who identified as part of a marginalized racial and/or ethnic group and "1" represents participants who identified as White.
Age - Years of Age; reported at T1.
CAPS5 - Clinician-Administered PTSD Scale for DSM-5; total PTSD symptom severity

measured at T2.
Note: “c” before a variable name indicates grand mean-centered.

Data Set-up

167 participants had usable DTI data. Of these participants, 9 could not be successfully geo-coded. 10 participants did not have scores on MEIM. A total of 148 participants are included in the dataset.

```
## Read 2wk_Day2 DTI Dataset
Project1<- read.spss("C:/Users/eksears/Desktop/ProjectOne_Dissertation Data_ALLraceethn.sav", to.data.frame=TRUE)

## re-encoding from UTF-8
```

Missing Data

Path reconstruction was poor in a number of tracts. For bilateral tracts, if one hemisphere was successfully reconstructed, then that hemisphere’s FA value was used in place of the average. If neither hemisphere was reconstructed, then data was mean imputed: UNC (n = 6), CAB (n = 16), FMINOR (n = 11), FMAJOR (n = 30).

Correlations of Left and Right Hemispheres

```
correlation_hemi <- c("ATR_L", "ATR_R", "CAB_L", "CAB_R", "CCG_L", "CCG_R", "CST_L", "CST_R", "ILF_L", "ILF_R", "SLFP_L", "SLFP_R", "SLFT_L", "SLFT_R", "UNC_L", "UNC_R")
corrdata_hemi <- Project1[correlation_hemi]

test = cor.mtest(corrdata_hemi, conf.level = 0.95)
plot<-cor(corrdata_hemi, use = "pairwise.complete.obs")
head(round(plot,2))

##       ATR_L ATR_R CAB_L CAB_R CCG_L CCG_R CST_L CST_R ILF_L ILF_R SLFP_L SLFP_R
## ATR_L  1.00  0.56 -0.09 -0.17  0.28  0.28  0.17  0.21  0.08  0.12  0.27   0.28
## ATR_R  0.56  1.00  0.08 -0.19  0.28  0.32  0.21  0.27  0.19  0.31  0.24   0.33
## CAB_L -0.09  0.08  1.00  0.35  0.17  0.14  0.18  0.12  0.27  0.26  0.16   0.22
## CAB_R -0.17 -0.19  0.35  1.00  0.12 -0.15  0.01  0.01  0.16  0.16 -0.12  -0.09
## CCG_L  0.28  0.28  0.17  0.12  1.00  0.69  0.06  0.05  0.25  0.26  0.30   0.29
## CCG_R  0.28  0.32  0.14 -0.15  0.69  1.00  0.08  0.10  0.16  0.17  0.30   0.28
##       SLFT_L SLFT_R UNC_L UNC_R
## ATR_L   0.18   0.13 -0.18 -0.11
## ATR_R   0.25   0.20  0.06  0.08
```

```
## CAB_L   0.23   0.23  0.34  0.36
## CAB_R   0.11   0.12 -0.01  0.21
## CCG_L   0.37   0.18  0.00  0.13
## CCG_R   0.34   0.17 -0.07  0.09

corrplot(plot, method = 'number', type = 'lower', tl.col = "black", tl
.srt = 45, diag = FALSE, family = "sans")

# Plot Hidden
```

Correlations of Study Measures

```
correlationvars <- c("Income", "Age", "PCL5", "ADI", "MEIM", "CAPS5",
"ATR_FA", "CAB_FA", "CCG_FA", "CST_FA", "FMINOR_FA", "FMAJOR_FA", "ILF
_FA", "SLFP_FA",  "SLFT_FA", "UNC_FA")
corrdata <- Project1[correlationvars]

corrdata1 <- rename(corrdata,
  "FMINOR" = "FMINOR_FA",
    "FMAJOR" = "FMAJOR_FA",
    "SLFT" = "SLFT_FA",
    "CST" = "CST_FA",
    "CCG" = "CCG_FA",
    "CAB" = "CAB_FA",
    "SLFP" = "SLFP_FA",
    "UNC" = "UNC_FA",
    "ATR" = "ATR_FA",
    "ILF" = "ILF_FA")

test = cor.mtest(corrdata1, conf.level = 0.95)
plot<-cor(corrdata1, use = "pairwise.complete.obs")
head(round(plot,2))
```

```
##        Income   Age  PCL5   ADI  MEIM CAPS5   ATR   CAB   CCG   CST
FMINOR
## Income   1.00  0.00 -0.04 -0.43 -0.04 -0.21  0.05 -0.03  0.04  0.20
-0.13
## Age      0.00  1.00 -0.23  0.04 -0.13 -0.12 -0.12  0.01 -0.09 -0.04
-0.17
## PCL5    -0.04 -0.23  1.00 -0.02  0.06  0.29  0.17  0.01  0.11  0.17
-0.07
## ADI     -0.43  0.04 -0.02  1.00  0.14  0.02  0.06 -0.13  0.11 -0.17
0.00
## MEIM    -0.04 -0.13  0.06  0.14  1.00  0.00  0.01 -0.01  0.02 -0.10
-0.06
## CAPS5   -0.21 -0.12  0.29  0.02  0.00  1.00 -0.11 -0.03 -0.17 -0.09
-0.02
```

```
##         FMAJOR   ILF  SLFP  SLFT   UNC
## Income    0.18  0.09  0.07  0.03  0.03
## Age      -0.02 -0.17 -0.17 -0.11 -0.02
## PCL5     -0.05  0.09  0.23  0.21  0.02
## ADI      -0.28 -0.11  0.01 -0.04  0.01
## MEIM      0.07  0.10  0.05  0.02  0.08
## CAPS5     0.03  0.00 -0.05  0.02  0.12
```

```
corrplot(plot, p.mat = test$p, type = "lower", tl.col = "black", tl.srt = 45, diag = FALSE, sig.level = c(0.001, 0.01, 0.05), pch.cex = 1, insig = 'label_sig', pch.col = 'grey20', family = "sans")

# Plot Hidden
```

Multivariate Multiple Regression Analysis

Neighborhood disadvantage is associated with lower FA values

This lm() model examines all of the FAs (using cbind()) as a function of ADI, ethnoracial group, Income, PCL, Gender, and Age.

```
model1<-lm(cbind(FMINOR_FA, FMAJOR_FA, SLFT_FA, CST_FA, CCG_FA, CAB_FA, ATR_FA, SLFP_FA, ILF_FA, UNC_FA)
           ~  Ethnoracial_group + c_Income + c_ADI + Gender + c_Age + c_PCL5, data=Project1)
summary(model1) # This provides the same output as 10 individual multiple regression models, each DV has their own separate set of coefficients, std. errors, and associated p-values
```

```
## Response FMINOR_FA :
##
## Call:
## lm(formula = FMINOR_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.116135 -0.025564  0.004776  0.026838  0.081496
##
## Coefficients:
##                            Estimate Std. Error t value Pr(>|t|)
## (Intercept)              0.4630968  0.0057317  80.795   <2e-16 ***
## Ethnoracial_groupWhite -0.0044849  0.0090963  -0.493   0.6227
## c_Income                -0.0021986  0.0012512  -1.757   0.0811 .
## c_ADI                   -0.0001793  0.0001872  -0.958   0.3398
## GenderFemale            -0.0132606  0.0070347  -1.885   0.0615 .
## c_Age                   -0.0006766  0.0003288  -2.058   0.0415 *
## c_PCL5                  -0.0002354  0.0002143  -1.098   0.2740
```

```
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.04134 on 141 degrees of freedom
## Multiple R-squared:  0.08554,    Adjusted R-squared:  0.04663
## F-statistic: 2.198 on 6 and 141 DF,  p-value: 0.04658
##
##
## Response FMAJOR_FA :
##
## Call:
## lm(formula = FMAJOR_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.221529 -0.016750  0.002816  0.027921  0.085948
##
## Coefficients:
##                            Estimate Std. Error t value Pr(>|t|)
## (Intercept)              5.676e-01  5.923e-03  95.835   <2e-16 ***
## Ethnoracial_groupWhite -4.451e-03  9.400e-03  -0.473   0.6366
## c_Income                 1.025e-03  1.293e-03   0.793   0.4292
## c_ADI                   -5.505e-04  1.935e-04  -2.845   0.0051 **
## GenderFemale            -7.302e-03  7.269e-03  -1.004   0.3169
## c_Age                   -3.218e-05  3.398e-04  -0.095   0.9247
## c_PCL5                  -1.117e-04  2.215e-04  -0.504   0.6149
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.04272 on 141 degrees of freedom
## Multiple R-squared:  0.09335,    Adjusted R-squared:  0.05477
## F-statistic:  2.42 on 6 and 141 DF,  p-value: 0.02951
##
##
## Response SLFT_FA :
##
## Call:
## lm(formula = SLFT_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.067911 -0.012328 -0.002311  0.012626  0.066236
##
## Coefficients:
```

```
##                            Estimate Std. Error t value Pr(>|t|)
## (Intercept)              4.015e-01  2.937e-03 136.717  < 2e-16 ***
## Ethnoracial_groupWhite   1.292e-02  4.660e-03   2.773  0.00631 **
## c_Income                -2.975e-04  6.411e-04  -0.464  0.64335
## c_ADI                    7.695e-05  9.593e-05   0.802  0.42381
## GenderFemale             9.639e-04  3.604e-03   0.267  0.78952
## c_Age                   -1.640e-04  1.685e-04  -0.974  0.33191
## c_PCL5                   2.408e-04  1.098e-04   2.193  0.02994 *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.02118 on 141 degrees of freedom
## Multiple R-squared:  0.098,  Adjusted R-squared:  0.05961
## F-statistic: 2.553 on 6 and 141 DF,  p-value: 0.02233
##
##
## Response CST_FA :
##
## Call:
## lm(formula = CST_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.045421 -0.011295 -0.000038  0.011348  0.050421
##
## Coefficients:
##                            Estimate Std. Error t value Pr(>|t|)
## (Intercept)              4.281e-01  2.583e-03 165.717   <2e-16 ***
## Ethnoracial_groupWhite   5.505e-03  4.100e-03   1.343   0.1815
## c_Income                 8.125e-04  5.639e-04   1.441   0.1519
## c_ADI                   -3.973e-05  8.439e-05  -0.471   0.6385
## GenderFemale            -1.354e-03  3.171e-03  -0.427   0.6700
## c_Age                   -1.501e-06  1.482e-04  -0.010   0.9919
## c_PCL5                   2.090e-04  9.661e-05   2.163   0.0322 *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.01863 on 141 degrees of freedom
## Multiple R-squared:  0.09308,    Adjusted R-squared:  0.05448
## F-statistic: 2.412 on 6 and 141 DF,  p-value: 0.02999
##
##
## Response CCG_FA :
##
## Call:
```

```
## lm(formula = CCG_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.097963 -0.026848 -0.001249  0.027160  0.085293
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)
## (Intercept)              0.5212377  0.0053604  97.239   <2e-16 ***
## Ethnoracial_groupWhite -0.0102531  0.0085069  -1.205    0.230
## c_Income                 0.0015662  0.0011702   1.338    0.183
## c_ADI                    0.0001936  0.0001751   1.105    0.271
## GenderFemale            -0.0128375  0.0065789  -1.951    0.053 .
## c_Age                   -0.0001744  0.0003075  -0.567    0.572
## c_PCL5                   0.0003095  0.0002005   1.544    0.125
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03866 on 141 degrees of freedom
## Multiple R-squared:  0.07408,    Adjusted R-squared:  0.03468
## F-statistic:  1.88 on 6 and 141 DF,  p-value: 0.08824
##
##
## Response CAB_FA :
##
## Call:
## lm(formula = CAB_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.119972 -0.018440 -0.001086  0.020164  0.142075
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)
## (Intercept)              3.352e-01  5.146e-03  65.142   <2e-16 ***
## Ethnoracial_groupWhite -1.636e-03  8.167e-03  -0.200   0.8415
## c_Income                -1.338e-03  1.123e-03  -1.191   0.2355
## c_ADI                   -3.367e-04  1.681e-04  -2.003   0.0471 *
## GenderFemale            -7.172e-03  6.316e-03  -1.136   0.2581
## c_Age                    1.189e-04  2.952e-04   0.403   0.6877
## c_PCL5                   4.827e-05  1.924e-04   0.251   0.8023
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
```

```
## Residual standard error: 0.03712 on 141 degrees of freedom
## Multiple R-squared:  0.03687,    Adjusted R-squared:  -0.004116
## F-statistic: 0.8996 on 6 and 141 DF,  p-value: 0.4971
##
##
## Response ATR_FA :
##
## Call:
## lm(formula = ATR_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##      Min       1Q   Median       3Q      Max
## -0.07802 -0.01572 -0.00230  0.01976  0.06362
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)
## (Intercept)              0.4254700  0.0035213 120.828   <2e-16 ***
## Ethnoracial_groupWhite   0.0041464  0.0055883   0.742   0.4593
## c_Income                 0.0005250  0.0007687   0.683   0.4957
## c_ADI                    0.0001472  0.0001150   1.280   0.2027
## GenderFemale            -0.0088490  0.0043217  -2.048   0.0425 *
## c_Age                   -0.0001542  0.0002020  -0.763   0.4466
## c_PCL5                   0.0002876  0.0001317   2.184   0.0306 *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.0254 on 141 degrees of freedom
## Multiple R-squared:  0.07922,    Adjusted R-squared:  0.04004
## F-statistic: 2.022 on 6 and 141 DF,  p-value: 0.06657
##
##
## Response SLFP_FA :
##
## Call:
## lm(formula = SLFP_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.053898 -0.016800  0.000195  0.014201  0.086495
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)
## (Intercept)              0.3992967  0.0032996 121.012  < 2e-16 ***
## Ethnoracial_groupWhite   0.0136783  0.0052365   2.612  0.00997 **
```

```
## c_Income                0.0004386  0.0007203   0.609  0.54359
## c_ADI                   0.0001882  0.0001078   1.746  0.08292 .
## GenderFemale            0.0044665  0.0040497   1.103  0.27194
## c_Age                  -0.0003367  0.0001893  -1.779  0.07742 .
## c_PCL5                  0.0002833  0.0001234   2.296  0.02317 *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.0238 on 141 degrees of freedom
## Multiple R-squared:  0.1268, Adjusted R-squared:  0.08962
## F-statistic: 3.412 on 6 and 141 DF,  p-value: 0.003565
##
##
## Response ILF_FA :
##
## Call:
## lm(formula = ILF_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.071422 -0.020558 -0.000751  0.020525  0.110988
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)
## (Intercept)             4.582e-01  4.558e-03 100.531   <2e-16 ***
## Ethnoracial_groupWhite  7.245e-03  7.234e-03   1.002   0.3183
## c_Income                2.813e-04  9.950e-04   0.283   0.7778
## c_ADI                  -7.896e-05  1.489e-04  -0.530   0.5968
## GenderFemale           -6.527e-03  5.594e-03  -1.167   0.2453
## c_Age                  -4.609e-04  2.615e-04  -1.762   0.0802 .
## c_PCL5                  1.447e-04  1.705e-04   0.849   0.3974
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03288 on 141 degrees of freedom
## Multiple R-squared:  0.06388,    Adjusted R-squared:  0.02404
## F-statistic: 1.603 on 6 and 141 DF,  p-value: 0.1504
##
##
## Response UNC_FA :
##
## Call:
## lm(formula = UNC_FA ~ Ethnoracial_group + c_Income + c_ADI +
##     Gender + c_Age + c_PCL5, data = Project1)
##
```

```
## Residuals:
##       Min        1Q    Median        3Q       Max 
## -0.126395 -0.015868  0.004721  0.024823  0.072863 
##
## Coefficients:
##                           Estimate Std. Error t value Pr(>|t|)    
## (Intercept)              3.795e-01  5.120e-03  74.116   <2e-16 ***
## Ethnoracial_groupWhite -3.935e-03  8.125e-03  -0.484    0.629    
## c_Income                 5.584e-04  1.118e-03   0.500    0.618    
## c_ADI                    7.599e-06  1.673e-04   0.045    0.964    
## GenderFemale            -6.231e-03  6.284e-03  -0.992    0.323    
## c_Age                   -9.621e-06  2.937e-04  -0.033    0.974    
## c_PCL5                   8.688e-05  1.915e-04   0.454    0.651    
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03693 on 141 degrees of freedom
## Multiple R-squared:  0.01101,    Adjusted R-squared:  -0.03108 
## F-statistic: 0.2616 on 6 and 141 DF,  p-value: 0.9538
```

```
Anova(model1)
```

```
##
## Type II MANOVA Tests: Pillai test statistic
##                    Df test stat approx F num Df den Df  Pr(>F)  
## Ethnoracial_group   1  0.109169   1.6176     10    132 0.10812  
## c_Income            1  0.090374   1.3115     10    132 0.23067  
## c_ADI               1  0.126278   1.9078     10    132 0.04935 *
## Gender              1  0.099736   1.4624     10    132 0.16049  
## c_Age               1  0.087304   1.2626     10    132 0.25803  
## c_PCL5              1  0.092560   1.3464     10    132 0.21252  
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

ADI is the only significant predictor in the multivariate model.

Exploratory analysis: MEIM as a protective factor

These glm models examined all of the FAs separately as a function of ADI, LEC, Income, PCL, Gender, and Age. It includes a ADI x MEIM interaction term. Ethnoracial group is not included in the following models, as this variable was not significant in the multivariate model (also because MEIM scores are so highly interrelated to race and ethnicity).

```
FMINOR<-lm(FMINOR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_In
come + c_Age + c_PCL5, data=Project1)
summary(FMINOR)
```

```
##
## Call:
## lm(formula = FMINOR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.117904 -0.026164  0.003421  0.026943  0.092269
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   0.4625611  0.0051434  89.934   <2e-16 ***
## c_MEIM       -0.0031217  0.0040831  -0.765   0.4458
## c_ADI        -0.0001219  0.0001748  -0.697   0.4867
## GenderFemale -0.0129739  0.0070079  -1.851   0.0662 .
## c_Income     -0.0020830  0.0012068  -1.726   0.0865 .
## c_Age        -0.0006896  0.0003298  -2.091   0.0384 *
## c_PCL5       -0.0001782  0.0002155  -0.827   0.4099
## c_MEIM:c_ADI -0.0003073  0.0001906  -1.612   0.1091
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.04104 on 140 degrees of freedom
## Multiple R-squared:  0.1051, Adjusted R-squared:  0.06036
## F-statistic: 2.349 on 7 and 140 DF,  p-value: 0.02681
```

```
FMAJOR<-lm(FMAJOR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_In
come + c_Age + c_PCL5, data=Project1)
summary(FMAJOR)
```

```
##
## Call:
## lm(formula = FMAJOR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.221747 -0.016226  0.003493  0.021965  0.083733
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   5.671e-01  5.335e-03 106.284   <2e-16 ***
## c_MEIM        6.093e-03  4.236e-03   1.439   0.1525
## c_ADI        -5.558e-04  1.813e-04  -3.066   0.0026 **
## GenderFemale -8.144e-03  7.270e-03  -1.120   0.2645
## c_Income      8.516e-04  1.252e-03   0.680   0.4975
```

```
## c_Age         3.631e-05  3.422e-04   0.106   0.9156
## c_PCL5       -1.058e-04  2.236e-04  -0.473   0.6369
## c_MEIM:c_ADI -8.101e-05  1.977e-04  -0.410   0.6826
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.04258 on 140 degrees of freedom
## Multiple R-squared:  0.1059, Adjusted R-squared:  0.06117
## F-statistic: 2.368 on 7 and 140 DF,  p-value: 0.02564
```

```
UNC<-lm(UNC_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_Income +
c_Age + c_PCL5, data=Project1)
summary(UNC)
```

```
##
## Call:
## lm(formula = UNC_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.126504 -0.016688  0.004501  0.024660  0.072294
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   3.790e-01  4.625e-03  81.938   <2e-16 ***
## c_MEIM        3.747e-03  3.672e-03   1.020    0.309
## c_ADI         1.401e-05  1.572e-04   0.089    0.929
## GenderFemale -6.773e-03  6.302e-03  -1.075    0.284
## c_Income      4.550e-04  1.085e-03   0.419    0.676
## c_Age         3.624e-05  2.966e-04   0.122    0.903
## c_PCL5        1.008e-04  1.938e-04   0.520    0.604
## c_MEIM:c_ADI -1.097e-04  1.714e-04  -0.640    0.523
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03691 on 140 degrees of freedom
## Multiple R-squared:  0.01912,    Adjusted R-squared:  -0.02993
## F-statistic: 0.3898 on 7 and 140 DF,  p-value: 0.9071
```

```
CAB<-lm(CAB_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_Income +
c_Age + c_PCL5, data=Project1)
summary(CAB)
```

```
##
## Call:
## lm(formula = CAB_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
```

```
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.118118 -0.018211 -0.000879  0.020493  0.142000
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   3.349e-01  4.668e-03  71.744   <2e-16 ***
## c_MEIM        1.031e-03  3.705e-03   0.278   0.7813
## c_ADI        -3.309e-04  1.586e-04  -2.087   0.0387 *
## GenderFemale -7.286e-03  6.360e-03  -1.146   0.2539
## c_Income     -1.407e-03  1.095e-03  -1.285   0.2010
## c_Age         1.287e-04  2.993e-04   0.430   0.6678
## c_PCL5        4.743e-05  1.956e-04   0.242   0.8087
## c_MEIM:c_ADI -5.142e-06  1.730e-04  -0.030   0.9763
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03725 on 140 degrees of freedom
## Multiple R-squared:  0.03713,    Adjusted R-squared:  -0.01101
## F-statistic: 0.7712 on 7 and 140 DF,  p-value: 0.6124

CST<-lm(CST_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_Income +
c_Age + c_PCL5, data=Project1)
summary(CST)

##
## Call:
## lm(formula = CST_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.045249 -0.012221 -0.000222  0.011604  0.047277
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   4.292e-01  2.341e-03 183.350   <2e-16 ***
## c_MEIM       -2.085e-03  1.858e-03  -1.122   0.2639
## c_ADI        -6.874e-05  7.955e-05  -0.864   0.3890
## GenderFemale -1.113e-03  3.190e-03  -0.349   0.7277
## c_Income      9.755e-04  5.493e-04   1.776   0.0779 .
## c_Age        -2.498e-05  1.501e-04  -0.166   0.8681
## c_PCL5        1.985e-04  9.810e-05   2.023   0.0450 *
## c_MEIM:c_ADI  8.256e-05  8.675e-05   0.952   0.3429
```

```
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.01868 on 140 degrees of freedom
## Multiple R-squared:  0.09485,    Adjusted R-squared:  0.04959
## F-statistic: 2.096 on 7 and 140 DF,  p-value: 0.04778
```

```
CCG<-lm(CCG_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_Income +
c_Age + c_PCL5, data=Project1)
summary(CCG)
```

```
##
## Call:
## lm(formula = CCG_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.087631 -0.027699 -0.000604  0.028057  0.080756
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   0.5173288  0.0048316 107.073   <2e-16 ***
## c_MEIM       -0.0006057  0.0038355  -0.158   0.8748
## c_ADI         0.0002726  0.0001642   1.661   0.0990 .
## GenderFemale -0.0122211  0.0065830  -1.856   0.0655 .
## c_Income      0.0009390  0.0011336   0.828   0.4089
## c_Age        -0.0002209  0.0003098  -0.713   0.4770
## c_PCL5        0.0002488  0.0002025   1.229   0.2211
## c_MEIM:c_ADI  0.0003227  0.0001790   1.803   0.0736 .
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03856 on 140 degrees of freedom
## Multiple R-squared:  0.0858, Adjusted R-squared:  0.04009
## F-statistic: 1.877 on 7 and 140 DF,  p-value: 0.07766
```

```
ILF<-lm(ILF_FA ~c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + c_Income +
c_Age + c_PCL5, data=Project1)
summary(ILF)
```

```
##
## Call:
## lm(formula = ILF_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
```

```
##       Min        1Q    Median        3Q       Max
## -0.075217 -0.021790  0.000308  0.022691  0.118073
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   0.4600903  0.0041145 111.823   <2e-16 ***
## c_MEIM        0.0037946  0.0032663   1.162    0.247
## c_ADI        -0.0001603  0.0001398  -1.147    0.253
## GenderFemale -0.0071180  0.0056060  -1.270    0.206
## c_Income      0.0003866  0.0009654   0.400    0.689
## c_Age        -0.0004235  0.0002639  -1.605    0.111
## c_PCL5        0.0001164  0.0001724   0.675    0.501
## c_MEIM:c_ADI  0.0001470  0.0001525   0.964    0.337
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.03283 on 140 degrees of freedom
## Multiple R-squared:  0.07304,    Adjusted R-squared:  0.02669
## F-statistic: 1.576 on 7 and 140 DF,  p-value: 0.1473
```

```
ATR<-lm(ATR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI +  Gender + c_Income
+ c_Age + c_PCL5, data=Project1)
summary(ATR)
```

```
##
## Call:
## lm(formula = ATR_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     c_Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.077651 -0.015992 -0.003025  0.019196  0.065900
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   4.264e-01  3.197e-03 133.388   <2e-16 ***
## c_MEIM       -2.227e-04  2.538e-03  -0.088   0.9302
## c_ADI         1.165e-04  1.086e-04   1.073   0.2853
## GenderFemale -8.858e-03  4.356e-03  -2.034   0.0439 *
## c_Income      6.281e-04  7.501e-04   0.837   0.4038
## c_Age        -1.575e-04  2.050e-04  -0.768   0.4437
## c_PCL5        2.773e-04  1.340e-04   2.070   0.0403 *
## c_MEIM:c_ADI  6.653e-05  1.185e-04   0.562   0.5752
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
```

```
## Residual standard error: 0.02551 on 140 degrees of freedom
## Multiple R-squared:  0.07773,    Adjusted R-squared:  0.03161
## F-statistic: 1.686 on 7 and 140 DF,  p-value: 0.1172
```

```
SLFT<-lm(SLFT_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender + Income +
c_Age + c_PCL5, data=Project1)
summary(SLFT)
```

```
##
## Call:
## lm(formula = SLFT_FA ~ c_MEIM * c_ADI + c_MEIM + c_ADI + Gender +
##     Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.074344 -0.012602 -0.001106  0.013587  0.078303
##
## Coefficients:
##                 Estimate Std. Error t value Pr(>|t|)
## (Intercept)   4.044e-01  4.020e-03 100.589   <2e-16 ***
## c_MEIM       -1.047e-04  2.144e-03  -0.049   0.9611
## c_ADI        -2.249e-05  9.176e-05  -0.245   0.8067
## GenderFemale  8.377e-04  3.680e-03   0.228   0.8202
## Income        2.959e-05  6.336e-04   0.047   0.9628
## c_Age        -1.665e-04  1.732e-04  -0.961   0.3381
## c_PCL5        2.111e-04  1.132e-04   1.866   0.0642 .
## c_MEIM:c_ADI  1.910e-04  1.001e-04   1.908   0.0584 .
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.02155 on 140 degrees of freedom
## Multiple R-squared:  0.07294,    Adjusted R-squared:  0.02659
## F-statistic: 1.574 on 7 and 140 DF,  p-value: 0.148
```

```
SLFP<-lm(SLFP_FA ~ c_MEIM * c_ADI + c_MEIM + + c_ADI + Gender + Income
+ c_Age + c_PCL5, data=Project1)
summary(SLFP) #There is a significant interaction between MEIM and ADI
, individuals living in more disadvantaged neighborhoods with higher M
EIM scores have greater integrity in SLFP.
```

```
##
## Call:
## lm(formula = SLFP_FA ~ c_MEIM * c_ADI + c_MEIM + +c_ADI + Gender +
##     Income + c_Age + c_PCL5, data = Project1)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
```

```
## -0.054433 -0.014212 -0.001325  0.013365  0.099291
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   3.991e-01  4.478e-03  89.106   <2e-16 ***
## c_MEIM       -6.006e-05  2.388e-03  -0.025   0.9800
## c_ADI         8.214e-05  1.022e-04   0.804   0.4230
## GenderFemale  4.373e-03  4.099e-03   1.067   0.2879
## Income        7.415e-04  7.059e-04   1.050   0.2953
## c_Age        -3.434e-04  1.929e-04  -1.780   0.0773 .
## c_PCL5        2.421e-04  1.261e-04   1.921   0.0568 .
## c_MEIM:c_ADI  2.560e-04  1.115e-04   2.296   0.0231 *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.02401 on 140 degrees of freedom
## Multiple R-squared:  0.1178, Adjusted R-squared:  0.07369
## F-statistic: 2.671 on 7 and 140 DF,  p-value: 0.01264
```

Plot MEIM and ADI by Ethnoracial Group

```
MEIM <- ggplot(Project1, aes(Ethnoracial_group, MEIM, fill = Ethnoraci
al_group)) +
  stat_boxplot(geom = "errorbar", width = 0.2) +
  geom_boxplot() +
  scale_fill_brewer(palette = "Greens")

MEIM + theme_classic(
  base_size = 12,
  base_family = "sans") +
  ggtitle("MEIM by Ethnoracial Group") +
  labs(
    x = "Ethnoracial Group",
    y = "Multigroup Ethnic Identity Measures") +
  theme(plot.title = element_text(size = 12, family = "sans", face = "
bold", hjust = 0.5),
        legend.position = "none",
        axis.title.x = element_blank(),
        axis.text = element_text(size = 12, color = "black"), axis.tit
le = element_text(color = "black"))

# Plot Hidden

t.test(MEIM ~ Ethnoracial_group, var.equal = TRUE, data = Project1)

##
##  Two Sample t-test
```

```
##
## data:  MEIM by Ethnoracial_group
## t = 3.224, df = 146, p-value = 0.00156
## alternative hypothesis: true difference in means between group Marg
inalized Ethnoracial Identity and group White is not equal to 0
## 95 percent confidence interval:
##  0.190325 0.793317
## sample estimates:
## mean in group Marginalized Ethnoracial Identity
##                                        2.904321
##                              mean in group White
##                                        2.412500
```

```
ADI <- ggplot(Project1, aes(Ethnoracial_group, ADI, fill = Ethnoracial
_group)) +
  stat_boxplot(geom = "errorbar", width = 0.2) +
  geom_boxplot() +
  scale_fill_brewer(palette = "Greens")

ADI + theme_classic(
  base_size = 12,
  base_family = "sans") +
  ggtitle("Neighborhood Disadvantage by Ethnoracial Group") +
  labs(
    x = "Ethnoracial Group",
    y = "Area Deprivation Index (National Ranking)") +
  theme(plot.title = element_text(size = 12, family = "sans", face = "
bold", hjust = 0.5),
        legend.position = "none",
        axis.title.x = element_blank(),
        axis.text = element_text(size = 12, color = "black"), axis.tit
le = element_text(color = "black"))

# Plot Hidden

t.test(ADI ~ Ethnoracial_group, var.equal = TRUE, data = Project1)
```

```
##
##  Two Sample t-test
##
## data:  ADI by Ethnoracial_group
## t = 6.6725, df = 146, p-value = 4.867e-10
## alternative hypothesis: true difference in means between group Marg
inalized Ethnoracial Identity and group White is not equal to 0
## 95 percent confidence interval:
##  16.67110 30.70297
## sample estimates:
```

```
## mean in group Marginalized Ethnoracial Identity
##                                          75.28704
##                               mean in group White
##                                          51.60000
```

Plot MEIM x ADI interactions

```
Plot1 <- interact_plot(SLFP,
                       pred = c_ADI, modx = c_MEIM,
                       x.label = "Area Deprivation Index (National Ran
king)",
                       y.label = "SLFP Integrity (FA Values)",
                       legend.main = "MEIM",
                       line.thickness = 2, point.size = 3, plot.points
= TRUE, colors = "Greens",
                       main.title = "Interaction Between Neighborhood
Disadvantage and MEIM")

Plot1 + theme_classic() +
    theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
          axis.title = element_text(size = 12, color = "black", family
= "sans"),
          panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
          legend.text = element_text(size = 12, color = "black", famil
y = "sans"),
          legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
          axis.line.x = element_line(size = 1, color = "black"),
          axis.line.y = element_line(size = 1, color = "black"),
          plot.title = element_text(size = 12, family = "sans", face =
"bold", hjust = 0.5))

# Plot Hidden
```

Appendix B. Code for Project II

Project 2 Introduction, N = 115

This project explored the effect of socioeconomic circumstances on resting-state connectivity of the anterior cingulate cortex (ACC). Below, is a brief codebook of variables included in this analysis.

Data Codebook

PCL-5 - PTSD Checklist for DSM-5; baseline total PTSD symptom severity measured at T1.
ADI - Area Deprivation Index; a measure of a neighborhoods socioeconomic position.
Income - Income; measured on a semi-continuous scale where "1" represents annual household income of $0-10,000.
Gender - Gender; dummy-coded where "1" represents women and "0" indicates men.
Ethnoracial_group - Grouping variable to examine racial and ethnic differences; dummy-coded so that "0" represents individuals who identified as part of a marginalized ethnoracial group and "1" represents White participants.
Age - Years of Age; reported at T1.
CAPS5 - Clinician-Administered PTSD Scale for DSM-5; total PTSD symptom severity measured at T2.
MOS - Medical Outcome Study Social Support; evaluates general social support available to participants.
MEIM - Multigroup Ethnic Identity Measure; probes racial-ethnic identity and assesses elements of commitment to and exploration of their ethnoracial group.
Note: "c" before the variable indicates it was grand-mean centered.

Data Set-up

120 participants had usable resting-state scans. Six participants could not be geocoded. 9 were missing MOS (grand mean-imputed) and 8 were missing MEIM (group mean-imputed). We were interested in potential interactions between socioeconomic circumstances and resilience factors. Due to small sample sizes across various ethnoracial groups we aggregated our sample to examine individuals from marginalized ethnoracial groups and White participants. A total of 115 participants are included in the dataset.

```
Project2<- read.spss("C:/Users/eksears/Desktop/ProjectTwo_Dissertation
Data_ALLraceethn.sav", to.data.frame=TRUE)

## re-encoding from UTF-8
```

Data Analysis

Plot Primary Aim Correlations

```
correlationvars <- c("c_Income", "c_Age", "c_PCL5", "c_ADI", "c_MEIM",
"c_MOS") # Variable names
corrdata <- Project2[correlationvars] # Pull these variables from the
```

```
Dataset

corrdata1 <- rename(corrdata, # rename the variables
  "Income" = "c_Income",
  "Age" = "c_Age",
  "PCL-5 Scores" = "c_PCL5",
  "Area Deprivation Index" = "c_ADI",
  "MEIM" = "c_MEIM",
  "MOS" = "c_MOS")

test = cor.mtest(corrdata1, conf.level = 0.95)
plot <- cor(corrdata1, use = "pairwise.complete.obs")
head(round(plot, 2))

##                          Income   Age PCL-5 Scores Area Deprivation I
ndex  MEIM
## Income                     1.00  0.08        -0.17                   -
0.49 -0.01
## Age                        0.08  1.00        -0.20
0.01  0.05
## PCL-5 Scores              -0.17 -0.20         1.00
0.00  0.01
## Area Deprivation Index    -0.49  0.01         0.00
1.00  0.09
## MEIM                      -0.01  0.05         0.01
0.09  1.00
## MOS                        0.20  0.08        -0.07                   -
0.06  0.17
##                            MOS
## Income                    0.20
## Age                       0.08
## PCL-5 Scores             -0.07
## Area Deprivation Index -0.06
## MEIM                      0.17
## MOS                       1.00

corrplot(plot, p.mat = test$p, type = "lower", tl.col = "black", tl.sr
t = 45, diag = FALSE, sig.level = c(0.001, 0.01, 0.05), pch.cex = 1.5,
insig = 'label_sig', pch.col = 'grey20', order = 'AOE', family = "sans
")

# Plot hidden
```

Plot study measures

```r
Income_Race <- ggplot(Project2, aes(x = Ethnoracial_group, y = Income,
fill = Ethnoracial_group)) +
  stat_boxplot(geom = "errorbar", width = 0.2) +
  geom_boxplot() +
  scale_fill_brewer(palette = "Blues")

Income_Race + theme_classic(
  base_size = 12,
  base_family = "sans") +
  ggtitle("Annual Household Income by Ethnoracial Group") +
  labs(
    x = "Ethnoracial Group",
    y = "Income [$10,000 increments]") +
  theme(plot.title = element_text(size = 12, family = "sans", face =
"bold", hjust = 0.5),
        legend.position = "none",
        axis.title.x = element_blank(),
        axis.text = element_text(size = 12, color = "black"),
axis.title = element_text(color = "black"))
# Plot hidden

ADI_Race <- ggplot(Project2, aes(Ethnoracial_group, ADI, fill = Ethnor
acial_group)) +
  stat_boxplot(geom = "errorbar", width = 0.2) +
  geom_boxplot() +
  scale_fill_brewer(palette = "Blues")

ADI_Race + theme_classic(
  base_size = 12,
  base_family = "sans") +
  ggtitle("Neighborhood Disadvantage by Ethnoracial Group") +
  labs(
    x = "Ethnoracial Group",
    y = "Area Deprivation Index (National Ranking)") +
  theme(plot.title = element_text(size = 12, family = "sans", face = "
bold", hjust = 0.5),
        legend.position = "none",
        axis.title.x = element_blank(),
        axis.text = element_text(size = 12, color = "black"), axis.tit
le = element_text(color = "black"))

# Plot hidden
```

Resting-state Connectivity Analyses

In this section, I plot relationships between study measures and functional connectivity values (pulled from linear models ran in CONN).

Analysis I: Relationship between functional connectivity values and socioeconomic factors

Greater income was associated with less connectivity between anterior cingulate cortex and the visual network.

```
IncomeCluster1_Income <- ggplot(Project2, aes(x = c_Income, y = Income
_Cluster1)) +
  geom_point(size = 2) +
  geom_smooth(method = lm, linetype = "dashed", color = "blue") +
  theme_classic(base_size = 12) +
  ggtitle("ACC - Precuneus") +
  labs(
    x = "Annual Household Income",
    y = "Functional Connectivity (z)") +
  theme(axis.title = element_text(size = 12),
        axis.text = element_text(size = 12),
        plot.title = element_text(size = 12, family = "sans", hjust =
0.5, face = "bold"))

IncomeCluster1_Income

## `geom_smooth()` using formula 'y ~ x'

# Plot hidden

IncomeCluster2_Income <- ggplot(Project2, aes(x = c_Income, y = Income
_Cluster2)) +
  geom_point(size = 2) +
  geom_smooth(method = lm, linetype = "dashed", color = "blue") +
  theme_classic(base_size = 12) +
  ggtitle("ACC - Cerebellum") +
  labs(
    x = "Annual Household Income",
    y = "Functional Connectivity (z)") +
  theme(axis.title = element_text(size = 12),
        axis.text = element_text(size = 12),
        plot.title = element_text(size = 12, family = "sans", hjust =
0.5, face = "bold"))

IncomeCluster2_Income

## `geom_smooth()` using formula 'y ~ x'
```

```
# Plot hidden

IncomeCluster3_Income <- ggplot(Project2, aes(x = c_Income, y = Income
_Cluster3)) +
  geom_point(size = 2) +
  geom_smooth(method = lm, linetype = "dashed", color = "blue") +
  theme_classic(base_size = 12) +
  ggtitle("ACC - Primary Visual Cortex") +
  labs(
    x = "Annual Household Income",
    y = "Functional Connectivity (z)") +
  theme(axis.title = element_text(size = 12),
        axis.text = element_text(size = 12),
        plot.title = element_text(size = 12, family = "sans", hjust =
0.5, face = "bold"))

IncomeCluster3_Income

## `geom_smooth()` using formula 'y ~ x'

# Plot hidden
```

Analysis II: Investigating protective effects of two resilience measures in relation to ACC resting-state connectivity

I was interested in how socioeconomic factors interact with well-documented resilience measures, including general social support (MOS) and commitment to and exploration of one's own racial and/or ethnic group.

Correlations between socioeconomic factors and resilience measures

```
Income_ADI <- cor.test(Project2$c_Income, Project2$c_ADI, method = "pe
arson")
Income_ADI #Neighborhood disadvantage and income are significantly rel
ated

##
##  Pearson's product-moment correlation
##
## data:  Project2$c_Income and Project2$c_ADI
## t = -5.9295, df = 113, p-value = 3.375e-08
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.6153598 -0.3338046
## sample estimates:
##       cor
## -0.4871396
```

```r
ADI_MEIM <- cor.test(Project2$c_ADI, Project2$c_MEIM, method = "pearso
n")
ADI_MEIM #Neighborhood disadvantage is not significantly related to ME
IM
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$c_ADI and Project2$c_MEIM
## t = 0.93108, df = 113, p-value = 0.3538
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.09741216  0.26611341
## sample estimates:
##        cor
## 0.08725469
```

```r
Income_MEIM <- cor.test(Project2$c_Income, Project2$c_MEIM, method = "
pearson")
Income_MEIM #Income is not significantly related to MEIM
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$c_Income and Project2$c_MEIM
## t = -0.075584, df = 113, p-value = 0.9399
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.1899733  0.1762297
## sample estimates:
##          cor
## -0.007110178
```

```r
MOS_Income <- cor.test(Project2$c_MOS, Project2$c_Income, method = "pe
arson")
MOS_Income #Income is significantly related to MOS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$c_MOS and Project2$c_Income
## t = 2.2202, df = 113, p-value = 0.0284
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  0.02216841 0.37357405
## sample estimates:
```

```
##      cor
## 0.204449
```

```
MOS_ADI <- cor.test(Project2$c_MOS, Project2$c_ADI, method = "pearson"
)
MOS_ADI #Neighborhood Disadvantage is not significantly related to MOS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$c_MOS and Project2$c_ADI
## t = -0.62558, df = 113, p-value = 0.5329
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.2392840  0.1257152
## sample estimates:
##         cor
## -0.05874759
```

```
MEIM_Race <- ggplot(Project2, aes(x = Ethnoracial_group, y = MEIM, fil
l = Ethnoracial_group)) +
  stat_boxplot(geom = "errorbar", width = 0.2) +
  geom_boxplot() +
  scale_fill_brewer(palette = "Blues") # Plotting MEIM scores by Ethno
racial Group
```

```
MEIM_Race + theme_classic(
  base_size = 12,
  base_family = "sans") +
  ggtitle("MEIM by Ethnoracial Group") +
  labs(
    x = "Ethnoracial_group",
    y = "Multigroup Ethnic Identity Measure Scores") +
  theme(plot.title = element_text(size = 12, family = "sans", hjust =
0.5, face = "bold"),
        legend.position = "none",
        axis.title.x = element_blank(),
        axis.text = element_text(size = 12, color = "black"))
```

```
# Plot hidden
```

ADI X MOS - Cluster 1 (Putamen)

```
fit1 <- lm(ADIxMOS_Cluster1 ~ Gender + c_Age + c_Income + c_PCL5 + c_A
DI + c_MOS + c_ADI * c_MOS, data = Project2)
summary(fit1)
```

```
##
## Call:
## lm(formula = ADIxMOS_Cluster1 ~ Gender + c_Age + c_Income + c_PCL5
+
##     c_ADI + c_MOS + c_ADI * c_MOS, data = Project2)
##
## Residuals:
##      Min       1Q   Median       3Q      Max
## -0.36321 -0.08787 -0.02009  0.07829  0.38597
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)   0.1578562  0.0187497   8.419 1.86e-13 ***
## GenderFemale  0.0069423  0.0261579   0.265   0.7912
## c_Age        -0.0024674  0.0012754  -1.935   0.0557 .
## c_Income     -0.0019175  0.0047598  -0.403   0.6879
## c_PCL5       -0.0006302  0.0007776  -0.810   0.4195
## c_ADI         0.0003210  0.0006575   0.488   0.6265
## c_MOS         0.0147586  0.0132101   1.117   0.2664
## c_ADI:c_MOS   0.0032778  0.0006614   4.956 2.71e-06 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.1309 on 107 degrees of freedom
## Multiple R-squared:  0.2202, Adjusted R-squared:  0.1692
## F-statistic: 4.316 on 7 and 107 DF,  p-value: 0.0003045
```

```
Plot1 <- interact_plot(fit1,
                       pred = c_ADI, modx = c_MOS,
                       x.label = "Area Deprivation Index (National Ran
king)",
                       y.label = "ACC - Putamen Connectivity",
                       legend.main = "MOS Social Support Scale",
                       line.thickness = 2, point.size = 2, plot.points
= TRUE, colors = "seagreen",
                       main.title = "Interaction Between Neighborhood
Disadvantage and Social Support")

Plot1 + theme_classic() +
    theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
          axis.title = element_text(size = 12, color = "black", family
= "sans"),
          panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
          legend.text = element_text(size = 12, color = "black", famil
```

```
y = "sans"),
          legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
          axis.line.x = element_line(size = 1, color = "black"),
          axis.line.y = element_line(size = 1, color = "black"),
          plot.title = element_text(size = 12, family = "sans", hjust
= 0, face = "bold"))

# Plot hidden
```

ADI X MOS - Cluster 2 (Inferior Frontal Gyrus, R)

```
fit2 <- lm(ADIxMOS_Cluster2 ~ Gender + c_Age + c_Income + c_PCL5 + c_A
DI + c_MOS + c_ADI * c_MOS, data = Project2)
summary(fit2)

##
## Call:
## lm(formula = ADIxMOS_Cluster2 ~ Gender + c_Age + c_Income + c_PCL5
+
##     c_ADI + c_MOS + c_ADI * c_MOS, data = Project2)
##
## Residuals:
##      Min       1Q   Median       3Q      Max
## -0.30716 -0.08881  0.01993  0.08024  0.38791
##
## Coefficients:
##                Estimate Std. Error t value Pr(>|t|)
## (Intercept)  -6.755e-02  1.841e-02  -3.668 0.000382 ***
## GenderFemale  7.017e-02  2.569e-02   2.732 0.007376 **
## c_Age         9.308e-06  1.253e-03   0.007 0.994085
## c_Income      2.995e-03  4.675e-03   0.641 0.523069
## c_PCL5       -6.333e-04  7.637e-04  -0.829 0.408816
## c_ADI         1.474e-03  6.458e-04   2.282 0.024473 *
## c_MOS        -5.572e-03  1.297e-02  -0.429 0.668433
## c_ADI:c_MOS   3.533e-03  6.495e-04   5.439 3.42e-07 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.1286 on 107 degrees of freedom
## Multiple R-squared:  0.2839, Adjusted R-squared:  0.2371
## F-statistic: 6.061 on 7 and 107 DF,  p-value: 5.731e-06

Plot2 <- interact_plot(fit2,
                       pred = c_ADI, modx = c_MOS,
                       x.label = "Area Deprivation Index (National Ran
king)",
```

```
                        y.label = "ACC - Inferior Frontal Gyrus Connect
ivity",
                        legend.main = "MOS Social Support Scale", line.
thickness = 2,
                        point.size = 2, plot.points = TRUE, colors = "s
eagreen",
                        main.title = "Interaction Between Neighborhood
Disadvantage and Social Support")

Plot2 + theme_classic() +
    theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
          axis.title = element_text(size = 12, color = "black", family
= "sans"),
          panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
          legend.text = element_text(size = 12, color = "black", famil
y = "sans"),
          legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
          axis.line.x = element_line(size = 1, color = "black"),
          axis.line.y = element_line(size = 1, color = "black"),
          plot.title = element_text(size = 12, family = "sans", hjust
= 0, face = "bold"))

# Plot hidden
```

Income X MEIM - Cluster 1 (Occipital Cortex, L)

There was no three-way interaction between MEIM, racial and ethnic group, and income. This analysis is excluded. Two-way interaction between MEIM and income is plotted below.

```
fit3 <- lm(IncomeXMEIM_Cluster1 ~ Gender + c_Age + c_ADI + c_PCL5 + c_
Income + c_MEIM + c_Income * c_MEIM, data = Project2)
summary(fit3)

##
## Call:
## lm(formula = IncomeXMEIM_Cluster1 ~ Gender + c_Age + c_ADI +
##     c_PCL5 + c_Income + c_MEIM + c_Income * c_MEIM, data = Project2
)
##
## Residuals:
##      Min       1Q   Median       3Q      Max
## -0.27007 -0.07620  0.00086  0.07082  0.32158
##
```

```
## Coefficients:
##                   Estimate Std. Error t value Pr(>|t|)
## (Intercept)     -0.0266470  0.0169732  -1.570   0.1194
## GenderFemale    -0.0016917  0.0234581  -0.072   0.9426
## c_Age           -0.0013838  0.0011828  -1.170   0.2446
## c_ADI           -0.0012315  0.0006160  -1.999   0.0481 *
## c_PCL5           0.0001553  0.0007234   0.215   0.8304
## c_Income        -0.0103990  0.0043330  -2.400   0.0181 *
## c_MEIM           0.0093393  0.0138029   0.677   0.5001
## c_Income:c_MEIM  0.0211417  0.0041704   5.069 1.68e-06 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.122 on 107 degrees of freedom
## Multiple R-squared:  0.2446, Adjusted R-squared:  0.1951
## F-statistic: 4.948 on 7 and 107 DF,  p-value: 7.1e-05
```

```
Plot3 <- interact_plot(fit3,
                       pred = c_Income, modx = c_MEIM,
                       x.label = "Income",
                       y.label = "ACC - OC Connectivity (L)",
                       legend.main = "MEIM Scores",
                       line.thickness = 2, plot.points = TRUE, point.s
ize = 2, colors = "seagreen",
                       main.title = "Interaction Between Income and ME
IM")

Plot3 + theme_classic() +
     theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
           axis.title = element_text(size = 12, color = "black", family
= "sans"),
           panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
           legend.text = element_text(size = 12, color = "black", famil
y = "sans"),
           legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
           axis.line.x = element_line(size = 1, color = "black"),
           axis.line.y = element_line(size = 1, color = "black"),
           plot.title = element_text(size = 12, family = "sans", hjust
= 0, face = "bold"))

# Plot hidden
```

Income X MEIM - Cluster 2 (Occipital Cortex, R)

There was no three-way interaction between MEIM, racial and ethnic group, and income. This analysis is excluded. Two-way interaction between MEIM and income is plotted below.

```
fit4 <- lm(IncomeXMEIM_Cluster2 ~ Gender + c_Age + c_ADI + c_PCL5 + c_
Income + c_MEIM + c_Income * c_MEIM, data = Project2)
summary(fit4)

##
## Call:
## lm(formula = IncomeXMEIM_Cluster2 ~ Gender + c_Age + c_ADI +
##     c_PCL5 + c_Income + c_MEIM + c_Income * c_MEIM, data = Project2
)
##
## Residuals:
##      Min       1Q   Median       3Q      Max
## -0.38081 -0.07837 -0.00661  0.09163  0.33890
##
## Coefficients:
##                   Estimate Std. Error t value Pr(>|t|)
## (Intercept)     -0.0234244  0.0181545  -1.290   0.1997
## GenderFemale     0.0302306  0.0250907   1.205   0.2309
## c_Age           -0.0024301  0.0012651  -1.921   0.0574 .
## c_ADI           -0.0006890  0.0006589  -1.046   0.2981
## c_PCL5           0.0002721  0.0007738   0.352   0.7258
## c_Income        -0.0039406  0.0046346  -0.850   0.3971
## c_MEIM           0.0097751  0.0147635   0.662   0.5093
## c_Income:c_MEIM  0.0213227  0.0044606   4.780 5.61e-06 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.1305 on 107 degrees of freedom
## Multiple R-squared:  0.218,  Adjusted R-squared:  0.1669
## F-statistic: 4.262 on 7 and 107 DF,  p-value: 0.0003452

Plot4 <- interact_plot(fit4,
                       pred = c_Income, modx = c_MEIM,
                       x.label = "Income",
                       y.label = "ACC - OC Connectivity (R)",
                       legend.main = "MEIM Scores",
                       point.size = 2, line.thickness = 2, plot.points
= TRUE, colors = "seagreen",
                       main.title = "Interaction Between Income and ME
IM")

Plot4 + theme_classic() +
```

```
    theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
          axis.title = element_text(size = 12, color = "black", family
= "sans"),
          panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
          legend.text = element_text(size = 12, color = "black", famil
y = "sans"),
          legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
          axis.line.x = element_line(size = 1, color = "black"),
          axis.line.y = element_line(size = 1, color = "black"),
          plot.title = element_text(size = 12, family = "sans", hjust
= 0, face = "bold"))

# Plot hidden
```

Income X MEIM - Cluster 3 (hippocampus)

There was a significant three-way interaction between MEIM, ethnoracial group, and income. This finding is plotted below.

```
fit5 <- lm(IncomeXMEIM_Cluster3 ~ Gender + c_Age + c_PCL5 + c_ADI + c_
Income + c_MEIM + c_Income * c_MEIM * Ethnoracial_group, data = Projec
t2)
summary(fit5)

##
## Call:
## lm(formula = IncomeXMEIM_Cluster3 ~ Gender + c_Age + c_PCL5 +
##     c_ADI + c_Income + c_MEIM + c_Income * c_MEIM * Ethnoracial_gro
up,
##     data = Project2)
##
## Residuals:
##       Min        1Q    Median        3Q       Max
## -0.273311 -0.065676  0.007856  0.066964  0.259610
##
## Coefficients:
##                                        Estimate Std. Error t valu
e Pr(>|t|)
## (Intercept)                           0.1082939  0.0165281   6.55
2 2.28e-09
## GenderFemale                          0.0027386  0.0200027   0.13
7   0.8914
## c_Age                                 0.0006549  0.0009997   0.65
5   0.5138
```

```
## c_PCL5                                    -0.0002091  0.0006380  -0.32
8   0.7438
## c_ADI                                      0.0005257  0.0005539   0.94
9   0.3448
## c_Income                                   0.0036813  0.0049557   0.74
3   0.4593
## c_MEIM                                    -0.0170283  0.0157793  -1.07
9   0.2830
## Ethnoracial_groupWhite                     0.0461716  0.0287584   1.60
5   0.1114
## c_Income:c_MEIM                           -0.0330142  0.0054601  -6.04
6 2.40e-08
## c_Income:Ethnoracial_groupWhite           -0.0120610  0.0075935  -1.58
8   0.1153
## c_MEIM:Ethnoracial_groupWhite              0.0049970  0.0307093   0.16
3   0.8711
## c_Income:c_MEIM:Ethnoracial_groupWhite     0.0187156  0.0082151   2.27
8   0.0248
##
## (Intercept)                               ***
## GenderFemale
## c_Age
## c_PCL5
## c_ADI
## c_Income
## c_MEIM
## Ethnoracial_groupWhite
## c_Income:c_MEIM                           ***
## c_Income:Ethnoracial_groupWhite
## c_MEIM:Ethnoracial_groupWhite
## c_Income:c_MEIM:Ethnoracial_groupWhite *
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.1014 on 103 degrees of freedom
## Multiple R-squared:  0.335,  Adjusted R-squared:  0.264
## F-statistic: 4.717 on 11 and 103 DF,  p-value: 7.949e-06

c <- c("Marginalized Ethnoracial Identity", "White") # label mod2

Plot5 <- interact_plot(fit5,
                       pred = c_Income, modx = c_MEIM, mod2 = Ethnorac
ial_group,
                       mod2.labels = c,
                       x.label = "Income",
                       y.label = "ACC - Hippocampus Connectivity",
```

```
                      legend.main = "MEIM Scores",
                      point.size = 2, line.thickness = 2, plot.points
= TRUE, colors = "seagreen",
                      main.title = "Interaction Between Income and ME
IM")

Plot5 + theme_classic() +
     theme(axis.text = element_text(size = 12, color = "black", family
= "sans"),
           axis.title = element_text(size = 12, color = "black", family
= "sans"),
           panel.grid.major = element_blank(), panel.grid.minor = eleme
nt_blank(),
           legend.text = element_text(size = 12, color = "black", famil
y = "sans"),
           legend.title = element_text(size = 12, color = "black", fami
ly = "sans"),
           axis.line.x = element_line(size = 1, color = "black"),
           axis.line.y = element_line(size = 1, color = "black"),
           plot.title = element_text(size = 12, family = "sans", hjust
= 0, face = "bold"))

# Plot hidden
```

Analysis III: ACC functional connectivity values and CAPS5

```
IncomeCluster1_CAPS5 <- cor.test(Project2$Income_Cluster1, Project2$CA
PS5, method = "pearson")
IncomeCluster1_CAPS5

##
##  Pearson's product-moment correlation
##
## data:  Project2$Income_Cluster1 and Project2$CAPS5
## t = -0.91017, df = 103, p-value = 0.3649
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.2762585  0.1041251
## sample estimates:
##         cor
## -0.08932278

IncomeCluster2_CAPS5 <- cor.test(Project2$Income_Cluster2, Project2$CA
PS5, method = "pearson")
IncomeCluster2_CAPS5
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$Income_Cluster2 and Project2$CAPS5
## t = 0.43251, df = 103, p-value = 0.6663
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.1503141  0.2323469
## sample estimates:
##       cor
## 0.0425778
```

```
IncomeCluster3_CAPS5 <- cor.test(Project2$Income_Cluster3, Project2$CA
PS5, method = "pearson")
IncomeCluster3_CAPS5
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$Income_Cluster3 and Project2$CAPS5
## t = 0.64947, df = 103, p-value = 0.5175
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.1293856  0.2524387
## sample estimates:
##        cor
## 0.06386342
```

```
IncomeMEIMCluster1_CAPS <- cor.test(Project2$IncomeXMEIM_Cluster1, Pro
ject2$CAPS5, method = "pearson")
IncomeMEIMCluster1_CAPS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$IncomeXMEIM_Cluster1 and Project2$CAPS5
## t = 0.48793, df = 103, p-value = 0.6266
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.1449779  0.2375010
## sample estimates:
##       cor
## 0.0480217
```

```
IncomeMEIMCluster2_CAPS <- cor.test(Project2$IncomeXMEIM_Cluster2, Pro
ject2$CAPS5, method = "pearson")
IncomeMEIMCluster2_CAPS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$IncomeXMEIM_Cluster2 and Project2$CAPS5
## t = 1.4743, df = 103, p-value = 0.1435
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.04926711  0.32642682
## sample estimates:
##       cor
## 0.1437556
```

```
IncomeMEIMCluster3_CAPS <- cor.test(Project2$IncomeXMEIM_Cluster3, Pro
ject2$CAPS5, method = "pearson")
IncomeMEIMCluster3_CAPS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$IncomeXMEIM_Cluster3 and Project2$CAPS5
## t = -0.092489, df = 103, p-value = 0.9265
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.2004280  0.1828718
## sample estimates:
##          cor
## -0.009112847
```

```
ADIMOSCluster1_CAPS <- cor.test(Project2$ADIxMOS_Cluster1, Project2$CA
PS5, method = "pearson")
ADIMOSCluster1_CAPS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$ADIxMOS_Cluster1 and Project2$CAPS5
## t = 1.4297, df = 103, p-value = 0.1558
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.0535992  0.3225406
## sample estimates:
##       cor
## 0.1394991
```

```
ADIMOSCluster2_CAPS <- cor.test(Project2$ADIxMOS_Cluster2, Project2$CA
PS5, method = "pearson")
ADIMOSCluster2_CAPS
```

```
##
##  Pearson's product-moment correlation
##
## data:  Project2$ADIxMOS_Cluster2 and Project2$CAPS5
## t = -0.50549, df = 103, p-value = 0.6143
## alternative hypothesis: true correlation is not equal to 0
## 95 percent confidence interval:
##  -0.2391313  0.1432854
## sample estimates:
##         cor
## -0.04974603
```

```
correlation_CAPS <- c("Income_Cluster1", "Income_Cluster2", "Income_Cl
uster3", "IncomeXMEIM_Cluster1", "IncomeXMEIM_Cluster2", "IncomeXMEIM_
Cluster3", "ADIxMOS_Cluster1", "ADIxMOS_Cluster2", "CAPS5") # Variable
names
corrdata1_CAPS <- Project2[correlation_CAPS] # Pull these variables fr
om the Dataset

corrdata2_CAPS <- rename(corrdata1_CAPS, # rename the variables
  "Income: Precuneus" = "Income_Cluster1",
  "Income: Cerebellum" = "Income_Cluster2",
  "Income: PVC" = "Income_Cluster3",
  "IncomexMEIM: OC (L)" = "IncomeXMEIM_Cluster1",
  "IncomexMEIM: OC (R)" = "IncomeXMEIM_Cluster2",
  "IncomexMEIM: Hippocampus" = "IncomeXMEIM_Cluster3",
  "ADIxMOS: Putamen" = "ADIxMOS_Cluster1",
  "ADIxMOS: IFG" = "ADIxMOS_Cluster2",
  "CAPS-5" = "CAPS5" )

test = cor.mtest(corrdata2_CAPS, conf.level = 0.95)
plot <- cor(corrdata2_CAPS, use = "pairwise.complete.obs")
head(round(plot, 2))
```

```
##                          Income: Precuneus Income: Cerebellum Incom
e: PVC
## Income: Precuneus                     1.00                0.35
0.31
## Income: Cerebellum                    0.35                1.00
0.50
## Income: PVC                           0.31                0.50
1.00
## IncomexMEIM: OC (L)                   0.19                0.51
0.54
## IncomexMEIM: OC (R)                   0.13                0.36
0.26
```

```
## IncomexMEIM: Hippocampus             -0.07                0.01
0.06
##                           IncomexMEIM: OC (L) IncomexMEIM: OC (R)
## Income: Precuneus                        0.19                0.13
## Income: Cerebellum                       0.51                0.36
## Income: PVC                              0.54                0.26
## IncomexMEIM: OC (L)                      1.00                0.68
## IncomexMEIM: OC (R)                      0.68                1.00
## IncomexMEIM: Hippocampus                -0.07               -0.08
##                           IncomexMEIM: Hippocampus ADIxMOS: Putamen
ADIxMOS: IFG
## Income: Precuneus                           -0.07             0.01
-0.04
## Income: Cerebellum                           0.01             0.03
-0.04
## Income: PVC                                  0.06             0.06
-0.07
## IncomexMEIM: OC (L)                         -0.07            -0.02
-0.10
## IncomexMEIM: OC (R)                         -0.08            -0.01
-0.11
## IncomexMEIM: Hippocampus                     1.00            -0.04
-0.03
##                           CAPS-5
## Income: Precuneus         -0.09
## Income: Cerebellum         0.04
## Income: PVC                0.06
## IncomexMEIM: OC (L)        0.05
## IncomexMEIM: OC (R)        0.14
## IncomexMEIM: Hippocampus  -0.01
```

```
corrplot(plot, p.mat = test$p, type = "lower", tl.col = "black", tl.sr
t = 45, diag = FALSE, sig.level = c(0.001, 0.01, 0.05), pch.cex = 1.5,
insig = 'label_sig', pch.col = 'grey20', family = "sans")

# Plot Hidden
```

None of the connectivity values help predict who will develop PTSD, even before adjusting for covariates.

The End of Empirical Work!

E. Kate Webb

Curriculum Vitae

Education

PhD **Psychology, University of Wisconsin-Milwaukee (UWM)**
2021
Dissertation: Effects of neighborhood socioeconomic disadvantage on brain functional and structural connectivity in trauma-exposed adults
Advisor: Christine Larson, PhD

MS **Psychology, UWM**
2020 Thesis: *Shaped by the environment: the influence of childhood trauma exposure, individual socioeconomic position, and neighborhood disadvantage on brain morphology*

BA **Albion College**, Albion, MI
2016
Major: Psychological Sciences, *Minor*: Cellular and Molecular Biology
Concentration: Neuroscience, *Cum Laude*

Appointments

University of Wisconsin-Milwaukee, Milwaukee, WI
2018 - 2021
Graduate Student Researcher (under the direction of Dr. Christine Larson)

- Design and implement neuroimaging experiments using functional magnetic imaging (fMRI) to investigate brain characteristics in the aftermath of a traumatic injury in both adults and adolescents.
- Utilize univariate, multivariate, machine learning, and multilevel statistical approaches to analyze cross-sectional, multi-modal, and longitudinal datasets.
- Program behavioral tasks in E-Prime and preprocess/analyze MRI data with the following programs: AFNI, SPM, Freesurfer, CONN, and PRONTO.

APOPO vzw, Morogoro, Tanzania.
Scientific Advisor 2019 – present

- Consult with research team on manuscripts and grants.
- Develop scientific outreach programming for APOPO's Zoo Program (partnered with American Zoological Association-accredited zoos).
- Create training materials for zoo biologists and provide on-the-ground support to zoo staff working with ambassador animals.

Research Technician (under the direction of Dr. Cynthia Fast) 2017 – 2019

- Developed training protocols to enhance the training and operations of APOPO's scent detection program (APOPO trains African giant pouched rats to detect landmines in post-conflict zones and tuberculosis in human sputum samples).
- Participated in the proposal, design, execution, and analysis of 5 research studies.

- Managed a team of 8 APOPO research trainers and 3 laboratory staff.

Duke University, Center for Child and Family Policy, Durham, NC
Research Aide (under the direction of Dr. Ken Dodge)
2016 - 2017

- Facilitated over 250 interviews (in-person and virtual) from two longitudinal studies, including the 25-year FAST Track Study.

Honors + Support

2021 Trauma Psychology Grant, American Psychological Foundation, $3,000
2021 APF / COGDOP scholarship, American Psychological Foundation, $2,000
2021-22 TL1 NRSA Predoctoral Trainee (renewed), National Institutes of Health
2021-22 Early Career Policy Ambassador Fellow, Society for Neuroscience
2020 Trainee Professional Development Award, Society for Neuroscience
2020 Team Science Grant, MCW Clinical and Translational Science Institute, $250
2020 Early Graduate Researcher Award, American Psychological Association, $1,000
2020-21 TL1 NRSA Predoctoral Trainee, National Institutes of Health
2020-22 Department Diversity Committee Fellow, UWM
2020-21 Distinguished Graduate Student Fellowship, UWM, *declined*
2020-21 Young Scientist, Annual Lindau Nobel Laureate Meeting, *postponed*
2020 Behavioral Management Fund Travel Award+, ABMA, $1,000
2019 Psychology Summer Graduate Research Fellowship, UWM, $4,000

Publications

15. Webb, E.K., Etter, J.A., & Kwasa, J.A. (*accepted*). Addressing racial bias in human neuroscience methods. *Nature Neuroscience*.
14. Webb, E.K.*, Ward, R.*, Mathew, A.S., Price, M., Weis, C., Trevino, C., deRoon-Cassini, & Larson, C. (*2021*). Role of Pain and Socioenvironmental Factors on PTSD Symptoms in Traumatically Injured Adults: A One-Year Prospective Study. *Journal of Traumatic Stress*. (*co-first authors).
13. Webb, E.K.*, Bird, C.M., de-Roon-Cassini, T., Weis, C., Huggins, A., Fitzgerald, J.M., Miskovich, T., Bennett, K., Krukowski, J., Torres, L., & Larson, C. (*2021).* Racial discrimination and resting-state functional connectivity of salience network nodes in traumatically injured adults. *JAMA Network Open*.
12. Weis, C.N., Huggins, A., Miskovich, T., Fitzgerald, J., Bennet, K., Krukowski, J., Webb, E.K., deRoon-Cassini, & Larson, C. (2021). Acute white matter integrity post-trauma and prospective PTSD symptoms. *Frontiers in Human Neuroscience*.
11. Fitzgerald, J.M.*, Webb, E.K.*, Weis, C., Huggins, A., Bennett, K., Parisi, E., Kallenbach, M., Krukowski, J., deRoon-Cassini, & Larson, C. (2021). Hippocampus resting-state functional connectivity forecasts individual PTSD symptoms: A data-driven approach. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*
10. Webb, E.K.*, Weis, C.*, Huggins, A., Fitzgerlad, J.M., Bennett, K., Bird, C.M., Parisi E.A., Kallenbach, M., Miskovich, T., Krukowski, J., deRoon-Cassini, & Larson, C. (*2021*). Neural impact of neighborhood socioeconomic disadvantage in traumatically injured adults. *Neurobiology of Stress*.

9. Weis, C.N., Webb, E.K., deRoon-Cassini, T.A., & Larson, C.L. (2021). Emotion dysregulation following trauma: shared neurocircuitry of traumatic brain injury and trauma-related psychiatric disorders. *Biological Psychiatry*.
8. Weis, C.N.*, Webb, E.K.*, Huggins, A.A, Kallenbach, M., Miskovich, T.A., Fitzgerald, J.M, Bennett, K.P, Krukowski, J.L, deRoon-Cassini, T., & Larson, C.L. (2021). Stability of hippocampal subfield volumes after trauma and relationship to development of PTSD symptoms. *Neuroimage*.
7. Weis, C.N, Webb, E.K., Damiano, S., Larson, C.L., & deRoon-Cassini, T. (2021). Scoring the Life Events Checklist: comparison of three scoring methods. *Psychological Trauma: Theory, Research, Practice, and Policy*.
6. Bird, C.*, Webb, E.K.*, Schramm, A., Torres, L., Larson, C., & deRoon-Cassini, T. (2021). Racial discrimination is associated with acute post-traumatic stress symptoms and predicts future PTSD symptom severity in trauma-exposed Black American adults. *Journal of Traumatic Stress*.

*** A non-academic version of this research (R shiny app) was created for dissemination: *http://milwaukeetrauma.com/science/projects/racial-discrimination-and-ptsd*

5. Webb, E.K., Weis, C., Huggins, A., Parisi, E., Bennett, K., Miskovich, T., Hanson, J., deRoon-Cassini, & Larson, C. (2021). Neighborhood disadvantage is associated with stable deficits in neurocognitive functioning in traumatically injured adults. *Health and Place, 67*, 102493.
4. Fried, A.*, Dunphy, C.*, Silverman, M.*, Webb, E.K.* (2020). Emerging issues in the responsible conduct of psychological science. *Translational Issues in Psychological Sciences, 6*(3), 191-195.
3. Webb, E.K.*, Huggins, A.*, Belleau, E., Taubitz, L., Hanson, J., deRoon-Cassini, T., & Larson, C. (2020). Altered resting-state functional connectivity of periaqueductal gray prospectively predicts posttraumatic stress symptoms. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 5*(9), 891-900. (* co-first authors).
2. Webb, E. K., Saccardo, C. C., Poling, A., Cox, C., & Fast, C. D. (2020). Rapidly training African giant pouched rats (Cricetomys ansorgei) with multiple targets for scent detection. *Behavioural Processes*, *174*, 104085.
1. Lewon, M., Webb, E.K., Brotheridge, S.M., Cox, C., & Fast, C. (2019). Behavioral skills training in scent detection research: interactions between trainer and animal behavior. *Journal of Applied Behavior Analysis, 52*(3), 682-700. DOI: 10.1002/jaba.566.

In Revisions + Under Review + In Preparation

Weis, C., Webb, E.K., Bennett, K., Huggins, A., Fitzgerald, J.M., Miskovich, T., Krukowski, J., deRoon-Cassini, & Larson, C. (*revise and resubmit at Biological Psychiatry: Global Open Science*). Neighborhood socioeconomic disadvantage and the neurobiology uncertainity in traumatically injured adults.

Webb, E.K., Weis, C., Huggins, A., Bennett, K., Krukowski, J., deRoon-Cassini, & Larson, C. (*in prep*). Resting-state connectivity of BNST predicts future PTSD symptoms in traumatically injured Black American adults.

Webb, E.K.*, Ward, R.*, Weis, C., Huggins, A., Fitzgerald, J., Bennett, K., Parisi, E., Krukowski, J., deRoon-Cassini, & Larson, C. (*in prep*). Neural effects of neighborhood disadvantage during response inhibition in traumatically injured adults.

Meetings + Conferences

KEYNOTE LECTURES

1. Webb, E.K. *Saving lives one sniff at a time! African giant pouched rats as humanitarian biosensors*. Invited Keynote at the Animal Behavior Management Alliance conference, Portland, OR, April 2019.

INVITED TALKS

4. Webb, E.K. *Training African pouched rats to save lives.* Invited talk at Milwaukee County Zoo, Milwaukee, WI, August 2019.
3. Webb, E.K. *Training African pouched rats to save lives: from pup to hero.* Invited talk at Point Defiance Zoo and Aquarium, Tacoma, WA, July 2019.
2. Webb, E.K. *Training African pouched rats to save lives: from pup to hero.* Public educational lecture at Point Defiance Zoo and Aquarium, Tacoma, WA, July 2019.
1. Webb, E.K. *Training African pouched rats to save lives: from pup to hero.* Invited talk at Oregon Zoo, Portland, OR, April 2019.

ORAL PRESENTATIONS

6. Webb, E.K., Weis, C., Huggins, A., Bennett, K., Miskovich, T., Krukowski, J., deRoon-Cassini, T., & Larson, C. (upcoming). Neighborhood socioeconomic disadvantage alters neural responding during cognitive and affective tasks: evidence for adaptive processes. *Anxiety & Depression Association of American annual meeting, Denver, CO.*
5. Webb, E.K., Weis, C., Bennett, K., Huggins, A., Parisi, E., Miskovich, T., Kallenbach, M., Krukowski, J., Fitzgerald, J., deRoon-Cassini, T., & Larson, C. (2021). Neural Impact of Neighborhood Disadvantage in Traumatically Injured Adults: A Multi-Modal Investigation. *76th annual meeting of the Society of Biological Psychiatry, virtual.*
4. Webb, E.K., Schneider, M., Lewon, M., Kuipers, D., Cox, C., & Fast, C.D. (2020). *Training the trainer: how behavioral skills training helped African pouched rats sniff out smuggled wildlife.* Animal Behavior Management Alliance conference, Amsterdam, Netherlands. *Cancelled due to COVID-19.*
3. Fast, C., Andrews, R., Webb, K., Brotheridge, S., Kuipers, D., & Cox, C. (2018). *Olfactory discrimination with a humanitarian purpose: African giant pouched rats as detectors of landmines and tuberculosis.* Talk presented by Webb, E.K. at the International Conference on Comparative Cognition, Melbourne Beach, FL.
2. Fast, C.D., Ellis, H., Webb, E.K., Lewon, M., Brotheridge, S., & Cox, C. (2017). *Investigating peak shift in an olfactory discrimination: Lessons for animal scent detection.* Keynote presented by Fast at the Pavlovian Society Annual Meeting, Philadelphia, PA.
1. Fast, C., Sears, E.K., Marnewick, K., & Pires, A. (2017). *Ratting on wildlife crime: Using African giant pouched rats Cricetomys ansorgei to detect wildlife contrabrand in shipping ports.* Talk presented by Marnewick at the SA Management Association Conference, Western Cape, South Africa.

INVITED PANELS

1. Fitzgerald, J.M., Ragozzino, M., & Webb, E.K. (2020). *Cross-species collaborations to advance translational neuroscience.* Pavlovian Society Meeting.

POSTER PRESENTATIONS

19. Webb, E.K., Etter, J.A., & Kwasa, J.A. (2021). *The "unusable" data crisis: origins of racial and phenotypic bias in electrophysiology and a roadmap for inclusion.* International Neuroethics Society Meeting, *virtual*.
18. Liuzzi, M.T., Webb, E.K., Miskovich, T.A., Bennett, K.P., Huggins, A.A., Weis, C.N., Parisi, E.A., Fitzgerald, J.M., Krukowski, J.L., Iseley, J.L., deRoon-Cassini, T.A., & Larson, C.L. (2021). *Changes in resting state functional amygdala connectivity predict posttraumatic stress symptoms in women at a 6-month follow-up*. 55th convention of the Association for Behavioral and Cognitive Therapies (ABCT), virtual.
17. Fitzgerald, J.M.*, Webb, E.K.*, Weis, C., Huggins, A., Bennett, K., Parisi, E., Kallenbach, M., Krukowski, J., deRoon-Cassini, & Larson, C. (2020). *Hippocampus resting-state functional connectivity forecasts individual PTSD symptoms: A data-driven approach*. *(*co-first authors).* 76th meeting of the Society of Biological Psychiatry, virtual.
16. Webb, E.K., Weis, C., Bennett, K., Huggins, A., Parisi, E., Miskovich, T., Kallenbach, M., Krukowski, J., Fitzgerald, J., deRoon-Cassini, T., & Larson, C. (2021). *Neural Impact of Neighborhood Disadvantage in Traumatically Injured Adults: A Multi-Modal Investigation.* Association for Clinical and Translational Sciences, virtual.
15. Huggins, A. A., Fitzgerald, J., Weis, C. N., Hanson, J., Webb, E. K., Bennett, K. P., Parisi, E. A., deRoon-Cassini, T. A., & Larson, C. L. (2021). *Neural activation during fear extinction acutely post-trauma predicts chronic PTSD severity.* 76th meeting of Society of Biological Psychiatry, virtual.
14. Webb, E.K., Broecke B.V., Abraham, L., Schneider, M., & Fast, C.D. (2021). *What makes a good hero great? Evidence for repeatable differences in African giant pouched rat personalities*. Animal Behavior Society Twitter Conference. Conference twitter thread: https://tinyurl.com/AnimBehav2021
13. Weis, C.N*, Webb, E.K.*, Huggins, A.A, Kallenbach, M., Miskovich, T.A., Fitzgerald, J.M, Bennett, K.P, Krukowski, J.L, deRoon-Cassini, T., & Larson, C.L. (2021). *Stability of hippocampal subfield volumes after trauma and relationship to development of PTSD symptoms.* Society for Neuroscience Global Connectome Meeting.
12. Kallenbach, M., Webb, E.K., de-Roon-Cassini, T. & Larson, C.L. (2020). *General self-efficacy as a mediator to gender differences in the development of posttraumatic stress disorder.* International Society for Traumatic Stress Studies. [+]
11. Skrober, A.L.**, Webb, E.K., & Larson, C.L. (2020). *Life stressors predict PTSD symptoms following a traumatic injury.* University of Wisconsin-Milwaukee Undergraduate Research Symposium, *virtual.* (** undergraduate mentee).
10. Webb, E.K., Weis, C., Huggins, A., Hanson., Sellnow, K., deRoon-Cassini, T., & Larson, C. (2020). *Neighborhood disadvantage is associated with smaller amygdala volume and altered amygdala functional activity.* 75th meeting of Society of Biological Psychiatry, *virtual*.
9. Fitzgerald, J.M., Huggins, A.A., Weis, C., Hanson, J., Bennett, K., Parisi, E., Webb, E.K., Larson, C., & deRoon-Cassini, T. (2020). *Differences in Endocannabinoids Relates to Intact Fear Learning After Traumatic Injury.* 75th meeting of Society of Biological Psychiatry, *virtual*.

8. Weis, C., Huggins, A., Fitzgerald, J., Miskovich, T., Bennett, K., Parisi, E., Webb, E.K., deRoon-Cassini, T., & Larson, C. (2020). *Data-driven approach to dynamic resting state functional connectivity in individuals at-risk for PTSD development.* Organization of Human Brain Mapping Meeting, *virtual*.
7. Webb, E.K., Cutright, E., Schneider, M., Mwampashi, R., Cox, C., & Fast, C.D. (2019). *Training African giant pouched rats as biosensors: new humanitarian applications.* Pavlovian Society, Vancouver, BC, Canada.
6. Webb, E.K., Huggins, A., Belleau, E., Taubitz, L., Hanson, J., deRoon-Cassini, T., & Larson, C. (2019). *Periaqueductal gray resting state functional connectivity prospectively predicts PTSD symptom severity.* 74th Society of Biological Psychiatry Annual Meeting, Chicago, IL.
5. Brotheridge, S., Ellis, H., Webb, E.K., Fast, C.D., & Cox, C. (2018). *Discrimination training and odorant concentration influence scent detection performance of Cricetomys ansorgei.* International Conference on Comparative Cognition, Melbourne, FL.
4. Webb, E.K., Saccardo C., Fast, C., & Cox, C. (2018). *Broadening the life-saving repertoire of African giant pouched rats: rapid re-training and maintenance of multiple odor discriminations.* International Conference on Comparative Cognition, Melbourne, FL.
3. Webb, E.K., Kuipers, D., Marnewick, K., Fast, C., & Cox, C. (2018). *Ratting on illegal trade: using African giant pouched rats (Cricetomys ansorgei) to detect contraband wildlife products.* International Conference on Comparative Cognition, Melbourne, FL.
2. Lewon, M., Webb, E.K., Brotheridge, S., & Fast, C.D. (2018). *Behavioral skills training for trainers of scent detection Cricetomys rats in research setting: interactions between trainer, rat, and observer behavior.* Nevada Association for Behavioral Analysis Annual Conference, Reno, NV.
1. Johnson, B.A., Sears, E.K., & Wilson, W.J. (2016). *Behavioral comparison of two methods of drug administration in the earthworm.* Society for Neuroscience annual meeting, San Diego, CA.

Teaching

University of Wisconsin-Milwaukee, Graduate Teaching Assistant

Psychological Statistics, 2018-2019 — Cognitive Neuroscience, 2020
Neuropsychology, 2020 — Experimental Social Psychology, 2021

Editorial Work

2019-20 Associate Editor: *Translational Issues in Psychological Science: Research Ethics*
ongoing Ad-Hoc Reviewer: *Translational Issues in Psychological Science*
ongoing Ad-Hoc Reviewer: *Psychneuroendocrinology*

Non-Academic Work + Notable Service + Media Coverage

2020-22 Psychology Department Diversity Committee, Fellow
2021 Removing racist cops: from implicit bias training to hiring the unbiased brain
www.theneuroethicsblog.com/2021/03/removing-racist-cops-from-implicit-bias.html
2020-22 Cognition, Learning, Attention, and Memory Society, Officer
2019 APOPO's Ambassadors to America
www.apopo.org/en/latest/2019/07/APOPOs-Ambassadors-to-America

2019 'Hero rats' go on display at Tacoma Zoo. Their relatives are still saving lives. http://www.tinyurl.com/y6eszh6x
2018 These giant rats save lives and may soon stop poachers. *Top of the Mind* [BYU Radio]
2018 Saving lives one sniff at a time: Albion alumnae train 'herorats'. [Online, Mar 26].
2014-16 Psi Chi Albion College Chapter President

Awards + Achievements

2020 Finalist, 3 Minute Thesis Competition, University of Wisconsin-Milwaukee
2019 Honorable Mention, International Neuroethics Society Essay Contest
2016 Peter T. Mitchell Lux Fiat Award, Albion College - honors outstanding graduating seniors for leadership and service
2012-16 Webster Scholarship, Albion College - recognizes strong academic performance
2014 1st Place, Gildart Creative Writing Prize, Albion College

Additional Training

2021 CONN Toolbox Workshop, *MGH Martinos Center for Biomedical Imaging*
2020 Network Analyses in R, International Society for Traumatic Stress Studies
2019 University of Michigan's Summer Training Course in fMRI supported by the NIH

Scientific Memberships

Society for Neuroscience (SfN)
Society of Biological Psychiatry (SOBP)
Pavlovian Society
Women in Learning (WiL)
International Neuroethics Society (INS)
American Psychological Association (APA)
International Society of Traumatic Stress Studies (ISTSS)