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Three stories about intergenerational trauma
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Abstract Cover Page

Three stories about intergenerational trauma

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An abstract of
A dissertation submitted to the Faculty of the James T. Laney School of Graduate Studies of Emory University in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Anthropology.
2022

Abstract

Abstract

Three stories about intergenerational trauma
By: Luisa Maria Rivera

This dissertation explores intergenerational trauma, embodiment, and epigenetics from a critical biocultural or biosocial anthropological lens. It is a three-paper dissertation that lays out a progressive vision for re-centering the social in the social epigenetics of intergenerational trauma. Chapter 1 begins with a biosocial conceptual model and systematic review of the relationship between preconception trauma and offspring epigenetic marks. It reveals the limited integration of ecological and social inheritance in this emerging field and suggests that epigenetic approaches have yet to fulfil the promise of precision screening and therapy. In Chapter 2, I present qualitative results of a planned but partially completed biosocial study of intergenerational trauma and embodiment in Nueva Esperanza Chaculá, a community of former refugees of the Guatemalan Civil War and their descendants living in the borderlands of highland Guatemala. I consider how participant subjectivity reveals different pathways by which trauma is transmitted and resisted between mothers and grandmothers. I use those insights to draw attention away from individualized indices of trauma and towards the inheritance of structural violence that links war-time experiences to contemporary inequality and violence in everyday life in 'postwar' Guatemala. In Chapter 3 I implement a study of structural racism, life-course stress, and accelerated epigenetic aging of the placenta in a cohort of mothers and children in Shelby County, TN. I use an intersectional theoretical framework to re-locate intergenerational trauma in social structures rather than individuals. I find no relationships between either structural racism or life course stress and placental epigenetic aging. However, the relationship between structural racism and risk of trauma exposure differed between Black and white women. Whereas increased residential segregation, income, and racialized income inequality buffer white women from life course trauma, none of these predictors are associated with trauma exposure in Black women. I conclude with interrogating the narrative choreography—the stories we tell ourselves and about ourselves—biosocial scientists and social epigeneticists like myself use to rationalize our work and suggest pathways for a more equitable anthropological epigenetics of the future.

Three stories about intergenerational trauma

A dissertation submitted in partial fulfillment of a doctorate in Anthropology at Emory University

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September 27, 2022

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This dissertation is dedicated to my grandmother, Luisa.

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Introduction

This dissertation explores intergenerational trauma, embodiment, and epigenetics from a critical biocultural or biosocial[1] anthropological lens (Leatherman & Goodman 2020). It is a three-paper dissertation—each drawing on different datasets and methods— that lays out a progressive vision for re-centering the social in the social epigenetics of intergenerational trauma. I begin with a view from above a biosocial conceptual model and systematic review of the relationship between preconception trauma and offspring epigenetic marks. Among other challenges, this review reveals the limited integration of ecological and social inheritance in this emerging field and suggests that epigenome-wide and candidate gene approaches have yet to fulfil the promise that they would identify "at-risk" precision phenotypes and/or potential therapeutic targets. In the second chapter, I then take a view from within, presenting qualitative results of a planned but partially completed biosocial study of intergenerational trauma and embodiment in Nueva Esperanza Chaculá, a community of former refugees of the Guatemalan Civil War and their descendants living in the borderlands of highland Guatemala. In this chapter, I consider how anthropological theories of subjectivity reveal different pathways by which trauma is transmitted and resisted between mothers and grandmothers. I use those insights to draw attention away from individualized indices of trauma

[1] These terms are basically interchangeable, but gently implicate different intellectual lineages. Biocultural work is solidly anthropological— it emerged from an attempt to draw together cultural and biological approaches to advance the idea of biology as local, variation as normal, and embodiments as reflecting evolutionarily conserved reaction norms revealed in their distinct expressions across different socioecologies (Armelagos et al. 1992). Biosocial research has a more interdisciplinary flair, including epidemiologists and sociologists who study postindustrial social structures and inequities and their effects on biomarkers and bodies (Goosby et al. 2018). Biocultural anthropologists do not just study exoticized non-Western cultures and hormones. Many, especially those that invoke a 'critical biocultural approach' also study the effects of neoliberal policies and their subjective embodiments much as biosocial scholars do (Sweet 2018). Throughout the dissertation I tend to use "biosocial" because by and large, my scientific collaborators and interlocutors recognize what it means and feel like they are a part of it.

and towards the importance of the inheritance of structural violence that links war-time experiences to contemporary inequality and violence in everyday life in 'postwar' Guatemala (Menjívar 2011).

In the final chapter, I take a view forward, integrating learnings from each of the first two chapters to implement a study of structural racism, life-course stress, and accelerated epigenetic aging of the placenta in a cohort of mothers and children in Shelby County, TN. I respond to the limits of traditional biosocial models of intergenerational transmission explored in Chapter 1 by nesting lived experiences of trauma and offspring epigenetics in a census-tract-level measure of structural racism (Chambers et al 2019). In addition, I use an intersectional theoretical framework and methods to probe the fundamental social causes of trauma exposure and re-locate intergenerational trauma in social structures rather than individuals (Crenshaw 2017). I use an algorithmically derived measure of epigenetic placental aging as my outcome measure to address the limitations posed by using surrogate tissues and candidate gene and/or epigenome-wide methods (Lee et al 2019). However, I find no relationships between either structural racism or life course stress and placental epigenetic aging. Instead, my intersectional analysis reveals that while the epigenetic component did little to shed light on the mechanisms by which structural violence is embodied, the relationship between structural racism and risk of trauma exposure differs between Black and white women. Whereas white women with greater individual wealth, living in wealthier census tracts, or living in more white census tracts experience significantly fewer stressors and traumatic events, Black women are not buffered from trauma by income, concentration of Black or white residents, or Black/white wealth inequality.

Despite the 'progressive' vision for social epigenetics and intergenerational trauma I try to employ, I interrupt each chapter of the dissertation with an autoethnographic vignette of my experiences conducting biosocial science which gave me pause and caused me to question the inherent logics and ethics of my work as a social epigeneticist. I conclude by interrogating the

narrative choreography (Müller & Kenney 2021) —the stories we tell ourselves and about ourselves—biosocial scientists and social epigeneticists like myself use to rationalize our work and suggest partial and potential pathways for a more equitable anthropological epigenetics of the future.

A dissertation disrupted: changes to the research following the COVID-19 pandemic

Like many doctoral students, my dissertation plans were disrupted by the COVID-19 pandemic, which was declared about halfway through my year of fieldwork. I began my biosocial study in September of 2019 and left Guatemala with short notice in March 2020. In the year following, I made the decision not to return to collect more data for my dissertation. Because of this, the dissertation I have written is substantially different than the one I proposed several years ago in ways that were ultimately transformative for myself as a researcher. The cessation of research provided an opportunity for deep reflection about the justifications I used in my original research plans, the different forms of risk that I exposed my research participants to, and the limitations of the core narrative I had constructed as a social epigeneticist interested in advancing human health and equity. In this section of the introduction, I describe my original research plans and the key conceptualizations of embodiment that motivated me.

My original study plan involved a mixed methods biocultural study of intergenerational trauma, cultural models of resilience, epigenetic marks, and hypothalamic-pituitary-adrenal axis activity in survivors and descendants of the Guatemalan civil war. My proposed field site was Nueva Esperanza Chaculá, usually simplified as just Chaculá by its residents, a village of around 2,000 mixed-ethnicity Maya and Ladino Guatemalans living in a community founded by repatriated refugees of Guatemala's 30-year civil conflict and genocide, located in the borderlands of northwest Guatemala (Rousseau et al 2005). In my proposed study design, I planned on conducting in-depth interviews and biospecimen collections using a tri-generational design of grandmothers who survived the war, daughters raised in refugee camps and in the repatriated community, and their

young children aged 3-9. I planned on using locally adapted trauma measures and mental health symptom inventories to quantify the amount, type, and timing of trauma exposure in each generation and its impacts on mental health and child socioemotional development. The main outcome measures in these analyses were in children: socioemotional development, chronic cortisol excretion measured in hair and DNA methylation of genes involved in the stress response, the hypothalamic-pituitary-adrenal axis[2]. To disrupt the deficit-centeredness of my study, I also planned on conducting qualitative coding of interviews to derive local cultural models of resilience and explore whether grandmothers and mothers who employed these models were able to buffer children from socioemotional problems and trauma-related cortisol and epigenetic patterns.[3]

I successfully completed approximately a third of the planned research. During pilot work, I conducted several focus groups and successfully translated and adapted the measures in my trauma and symptom battery as well as workshopped open-ended questions for life history interviews. I lived in Chaculá for 7 months, and along with the 27 mother and 19 grandmother interviews I conducted, I spoke with many community leaders, healthcare workers, and teachers about the history of the community and the challenges families faced.[4] I also witnessed these challenges first-hand as a resident of the community and member of a local multi-generational household. I volunteered as an English teacher and participated in the daily rhythms of household life, taking regular fieldnotes on my experiences. After the pandemic was declared in late March of 2020, I was asked by my university to return immediately to the United States. I understood I might not return for several years. To meaningfully conduct the epigenetic and cortisol related analyses, I would have needed a sample size of at least 50 (but much more likely 100) triads to uncover small to medium effects; what I had collected was insufficiently powered to interpret. My options were to 1) postpone

[2] A full review of the stress response system, HPA, and epigenetic control of its responsivity is provided in Chapter 1.
[3] A description of cultural models is provided later in this introduction.
[4] See Chapter 2 for a full summary of the ethnographic and psychometric work completed.

data collection until after pandemic restrictions were lifted, 2) write a purely qualitative dissertation, or 3) write up qualitative data and conduct biosocial analyses with similar data in a separate dataset.

My reasons for ultimately choosing the third option were complex. Among them were a reconsideration of the ethics of my work. The risks of collecting biological samples during the pandemic made me uneasy, although I did develop a remote protocol, I encountered communication and connection issues when I tried to do them over Zoom. I felt it was wrong to push on and attempt to finish the research given the immediate harms of infection, scarce medical resources and delayed vaccine rollout that Chaculenses lived through while I sat comfortably in the U.S. with access to monoclonal antibodies and ICU care. Feelings that my work was creating harm for others and benefit for myself had been with me for many years. Like most of the graduate students I had trained with, I was critical of the neocolonial relations, power inequality and unequal benefits to researcher vs. research inherent in anthropology (Jobson 2020). In a presentation for the American Anthropological Association's annual meeting in 2018, I gave a paper about my recognizing the discomfort I felt at being someone who would make a career and life for myself by "cutting, extracting, and plotting" the hormones and epigenetics of families living in poverty.

Even before the pandemic, I had come to feel a sense of dissociation when I presented my work among my peers, other biosocial scientists. At times, I would be asked how I planned on addressing genetic susceptibility to trauma or whether I could comment on the possibility of epigenetic pharmacological therapies that my participants might one day use. These questions seemed profoundly out of touch with the potential for stigma and lack of access to even basic medical care I witnessed in the field. The person who presented at conferences, wrote grant proposals, and debated during seminars felt like a different self than the one who sat long and oftentimes intensely painful and emotional interviews, dreading the moment I would gently ask her to finish up her life story so we would have time for me to swab her cheeks and cut her hair. Up

until I left the field, I had negotiated this moral discomfort or ambivalence by constructing what Müller and Kenney (2021) call "a narrative choreography" of my work. The narrative I told myself was that my work could advance health equity by revealing the mechanisms by which structural harm is materially embodied while not essentializing, naturalizing, or stigmatizing the people I worked with. For Müller and Kenney, scientific narrative choreographies are stories about our research, and ourselves as researchers, that we use to make sense of, rationalize, and locate ourselves within our work as biosocial scientists.

Embodiment as social forensics

The core of the story I had told myself was that I was using material embodiment—epigenetic marks, gene expression, neuroendocrine function—as a means of revealing and indicting the harms created by political violence and political-economic inequality. I call this, tentatively, embodiment as social forensics, a kind of uncovering evidence of bodily harm with the goal of implicating the social forces the perpetrate it. It is, in this sense, activist biological anthropology. It has precedent; forensic anthropology has played an important role in post-conflict activist anthropology, particularly in the wake of the Guatemalan civil war and genocide (Torres 2005). Indeed, this activist orientation suffuses several fields of biological anthropology, including critical biocultural approaches. For Leatherman and Hoke (2016), critical biocultural anthropology means an interdisciplinary approach that 'links structures of inequality, constrained agency, and pathways to embodiment within ethnographically grounded local contexts, lived experience realities, and local biologies" (p.287). I interpreted my biocultural approach as 'critical' in that it focused on an 'underserved' population in the Global South that is not typically represented in studies of intergenerational trauma and actively studied the local, embodied effects of state violence.

The term 'embodiment' itself is interpreted many ways, and my own use of it here should be contextualized. For social epidemiologists, embodiment signals a key process in Nancy Krieger's

ecosocial theory of population health, an "idea [that] refers to how we, like any living organism, literally incorporate, biologically, the world in which we live, including our societal and ecological circumstances." (Krieger, 2005, p. 351). For Krieger and epidemiologists like her, although embodiment can take on metaphoric usage, it is always a literal process upon a material body. In many ways, biological anthropologists use the term similarly—embodiment involves the literal inscription of the environment on the body through the life course (Snodgrass, 2016). Forensic anthropology identifies the marks that trauma leaves on bones and bodies that can no longer tell their own stories—and as Krieger tells us, all bodies tell stories. My unique contribution, I told myself, was to bring a critical biocultural anthropological to the emerging field of social epigenetics and intergenerational transmission, an area explored by anthropologists like Lance Gravlee, Connie Mulligan, Chris Kuzawa, Thomas McDade, and Zaneta Thayer. Outside of anthropology, psychiatry and social epidemiology has explored the inter and transgenerational transmission of embodied stress through biological pathways, oftentimes focusing on the regulation of the hypothalamic pituitary axis, the major neuroendocrine pathway implicated in the stress response. Dysregulation of the HPA axis is associated with changes that lead to insulin resistance, cardiovascular illness, anxiety, and depression, all of which are on the rise in rapidly acculturating and structurally precarious communities like Chaculá. This is embodiment as I understood it when I began my doctorate, and I would argue that it is a definition of embodiment shared by many 'biosocial' scientists across different disciplines: social epidemiologists, psychobiologists, health psychologists and more.

Within anthropology more broadly—i.e., sociocultural, psychological and medical anthropology—embodiment entails a different set of associations. One of its origins can be linked to the work of Franz Boas, who famously integrated the body into his 1912 critique of eugenicist theories of race that historically permeated anthropology and human biology. By deploying the tools of the eugenicist trade—craniometrics, statistics, scientific sampling—Boas made a compelling case

for a socially embedded body that was, especially in development, physically plastic in response to its environment (Boas, 1940, 82-85). For Boas, ethnography and biology played complementary roles; he was willing to draw on either as forms of evidence to make his case against the "shackles of tradition"— discrimination and oppression based on pseudo scientifically produced ideas of race and ethnicity that could be scientifically extinguished. Boasian embodiment-as-social-forensics sought to contest scientific racism and biological essentialism that characterized the rise of anthropology as an academic discipline. The fissures within anthropology in the post-Boasian era would cause material approaches to embodiment to recede after Kroeber would lower what Lock calls "the black box" over the material body in his 1917 essay The Superorganic (Lock, 2013).

The post-structural turn in anthropology and subsequent problematization of knowledge production and authorial intent strengthened this division (Clifford & Marcus, 1986). That post-structural turn is evident in Csordas' (1993) definition of embodiment in parallel to Roland Barthes' differentiation between written "works" and written "texts"; while the work is a material artifact, the "text" arises as a methodological field only through discourse. For Csordas, "the body is a biological, material entity, while embodiment can be understood as an indeterminate methodological field defined by perceptual experience and the mode of presence and engagement in the word." (Csordas, 1993, p. 135). The subsequent historical and post-structural lenses brought by Foucault and Bourdieu went on to influence the anthropology of biomedicine in the tradition of Margaret Lock and Nancy Scheper-Hughes, whose work continues to be paradigmatic for notions of embodiment within sociocultural anthropology. Unlike Csordas, for whom embodiment functions as an abiologic 'problematic' or discourse, Lock and Scheper-Hughes (1987) call for the return of the physical body within anthropology as "both naturally and culturally produced, and as securely anchored in a particular historical moment" (p.8). Noting that the divide between consciousness and corporeality

is itself a socially produced distinction within Western metaphysics, they assert the possibility of holism between flesh, memory, and emotion in meaningful accounts of socially situated bodies.

The rapprochement between the material and political body—and the ability of genetic and biological anthropology to contest scientific racism, sexism, and biological essentialism—remains tenuous (Livingstone 1971; Livingstone & Dobzhansky 1962; Baker 2021). However, recent cross-disciplinary engagements within anthropology and other biosocial sciences have explored the social forensics of embodiment, especially within the context of social epigenetics and intergenerational trauma. I will begin by briefly reviewing the history of the term 'epigenetics' and its implications for intergenerational transmission and social theory below.

Social epigenetics meets social science

The term 'epigenetics' was first used by developmental biologist Conrad Waddington in 1942 (Waddington, 1942a). Waddington was interested in the static, predictable qualities of cell differentiation in the embryo as well as dynamic responses to environmental stimuli. He coined the term epigenetics to describe developmental plasticity in response to environmental differences, highlighting the idea that the same genes can produce different phenotypes under different conditions. Simultaneously, he described processes of canalization—the stability of phenotypes across different genotypes and environments (Waddington, 1942b; Waddington, 1957). Epigenesis as imagined by Waddington implied multiple, complex, and interacting pathways to expression of the genome in response to environmental cues, the biochemistry of which would be elucidated by the researchers who followed him. He theorized that traits that were not heavily canalized would demonstrate a normally distributed range of plasticity in response to environmental insults, and that such changes in a developmental plan could be heritable across generations. He demonstrated this compellingly in Drosophila, exposing pupae to heat-shock to produce a vein-less wing phenotype which he then selectively bred; after twenty generations of breeding, the heat-shock phenotype arose

without the environmental stimuli. This theory of genetic assimilation was attacked as Lamarckism and perceived as an attack on the Neo-Darwinist synthesis in biology (Noble, 2015).

Later work by David Nanney emphasized the variability of genetic expression mechanisms, as well as their persistence across mitosis (Nanney, 1958). Advances in chemical genetics pushed research in this area during the 1970's, with several research groups independently suggesting DNA methylation as a key mechanism of gene expression. Holliday and Pugh (1975) demonstrated that DNA methylation was heritable across somatic cell populations following mitosis. Meanwhile, Griffith & Mahler (1969) hypothesized that literal long-term human memory might be a function of persistent DNA methylation in brain cells. While the attachment of methyl groups to cytosine nucleotides in genomic DNA (e.g., DNA methylation) was the earliest and remains the most studied form of epigenetic control of gene expression, a proliferation of research in molecular biology through 1990's revealed additional epigenetic mechanisms including changes in the arrangement of chromatin and non-coding RNA.

Lanedecker and Panofsky (2013) trace the rise of a particularly social epigenetics that emerged from biomedical environmental health research streams. These were in turn guided by the field's original focus on developmental processes and critical or sensitive periods during the lifespan during which environmental insults might disproportionately affect adult health, the developmental origins of health and disease or DOHaD hypothesis (Hertzman & Boyce, 2010). Social epidemiology and public health research had already established the salience of prenatal and early-life experience on later adult health outcomes such as obesity, cardiovascular disease, mental illness, and substance abuse (Dube et al 2003; Anda et al 2006). The age of the epigenome promised new insights into the proximal mechanisms of the biological embedding of social experience already demonstrated in large, population-based studies.

The intergenerational transmission of stressful social experiences or suffering were key elements of the series of seminal papers that shaped the field of social epigenetics as we know it today. The canonical reference in this literature by Weaver and colleagues (2004), who found differential DNA methylation of the promoter region of the gene that encodes the glucocorticoid receptor (NRC31) of rat pups that were neglected or given suboptimal care by their mothers. These changes were conserved into adulthood and reversible with cross-fostering; pups that that received less maternal grooming produced fewer receptors for glucocorticoids and demonstrated anxious behaviors. When reared by more attentive mothers, the epigenetic and behavioral signal was restored to the 'healthy' baseline. Subsequent work by Moshe Szyf and Michael Meaney replicated and elaborated on these findings, resulting in a "molecular conduit" model of early life social experience and the long-term programming of multiple genes implicated in the stress response (Landecker & Panofsky, 2013; Weaver et al 2006).

Landecker and Panofsky (2013) note (emphasis mine): "The molecular conduit model is important to understand. It is the argument for how behavior is embodied in molecules that themselves go on to pattern behavior in the future; in other words, the conduit goes into the body (from behavior to gene methylation), but it also runs back out again (from gene methylation to behavior)" (p. 335). This molecular conduit view of embodiment was subsequently tested in the postmortem brains of suicide victims. Those who experienced childhood trauma, like the rat model, demonstrated more methylated NR3C1 promoter regions and decreased NR3C1 expression as compared with non-abused suicide victims and non-suicide controls (McGowan et al 2009). Moving from the brain to the periphery, Oberlander and colleagues (2008) demonstrated that the cord blood of neonates born to depressed and anxious mothers showed lower NR3C1 expression with higher methylation of NR3C1. When gently stressed themselves, babies with this pattern of NR3C1 methylation mounted dysregulated stress responses as measured through their salivary cortisol, a

proxy measure for the functioning of their hypothalamic pituitary axis. Four years after Weaver et al (2004), social epigenetics had emerged as a nexus for ongoing discourses about the embodiment of experience and the nature/nurture dialectic and biosocial inheritance.

Perhaps because of this orientation, Lock and other social theorists have turned significant attention to the anthropology of embodiment in the epigenomic era (Lock, 2013; Lock 2015; Meloni, 2015; Niewöhner, 2011; Pickersgill et al 2013). For such theorists, epigenetic research provides both an intriguing opportunity to legitimize the importance of both history and subjectivity as well as a potentially dangerously reductionist mode of inquiry into social life (Lock, 2015; Landecker & Panofsky, 2013). Maurizio Meloni (2015) views the rise of this new (and potentially "overhyped") science as having profound implications for political notions of justice. He argues that previous sociobiological constructions of innate and predetermined differences were not subject to the interventions of justice or morality. One either won the genetic lottery or not, and society was under limited obligations to restructure such a 'natural' meritocracy. Increasing evidence for epigenetic embedding and reproduction of social inequality leads him to question, "where are the boundaries between personal and collective responsibility in a context where social factors seem so massively engaged in producing aspects of our own individual biology?" (p. 134).

Lock suggests both her term, 'local biologies' and Niewöner's 'embedded bodies' as helpful frameworks for making meaning of social epigenetics. In her ethnography of menopause (or rather, its absence) in Japan, Lock suggested the notion of 'local biology' to help move sociocultural anthropology to recognize that quantifiable, hormonal variation accompanied the different experiences and medicalization of menopause for Japanese women. Her interpretation of the 'embedded body' concept, however, is that it carries a more critical view of how social lives and histories are operationalized by social epigeneticists (Niewohner, 2011; Lock, 2001; Lock, 2013). Meloni also highlights a similar tradition of critiquing the 'primacy of the gene' in sociocultural

constructions of the body. Thus, along with providing a causal mechanism explaining why similar genes produce different phenotypes, the concept of materially embodied trauma due to social harms can do important cultural work for both the researchers who conduct these studies and the audiences that consume them (Lock, 2017; Mol, 2002; Müller, 2020; Warin & Hammarström, 2018).

Anthropology of embodied intergenerational trauma

Western psychiatric models of trauma and embodiment place trauma in the mind of the individual and search for its imprint in the body. The etymology of trauma is from the Greek trauma or literal "wound"; psychiatric paradigms of post-traumatic stress are rooted in the idea that some of us are vulnerable to impaired ability to recover after experiencing life or self-threatening events, a sort of impaired psychic wound healing (Pillen, 2016). Western psychiatry locates the etiology of such impaired wound healing in disruptions of neurological fear conditioning (Hinton & Good, 2015). Improper consolidation of the memories associated with terrifying, threatening, or overwhelming experiences are in turn thought to cause the symptoms used to identify post-traumatic stress disorder (PTSD) with the current DSM-5 diagnostic criteria: re-experiencing traumatic events, hypervigilance, negative mood and cognition, and hyperarousal long after immediate threats have passed (Hinton & Lewis-Fernández, 2011; Kirmayer et al., 2007). A large body of research in psychiatry and neuroscience advances the embodiment of trauma as a material condition, seeking to elucidate biological mechanisms that drive PTSD risk and symptomatology, such as the blunting of cortisol reactivity, neuroinflammation, or altered amygdala functional connectivity (Agorastos & Chrousos, 2022; Clausen et al., 2017).

Anthropologists from multiple subdisciplines have interrogated the universalizing assumptions embedded in Western models of trauma and post-traumatic stress. Rather than a universal experience of impaired wound healing, their work has shown that post-traumatic symptom constellations, idioms of distress, subjective bodily experiences, and neurobiological correlates vary

demonstrably across cultures (Hinton & Good, 2015; Hinton & Lewis-Fernández, 2010; Kohrt & Hruschka, 2010; Rechtman, 2000; Schechter, 2010). In addition, feminist scholars and cultural anthropologists highlight that psychiatric frameworks of PTSD pathologize normative responses to ongoing precarity and structural violence, export neoliberal notions of inherent individual vulnerability to suffering and responsibility for healing and support technocratic psychological 'solutions' to human suffering rather than address its root causes (Fassin & Rechtman, 2009; Müller & Kenney, 2021; Ruíz, 2020; Traverso & Broderick, 2010). In her critique, psychological anthropologist Rebecca Lester suggests that rather than a universal failure to heal after life-threatening events, an anthropological perspective of trauma redefines it as the experience of radical rupture from the socially constructed expectations for what the self can endure. In her analysis, pathways to healing must always involve a 'retethering' to society that re-accommodates the self along with their lived experience of the unthinkable (Lester, 2013). An interpretation of trauma as an evolved process of emotional distress following the rupture of local social norms— a form of profound moral injury rather than a failure of fear conditioning—shifts the question from 'which bodies are vulnerable?' to 'what social conditions create moral injury, to whom, and where?' (Luhrmann, 2006; Zefferman & Mathew, 2020).

Unlike the study of trauma writ large, intergenerational trauma has conceptual links to the generational re-perpetuation of social inequality and sociocultural erasure through its relationship to historical trauma concepts (Denham, 2008; Heart & Horse, 2000; Kirmayer et al., 2014). Indigenous, Black, and Latinx studies have leveraged intergenerational trauma concepts in their exploration of how people experience legacies of cultural loss, displacement, enslavement, and mass trauma (Bombay, 2009; Graff 2014; Ruiz 2020. Despite this, biosocial approaches to intergenerational trauma still tend to localize it within individual biology and behavior; for example, in our recent systematic review, we found that studies of intergenerational trauma in Latinx populations failed to

account for structural inequalities and the fundamental causes of intergenerational experiences of violence, loss, and maltreatment (Cerdeña et al., 2021). I include Figure 1. below for an overview of how quantitative research on Latinx intergenerational tends to conceptualize it and the mechanisms and moderators of its transmission.

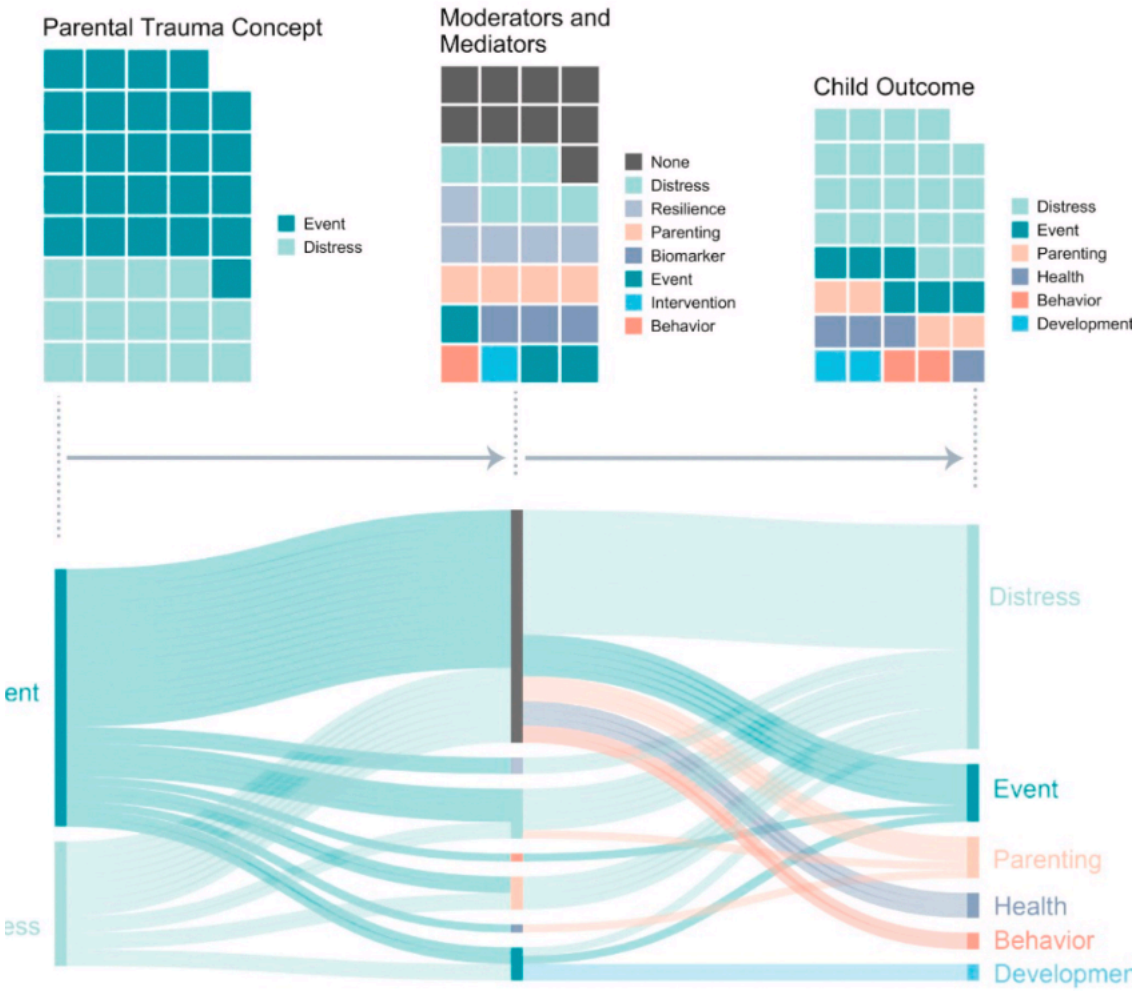

Figure 1. Intergenerational trauma concepts, moderators, and mechanisms in Latinx populations. Adapted from Figure 4b of Cerdeña, Rivera & Spak 2020

The perception that intergenerational trauma is embodied as a biological process in which parental experiences epigenetically program poor outcomes in offspring has increasingly entered academic, public health, education, and lay discourse in the last ten years (Dubois et al., 2020; Lappé, 2016; Romijn & Louvel, 2021). As I review in Chapter 1, many researchers rationalize

neuroendocrine and biomarker research on the biological imprint of trauma as a means of developing precision screening and treatment. But a second rationale, more political, is grounded in social forensics: material embodiments of trauma provide objective evidence of enduring harm. "Elucidating biological mechanisms" may serve to provide avenues for precision care, but I argue a hidden logic animating our research is that they absolve parents and communities of blame. Much like the belief that the 'chemical imbalance' model of mental illness reduces stigma, so does a "stress under the skin" model absolve trauma exposed families from being labeled as behaviorally or constitutionally pathological (Shonkoff 2012). By elucidating the mechanisms of the biological embedding of toxic stress, we aim to render invisible psychic wounds real, measurable, and legible across cultures in the language of biology. In our contemporary hierarchies of knowledge, the biological is ranked more highly than testimony, oral history, or witnessing. When that biology is tied to social conditions, we suggest our work can provide proof that the pain of wounding expressed in testimonies past and present is not just subjectively experienced, but concretely true (Warin et al 2020).

Social forensics as a failed theory of social change

In context, I can understand why I might have adopted this problematic view. Some of the most influential studies of intergenerational trauma and epigenetics were being published as I began my doctorate—in retrospect, it was the peak of a particular 'biomarker hype' curve. In the fall of 2016, Rachel Yehuda's group published a paper finding that children of Holocaust survivors had less methylation of FKBP5, a gene that encodes a chaperone protein for the glucocorticoid receptor, a potential epigenetic mechanism underlying the blunted cortisol reactivity seen in survivors of early life adversity (Yehuda et al 2016, Gunnar et al 2018). That same year, Kertes and colleagues (2016) published their work examining culturally salient war-time stressors and newborn glucocorticoid receptor methylation in the Democratic Republic of Congo. Two years earlier, Brian Dias and Kerry

Ressler had found evidence that paternal rat exposure to painful stimuli and an olfactory cue produced epigenetic, morphological, and behavioral changes their unexposed offspring (Dias & Ressler 2014). While Palma-Gudiel and colleagues (2014) had published a critical review of the replication failures of prenatal stress and infant NR3C1 DNA methylation research around the same time, many of the collaborators I knew and admired were deeply invested in leveraging DNA methylation microarrays in large cohorts to finally elucidate the mechanisms underlying the intergenerational impact of traumatic stress and adversity.

Epigenetic discourse had also begun to circulate among my non-scientist friends and among the social scientists I knew, as well as popular and social media. But just as the idea that epigenetics 'explained' why genetic determinism was wrong began to pick up speed, methodological critiques within social epigenetic communities began to mount. Numerous studies demonstrated that a fair amount of DNA methylation was under direct genetic control (Husquin et al 2018; Hanon et al 2018). Large, well-conducted, and deeply phenotyped cohort studies failed to replicate widely cited previous findings (Marzi et al 2018). This strange seesaw unmoored me: what did it mean to 'validate' trauma through the body with such unstable signatures? Another way of putting it—what does it mean to make a career off an imprecise biomarker in order to make a point about injustice? If your biomarker fails, does your justice fail? I found myself anxious about every possible outcome of my work. What if I had significant hits that I felt little confidence in? What if I had no significant hits, and had to explain that this did not invalidate the lived suffering of the people who agreed to share their stories and bodies with me? What if I had significant hits that I had good confidence in, and that could be used to profile, surveil, and control people like my participants?

These questions disturbed me, especially as I found my grant applications successful and a pathway opening before me for academic success. I am ashamed that it took a crisis and forced cessation of work for me to decide to stop the research, but it is the truth. By the time I got to

Chaculá, I was aware that the cortisol and epigenetic assays offered little benefit to the community—and potentially society at large. Epigenetic marks are not like bone fractures or DNA left at a crime scene. They vary over circadian rhythms, across tissues and cell lines. They cannot give voice to the voiceless—and of course, my participants were not voiceless. Locking away the saliva and hair in a little hand-made cabinet and leaving it behind me as I left in March 2020, I came fully into the realization that it did not matter whether the study 'worked' or not, the core narrative of my work as social forensics was fundamentally flawed.

In her widely cited open letter "Suspending Damage: A Letter to Communities", Eve Tuck names this flawed theory of social change at the heart of research highlights social and health disparities in the hope of seeking reparation (Tuck, 2009).

> This theory of change, testifying to damage so that persecutors will be forced to be accountable, is extremely popular in social science research—so popular that it serves as a default theory of change, so ubiquitous that folks might think that it is entirely what social science is about (p. 414).

Rather than implicating structures of power and bringing about accountability, Tuck argues that "damage-centered" research in Indigenous and other historically oppressed communities primarily serves to naturalize and essentialize that Indigenous bodies and futures are hopeless or ruined. Calling for a moratorium on damage-centered research, Tuck argues that work should center ways in which subjectivities come to form, articulate, and enact their desires amid systems of inequality. Such approaches, she argues, serve to re-humanize Indigenous lifeworlds and imbue them with hopeful agency and self-determination.

Similar discourses have emerged within the anthropology of violence and hope. The ontological or reflexive turn of post structural anthropology of the 1980's faced criticism from within the discipline for being indulgent, cynical, or even destructive (Ortner, 2016; Vigh & Sausdal,

2014). The medical anthropology of the 1990's and early 2000's responded with theories of engaged or activist anthropology that sought to give evidence of suffering produced by social conditions and demand accountability (Good et al., 2010). In turn, new criticisms of the value of this approach, its tendency to depict its subjects as damaged, and the complicated relationship between anthropologists who make careers by 'giving voice' to communities who might voice their concerns differently have emerged from within anthropology as well as from without (Jobson, 2020; Ortner, 2016). Anthropologies of 'the good'—those that explore the experience of hope, empathy, care, and value have risen in tandem with calls for studying the 'resilience' of trauma-affected communities (Denham, 2008; Robbins, 2013).

I was drawn to including resilience perspectives in my work precisely because of this reason, but I found that like epigenetics, a focus on resilience was not without its own problems. Definitions of resilience within psychology are usually conceptualized as properties of the individual; in one sense they refer to the ability to return to a normative baseline quickly after stress exposure, in another they refer to the ability to not be disrupted from that baseline in the first place, and in a practical sense they oftentimes just refer to subjects with an adverse exposure but not a bad outcome (Cutter, 2016). Research on 'grittiness' and 'resilience' can open the door for interventions to maximize resilience as opposed to public policy to address underlying social conditions.[5] Just as deficit narratives essentialize the damage and vulnerability of certain bodies, neoliberal, individualized concepts of resilience may serve to identify ideal subjects able to withstand adversity without becoming burdensome to society. While I had originally planned on coding 'resilience' in the study interviews using a cultural models approach to find potential moderators of embodied

[5] A couple of studies to note here for those interested. A recent RCT showing mindfulness interventions in early adolescents *decreased* mental wellbeing (Montero-Marin et al., 2022), and the failure of several "growth mindset" interventions meant to foster gritty intrinsic and extrinsic motivation when deployed in low SES contexts (Yeager et al., 2019).

trauma, I ultimately decided to analyze the qualitative data in my study with a related—but as I describe below, potentially less damage-centered—framework of subjectivity.

Cultural models and intergenerational subjectivity

As noted earlier in this introduction, my original research plan also used the framework of cultural models of resilience and caregiving to explore local forms of buffering in Chaculá. A cultural model is a form of shared cultural knowledge that simplifies sensory and subjective experience and allows individuals to effectively function in society and navigate their sociospatial worlds (Herring 1987; Shore 1991). Biocultural anthropologists elicit cultural models using systematic methods, such as surveys, interviews, pile sorts and cultural consensus and consonance analysis (Dressler et al 2005). In response to the ethnocentric standards of stress, wellness, and health in Western biomedicine, biocultural anthropology sought to localize expectations (and disruptions) for the self by identifying how an individual's knowledge of and ability to adhere to their local cultural models shapes the subjective experience of stress or suffering. Indeed, the ability to enact a cultural model of wellbeing or social equality has been shown to predict mental and physical health, hypertension, HPA activity, and immune function (Decker, Flinn, England, & Worthman, 2003; Dressler, Balieiro, & dos Santos, 2017; Dressler, Balieiro, Ribeiro, & dos Santos, 2016; McDade, 2002; Read-Wahidi & DeCaro, 2017).

I was drawn to cultural models as a framework for understanding resilience because of its contestation of ethnocentric parenting or child maltreatment norms (Keller 2018; Kohrt 2016; Korbin 2003). But the quantitative identification of a cultural model and categorization of individuals as congruent and/or consonant with them required a kind of approach to data collection (and qualities of the data) that I found challenging to implement in a way that felt respectful of and sensitive to the emotional toll that conducting the trauma inventories took during interviews. In total, I asked women almost seventy different items about traumatic experience they had

experienced. Many spoke for long periods of time to process and contextualize their response to one question, and the urge to move on to the next question—and then the next—felt profoundly insensitive to their need to tell their story on their own terms. It is true that I could have developed a cultural consensus and consonance measure and implemented it, but it is also true that I did not see the value in doing so given the participant burden I had already placed on the Chaculenses.

Another limitation to the implementation of cultural models that became apparent to me were the relatively invariant ways in which women provided care for infants and young children. In my proposal, I suggested that local childcare norms might function as buffers of intergenerational trauma given their ability to support healthy infant attachment (McKenna & Gettler 2016). Extended breastfeeding through the first year of life was the default choice for infant feeding, except for a few women who were physically unable to breastfeed. Similarly, co-sleeping, infant-carrying, care from alloparents, and extended interactions and play with near aged peers was the norm across all families. In those areas that were variable, I felt conflicted about the stigmatization inherent in classifying individuals as unable to enact it. It is true that a general cultural model of appropriate child development might have been elicited with few surprises; many of my participants espoused children's need for a stable family life, non-excessive corporal punishment, access to education and opportunity, sufficient nutrition, and good moral development with a focus on being prosocial and obedient, but spending hours with women who wept as they explained that they were failing their children in these ways made me doubt that classifying them as such would somehow be the answer to 'damage-centered' research critiques.

Instead, I chose to analyze their interviews with a thematic analysis that used anthropological theories of subjectivity to explore the shared and individual processes by which mothers and grandmothers made sense of their experiences and processed their desires for themselves and the ones they loved. Subjectivity refers to the inner emotional life, constructions of the self, and self-

world relations experienced by political subjects (Holland and Leander 2004). Subjectivity is structured by power, history, and culture, but is individually experienced in the body and mind (Luhrmann 2006). Cultural models and subjectivity are entwined concepts; one way I think of subjectivity is the view from the driver's seat as one navigates a culturally constructed model, a labyrinth of meaning. An advantage to focusing on first-person subjectivity is its flexibility—no one model need be shared or not shared, and individual, creative, and spontaneous re-formations of extant models are possible within the same narrative. Disadvantages are, perhaps, a perception that such an approach is less empirical, lacks rigor, or fails to make falsifiable hypotheses. In Chapter 2, I engage subjectivity to explore how individual appraisals of where one is in the labyrinth of culturally shaped expectations for motherhood and womanhood return agency and dignity to my interlocutors and locate trauma in structures, not individuals.

Taking the third option

Given my concerns about damage-centered research and my reflection that my personal narrative of biosocial science as social forensics was problematic, you may ask why I chose to not write a purely qualitative and/or theoretical dissertation. This decision comes from my own desire to enact my own version of critical biocultural anthropology, which engages through practice the issues of biopower, knowledge hierarchies, different ways of knowing, and decolonial approaches to embodiment that I have struggled with navigating throughout my doctorate. More colloquially, I would say that I think it is important to have skin in the game, to know how the sausage is made to intimately participate in biosocial scientific practice and logics—if I am every to say anything meaningful about it. I do not argue this is the case for everyone, but to me it seemed like the path worth taking. I see value in social epigeneticists engaging social theory, reflecting on our shared scientific subjectivities and self-narratives, and allowing those insights to transform our science. I

have committed myself (at least for this dissertation) to engaging these ideas actively as an observant-participant rather than outside observer.

In Chapter 3, I chose to work with data from the CANDLE cohort for practical and theoretical reasons. I knew that I would not collect more data from Chaculá, but still wanted to attempt to implement critical biosocial epigenetic study and explore its assumptions and implications. Practically, I had worked closely with the CANDLE research team and principal investigators on different projects for several years, and their ongoing mentorship and support of my goals was central to my ability to work with the data. Theoretically, I wished to shift to working with a tissue in which causal inferences about epigenetics and physiology were less shaky than surrogate tissues and neuroendocrine function. I also wanted to work with data that would allow me to implement a non-individualized measure of structural violence. Finally, I was interested in working with data from the United States, where I would have greater ability to frame the health policy and clinical implications of the work relative to my marginal influence on Guatemalan mental health services.

Chapter overview and structure of the dissertation

Each paper in this dissertation serves twin narrative functions. The first is to show (not tell) that I have sufficient content expertise and methodological skill to practice as a biocultural anthropologist. The second is to build a progressive vision for biosocial approaches in studies of intergenerational trauma. Each chapter, in its own genre, sets the rising action for the next, culminating in an anti-racist, interdisciplinary, biosocial resolution. This meta-structure illustrates my "narrative choreography", a kind of carefully sequenced dance of knowledge claims that biosocial researchers use to emphasize the value of their work while avoiding "the determinism and pathologization that are often associated with biological explanations for behavior" (Muller & Kenney, p. 1256).

In my first chapter, I take a view from above. I conduct a systematic review and synthesis of empirical research examining the impact of trauma experience prior to conception (whether in a parent or grandparent) and offspring epigenetic changes. Public and political awareness of the impact of ancestral traumas on the stress-related biology of offspring had exploded in the post-epigenomic era; the science of epigenetics has provided a mechanism for the intergenerational transmission of trauma that media, policy makers, activists, and social media content creators have up taken with phenomenal zeal (Richardson 2021; Warin et al. 2020; Yehuda et al. 2018). In this paper I sketch an integrative biosocial model of the nested mechanisms of transmission at the germline, in the maternal soma, in the caregiving environment, and in macro-social structure. I then systematically review and synthesize the extant literature on preconception trauma and offspring epigenetics, finding little evidence for replicable signatures or effects. In addition, I review the conceptual frameworks, rationales, and implications that study authors lay out in their research studies. I find that authors suggested their work might lead to precision screening and treatment of "at-risk" children, and that it also might serve as justification for improved social policies. Despite this, very little replicable evidence of a durable epigenetic signature of preconception trauma emerged from my review. As other scholars before me, I note the problematics of research that localizes trauma transmission in the (mostly) the maternal body and maternal care and call for deeper attention to the re-perpetuation of adversity across generations via structural violence.

The inattention to the social inheritance of trauma— as opposed to behavioral, psychological, or biological—motivates the second chapter, a view from within. In this chapter I draw on the qualitative and ethnographic data gathered in my study of trauma and resilience in a community of repatriated refugees and descendants living in the borderlands of northwest Guatemala. I explore how generational experiences of trauma lived in intimate relationships are embedded in the neo-colonial structures that shape the structural vulnerability of everyday life. I

explore the contributions of anthropological theories of subjectivity to understanding intergenerational trauma. My thematic analysis brings ethnographic and psychometric data into conversation to show the social, cultural, and personal resources women employ to resist violence and construct the lifeworlds they desire for themselves and their families.

In Chapter 3, I take a view forward. Building on the insights from Chapter 1 and 2, I attempt to implement a critical biosocial study of epigenetic programming of the placenta and structural violence in the CANDLE cohort. The CANDLE cohort is a population representative sample of women and children linking maternal life-course trauma to a measure of structural racism and exploring its embodiment in the epigenetics of the placenta. Drawing on intersectional theory and methods from social epidemiology, I use the Index of Concentration at the Extremes to examine how structural racism shapes Black and white women's experiences of life-course trauma, and how those are in turn associated with epigenetic accelerated aging of the placenta (Chambers et al 2019). I employ Arline Geronimus' concept of "weathering' to suggest that accelerated aging of the placenta might be an indicator of the excess immunological and stress physiology related demands made of Black women's bodies due to the interaction of structural racism and life-course stress (Geronimus 1992).

In between each chapter, I offer a series of vignettes that trace key moments of doubt or reflection that helped raise my critical consciousness as a scientist and awakened me to the limitations of the theories of social change and ethical implications of my work. While these vignettes are presented with relatively little analysis, they are meant to mark key developmental milestones in my own experience of transformation throughout the doctorate.

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Interlude 1

True Story

From the beginning, we are trained to tell stories. Whether humanistic or empirical, we coerce a data frame of observation, theory, and power into the story we think we ought to tell about the world. We murmur our stories at cocktail hours or write them up and let them go, to be diffused through seminar rooms and Twitter for consumption and dissemination. The insight— that science is culture— is trite in contemporary anthropology, but some of the best insights we have are. Nature papers are not so different from fables and sometimes they too become myths. In my culture— the culture of developmental psychobiology— scientific stories have a plot, a twist, and probably a moral. Yes, Reader, I know that you know I majored in literature. Does it surprise you that I was first trained explicitly in this craft in a psychobiology lab? How many times in lab meeting did I hear the PI say, not without contempt, "Where is the story? There is no story in this paper."

This dissertation is a long story about intergenerational trauma, embodiment, and scientific culture. In it, I am going to tell you the story of the story. I will not do this instead of what I am supposed to do. That is, I will indeed tell you three stories— three chapters— each conforming to the narrative conventions of their literatures. But I am also going to tell you the true story, the story of stories. I am going to trace the developmental trajectory of the thesis, the way it was gestated, birthed, and grown up— and aged. In developmental science, we mark time with milestones. As we move along the story, I will tell you about the milestones I achieved, the moments that marked a transition for me as a scholar. At first, I thought I'd use a different metaphor for these stories—

sentinel events. Sentinel events are catastrophic events— usually a patient death— in hospital settings that indicate deep failures across multiple systematic layers of protection. But while sentinel events are meant to provoke repair, they do not in and of themselves imply transformation. They just tell you that the system is rotting beneath your feet. I don't think that metaphor captures the moral of my story. I am not writing this story to show you that science has broken down. I am writing this story to help myself decide what I want to make of it.

Sometimes cultural anthropologists call something a method, and I don't know what they mean. I have a behavioral science generalist's notion of methods; they are probably congruent with Russ Bernard's. The story of stories might better be called autoethnography, a method that involves writing about one's lived experience and analyzing those narratives as a means of addressing a research question. But this isn't really what I am doing. The methodological aspect is not that I might use my experiences to better understand the implicit logics of the science of intergenerational trauma and biology.

This is more like archeology in the Foucauldian sense. How do I know which moments were milestones? I knew them before I ever sat down to write this introduction. These are the stories I have already told many times. I tell them when I try to explain to people the nature of my doubt, my ambivalence, my concern that the scaffolding might be breaking down beneath my feet. They are moments that projected me forward into different stages of scholarly life.

They are also my method in another way, in a way that is hopeful. They are a method for finding my way forward. A sentinel event sounds an alarm, warns you that the ground is about to collapse. But a milestone signals a change. Milestones are teleological. I am hopeful that, by telling you this story, I find out how it ends.

The Father

Early in this process, in the second year of my doctorate, I helped organize an annual visit to Chaculá through the nonprofit that had brought me there, KGAP. A portion of KGAP's mission was to support maternal and child health, and we ran annual traditional birth attendant trainings for the birth attendants and nurses who cared for birthing parents in the village and surrounding region. This year, we had planned a larger, more expansive multi-day training held outside of Chaculá in the nearby village of Yalambojoch, hosted at the beautiful school run by Colach Nac Luum, a Swedish NGO. I had heard about el sueco and his school for years; there was a tension between KGAP— which emphasized funding the local public school— and Colach Nac Luum's beautifully designed and maintained private school, run more as a cooperative with external NGO funding. It sat, quite literally, on top of a hill, overlooking the dusty and modestly appointed public school below.

We decided on Colach Nac Luum for practical and cultural reasons. It was big and had a large dormitory that we could all share overnight between the trainings. It was also located in the home village of Petrona, the Chuj midwife who would lead the training. Previous trainings in Chaculá had been led by the nurse-midwives there, Blandina, Juana, and promotores de salud Chepe and Natalio, none of whom spoke the language of the ethnic majority Chuj people, and none of whom wore traditional dress. Petrona was both highly trained as a nurse and traditional birth attendant, committed to her Maya identity and expression of her Chuj culture, language, and religion. The goal of this training was to foster stronger connections between Maya midwives of the

Nentón region and reach senior midwives from further regions who could help diffuse knowledge into their apprentice communities. It seemed right to do it in Yalambojoch.

The American nurse-midwives were trained in pedagogical techniques for basic emergency obstetric and newborn care but spoke halting Spanish and no Chuj. I was an adequately skilled Spanish-English medical interpreter with specialist knowledge in midwifery, but no Chuj language skills. Petrona was native bilingual Chuj and Spanish speaker. Among the four of us, we spent days leading the forty-odd midwives in trainings emphasizing neonatal resuscitation, the prevention and treatment of postpartum hemorrhage, and coordination of transfer for complications. The work was physical (we used a birth and neonate simulator [2] to demonstrate and test knowledge) and tiring, and the communication challenging at best. After three long days simulating uterine rupture, non-responsive neonates, and splashing red food-coloring tinged "blood" over the floor, we packed up our dolls and headed back to Chaculá.

A man I call The Father was with us through the visit. He had come as one of the human rights delegation members for KGAP, which through Witness for Peace continued to fundraise through different churches in the Midwest, including The Father's, a Methodist congregation from Wisconsin. The five delegation members were all progressive, white, and over 65. The Father came because of a deep interest in Guatemala that emerged from his adoption of a Guatemalan infant girl some twenty years before. He spoke little Spanish but brought games of Jax and candy to offer the children in the village. He claimed that this was the 'secret' to connecting with the community and had been on more than ten such trips in the past.

The Father was a foil for many feelings each of us may have had about human rights, global health, and the colonial attitudes nested therein. That is clear. Relaxing in the evening after the final training, he mused that he felt that he too could deliver a baby, having observed the trainings and helped register participant names.

But much as The Father bothered us with his candy, his games, his self-assurance, I think back on him now as one of the most important people I have encountered in my work in Chaculá. I think that, in many ways, The Father helped me see what I was doing without the layers of approval, investment, and complicity that myself, my colleagues, and my friends were steeped in. The Father was illuminating.

After the training, I was asked to give a brief presentation to the delegation and medical team on my research— epigenetics and intergenerational trauma in the context of conflict and inequality. I unfurled a large roll of white paper and taped it over the cabinets in our little cinderblock house to make a kind of whiteboard. I started with the gene, passed it through the maternal body, and passed her through society. I made a big arrow looping back to the gene. I drew methyl marks, histone tails. I erased them and drew earmuffs, a gene silencing metaphor that is a bit more on the nose. I reminded my audience that (almost) every cell in your body contains your whole genome, that the information for the whole of you is in every speck of skin you leave in the dust behind you. I explained that the epigenome, like the dog-ears in your books, marks for each cell what to read and when to read it. And that while the pages you have, half from your mother, half from your father, are stable and set— the dog-ears can change over time. I told them that each cell responds to what is asked of it by shifting what it reads and when, ever seeking equilibrium. I tell them— we cannot change the words on the page, but we can blot them out, rearrange them, make with the same ingredients a custard or an omelet. And if we do this early, if those earliest progenitor cells that will go on to make a brain and body are dog-eared just so, those changes might be with us through our lives.

It is quite a story, isn't it?

I felt good, that day, telling it. I could see I had caught my audience's spirit. I had not lost them. They looked back brightly at me. I asked them if they had any questions.

The Father stood up.

He thanked me. He said that finally, he understood why his daughter had overdosed on heroin and died. He said he had never understood that before. He and his wife had given her, he said, a perfect childhood. They had never understood why she had been drawn into addiction. Thank you, he said. It must have happened long before she came to us.

In a millisecond, I reappraised everything The Father had said and done, all the antipathy I had felt towards him. I thought about all the times he had proudly shown a photo of the young woman, a cascade of curling hair down her to her waist and had never said she was dead. I was dumbstruck at the quality of that kind of grief and what the trip must have meant to him.

When I found my words, I said, I am so sorry, I didn't know she had passed.

Well, she didn't, he said. They brought her back with Narcan. She isn't speaking to us anymore, but I hope she'll come back home, once she figures out whatever she needs to.

Chapter 1: Preconception trauma and offspring epigenetics: a systematic review

Social epigenetics and the intergenerational transmission of trauma

Stressful experiences lived by earlier generations can impact the health and well-being of descendants, processes some have proposed to be physiologically programmed by epigenetic mechanisms. Intergenerational patterns of traumatic re-exposure and poor mental and physical health are broadly known as the 'intergenerational transmission of trauma' (Cerdeña et al., 2021). The idea that trauma may be inscribed on the genome itself and transmitted to offspring has proven compelling to clinical and popular audiences alike (Yehuda et al., 2018), but scientific clarity around the type and timescale of trauma exposure and its replicable impact on human epigenetic variation remains elusive. Under the influence of the Developmental Origins of Health and Disease hypothesis, most research in this area has examined the impact of maternal prenatal stress on offspring epigenetics (Nowak et al., 2020; H. Palma-Gudiel et al., 2015; Helena Palma-Gudiel et al., 2018). However, to our knowledge no review has examined systematically the impact of traumatic stress experienced by either parent (or other ancestors, such as grandparents) prior to conception. Given the ethical, scientific, and political implications of preconception epigenetic effects and to better characterize this emergent literature, we conducted a systematic review of traumatic stress

(defined as exposure to traumatic life events and/or child maltreatment) in parents or grandparents prior to conception and offspring epigenetic changes at any age.

We use the term “epigenetics" to refer to chemical or structural changes that alter gene expression or chromatin structure without changing underlying genetic code (Greally, 2018). These include histone modifications, DNA methylation, and RNAs that regulate gene expression, protein synthesis and cellular function. Although mRNA is not traditionally understood as performing regulatory functions, recent evidence has shown that protein-coding RNAs also may exert regulatory effects, and so gene expression itself is included here as an epigenetic outcome of interest (Hubé & Francastel, 2018). The most widely studied of these mechanisms in human populations are DNA methylation and gene expression, for which high throughput microarrays have been developed and increasingly deployed in biosocial epidemiologic studies. Changes in DNA methylation or gene expression are thought to index stable alterations in cellular function (or cellular ‘reprogramming’) including the regulation of cellular responses to its nutritional, endocrine and immune environment. Because DNA methylation and histone modification also are responsible for cellular commitment and tissue differentiation, they may index organizational changes to organismal tissue-level somatic investments (Suelves et al., 2016). Thus, epigenetic changes in signaling pathways during developmentally sensitive windows may be responsible for altered trajectories of organismal physiology such as regulation of hormonal axes and immune function, to result in changes to susceptibility to chronic disease, mental illness, and reproductive disorders (Hunter & McEwen, 2013). Most DNA methylation at a CpG sites, cytosine-guanine nucleotide sequences along the 5’ end of the DNA strand. Methods for assessing DNA methylation have evolved from pyrosequencing to large commercial microarrays, to whole genome bisulfite sequencing and beyond, but most begin with the bisulfite conversion of unmethylated cytosines into uracil, leaving methylated cytosines unchanged.

The continuum of stressful experiences ranging from mundane to traumatic is challenging for researchers to operationalize, given the multidimensional, culture-bound, and intersectional nature of what constitutes difficult, frightening, or emotionally overwhelming experiences across the human life course (Kaiser & Weaver, 2019; Lester, 2013). Intergenerational trauma as a term is used by researchers, clinicians, and activists to mean multiple things: cumulative psychological or emotional wounding that is passed from generation to generation (Heart & Horse, 2000) as well as the broader multi-generational psychological, behavioral, and biological impact of stressful experiences experienced by ancestors (Bombay et al., 2009). Most psychobiological research paradigms conceptualize intergenerational trauma in more limited terms as the relationship between ancestral PTSD symptoms and/or life-time exposure to violence, death, loss, and severe illness using DSM criteria (Weathers & Keane, 2007). Some also include child maltreatment under the umbrella of intergenerational trauma to encompass experiences of neglect, abuse, and "household dysfunction" such as parental incarceration, mental illness, divorce, or substance use (Negriff, 2020).

An extensive literature concerns the epigenetic 'prenatal programming' of offspring gene expression to influence cognitive, mental, and physical health following in utero exposure to the maternal stress hormone milieu (Thayer et al., 2018). This research emphasizes a pathway from perceived stress, stressful life events, or maternal mood disorders that occur specifically during the prenatal period leading to dysregulated nutritional availability, glucocorticoid release, and diminished immune tolerance, which in turn trigger activational and organizational changes in fetal neurobiology to produce increased vulnerability to mental and physical health problems after birth (Gillespie et al., 2019; Marini et al., 2020). But rather than be limited to the window of shared prenatal experience, evidence further suggests that parental traumatic stress experienced prior to conception— especially during sensitive periods of their own development— can have lasting impacts on the future in utero and postnatal environment parents provide to their children (Scorza et al., 2018). Indeed, evidence

from perinatal epidemiology has highlighted the limited efficacy of interventions focused on the prenatal period and called for renewed attention to preconception health as an important driver of pregnancy and neonatal outcomes (Stephenson et al., 2018).

The precise mechanism by which stress experienced by one generation results in altered epigenetic patterns in offspring and whether they mediate observed associations between stress experienced in ancestors and offspring outcomes is unclear and often controversial in human biosocial research (Horsthemke, 2018; Warin et al., 2020). Such inheritance might occur via germline, vertical, social, and/or ecological pathways, and indeed in humans apparently represents a complex combination of each of these pathways. We begin with a broad overview of the development of the stress responses system and then review potential pathways of preconception trauma transmission in further detail below.

Inheritance of the stress response system

Models of the epigenomic impact of intergenerational trauma center on the developmental programming of the stress response system, an evolutionarily conserved set of central and peripheral neuroendocrine systems that appraise potential threats and coordinate physiological responses to promote survival and fitness (Kültz, 2005). The major components of the stress response system include the sympathetic adrenomedullary axis— which coordinates short-term responses via catecholamine release to raise heart rate, blood pressure, vigilance, and reflexive physical responses— and the longer-term responses of the hypothalamic pituitary adrenal (HPA) axis. Because of the HPA axis' lasting and global impacts on neurobiology, behavior, immune function, metabolism, and reproductive health, the HPA has been a historic target of research examining the biological embedding of early life experience and intergenerational trauma (Bowers & Yehuda, 2015; Ponzi et al., 2020; Somvanshi et al., 2020). The HPA cascade begins with the activation of stressor

and threat appraisal in neural circuits connecting the prefrontal cortex with the meso-limbic system, which result in the secretion of CRH from the hypothalamus. CRH binds to receptors in the pituitary, which then results in ACTH release from the pituitary into peripheral circulation and subsequently synthesis and release of glucocorticoids from the adrenal cortex (Danese & McEwen, 2012). Circulating glucocorticoids enter the peripheral bloodstream and bind with cytosolic glucocorticoid receptors (GR) that are present in nearly all tissues. As steroid hormones, unbound glucocorticoids pass freely through cell membranes. After ligand binding, the activated GR is joined by a series of heat shock chaperone proteins beginning with FKBP5-1 that enable translocation to the nucleus and transcription of glucocorticoid response elements (GRE). Expression of GRE coordinate a suite of responses to promote survival in hours following stress exposure including cell differentiation, anti-inflammatory signaling, catabolism of cellular proteins, and increases in blood glucose (Nesse & Young, 2000).

The genes that encode the GR and its co-chaperone protein FKBP5-1 have been the most widely investigated candidate genes in studies of intergenerational trauma and early life adversity. The GR is encoded by the gene NRC31 (nuclear receptor subfamily 3, group C, member 1) which contains multiple promoter regions that regulate glucocorticoid sensitivity across tissues (Turner & Muller, 2005). Exon 1F is in the 5' UTR of NRC31 and is associated with GR expression in immune and hypothalamic cells. Methylation in this region is thought to act as a stable repressor to decrease binding of transcription factors and decrease GR expression, which in turn decreases cellular glucocorticoid sensitivity (Armstrong et al., 2014). Similarly, methylation within the intergenic region of FKBP5 is thought to decrease expression, but here the effects on glucocorticoid sensitivity are reversed. This is because while necessary for activation of the GR at low levels, when present in high levels FKBP5-1 decreases the GR's affinity for glucocorticoids, resulting in diminished translocation and transcription of GREs (Binder, 2009). FKBP5 is itself a GRE whose transcription is instigated

by the GR, and thus acts as a mechanism of rapid negative feedback in peripheral tissues. Its expression in the hypothalamus also impacts central down-regulation of the HPA itself.

Despite the ubiquity of epigenetic reprogramming of the HPA axis as a key mechanism of the intergenerational transmission of trauma, reviews of human and animal literatures indicate variation in the directionality of NRC31 methylation in the wake of trauma. Turecki and Meaney (2016) found increased DNA methylation of exon 1F of NRC31 in all studies of early life adversity (child maltreatment or exposure to traumatic stressors) and/or parental stress, but results from studies of adult psychopathology and NRC31 methylation were inconsistent (Turecki & Meaney, 2016). Palma-Gudiel and colleagues (2015) attempted to review literature demonstrating that NRC31 methylation mediates the relationship between early life stress and later psychopathology. They found few studies that included full mediation models and instead reviewed 21 studies linking early life stress to NRC31 methylation, of which 17 reported significant findings. Nevertheless, they note the lack of replication of patterns of hyper- and hypo-methylation of different CpG sites within the gene and call for both standardization of assays and methylation reporting statistics as well as site annotation. Finally, in a recent systematic review, increased NR3C1 methylation (across various regions) was "robustly" associated with childhood trauma in adults who were healthy, depressed, or suicide-completers, but not those with PTSD or anxiety disorders (Nöthling et al., 2020).

Pathways of preconception intergenerational transmission

Germline transmission

The capacity for transgenerational or ancestral exposures to impact offspring phenotypes is both commonly recognized (for example, in the intergenerational transmission of stunting, low birthweight, or teratogen exposure) and polemic, as it carries associations with Lamarckism (Horsthemke, 2010; Jablonka & Lamb, 2007). Popular discourse typically conflates transgenerational

epigenetic effects with durable epigenetic changes to the germline (and vice versa), although such changes need not occur for an epigenetically programmed effect to be perpetuated through somatic, social, or ecological mechanisms of transmission. DNA methylation is mitotically heritable across cell divisions, but its stability during gametogenesis and post-fertilization is less clear (Eckersley-Maslin et al., 2018; Radford et al., 2014). The genomic integrity of the gametes traditionally have been understood as buffered from insults to the soma— the so-called "Weismann barrier" (Nilsson et al., 2020). Recent evidence has shown this barrier to be more porous than canonically thought; extracellular vesicles known as exosomes have been found to transport microRNAs from parental serum to the gametes prior to conception, resulting in potentially advantageous changes in offspring phenotypes. Experimental evidence for the role of small noncoding RNAs in conferring key experiential information to the future fetus is strongest in the literature on paternal sperm-based epigenetic research (Immler, 2018; Ryan & Kuzawa, 2020). However, emerging work has also shown that exosomally packaged RNA influences oocyte development in ways that might improve fetal viability based on maternal experience (de Ávila & da Silveira, 2019).

The transgenerational durability of DNA methylation is undermined by the partial stripping of methyl groups from the genome of the pre-implantation human embryo and near-universal demethylation of the fetal primordial germ cells (excepting some transposons) prior to migration to the gametes (Smallwood et al., 2011). However, this demethylation event is not truly complete; maternal methylation of some CpG islands is retained throughout the pre-implantation period, as is methylation of imprinting control centers and some transposable elements (A. T. Clark, 2015; Luo et al., 2018). Germline effects also may occur independently of retained DNA methylation; although the paternal genome is ~~also~~ rapidly hypomethylated at conception, research on the regulatory effects of microRNAs within sperm has provided some of the most powerful experimental evidence for the

transgenerational inheritance of paternal experience in rodent models (Dias & Ressler, 2014; Rodgers et al., 2015; Ryan & Kuzawa, 2020)).

Researchers and bioethicists correctly have cautioned against over-interpretation of potential germline effects and the rise of 'epigenetic determinism' (Waggoner & Uller, 2015). Rather than bridging nature-nurture divides and emphasizing organismal responsivity to environmental cues, epigenetic 'soft-inheritance' may reinforce biopolitical paradigms of inherent vulnerability and impairment of trauma-exposed populations (Meloni, 2015). Paradoxically, it may also serve to reify diploid genome identity as "true identity", positing epigenetic editing or mitochondrial replacement as ethically acceptable where embryonic selection or gene editing would not be (Darnovsky, 2013).

Maternal somatic transmission

Without germline changes, preconception traumatic stress may yet exert important effects on a future developing fetus and child through non-genetic parental effects. These broadly include parental care and the developmental environment, but also what we term here as maternal somatic transmission. Somatic transmission here refers to changes in the development of maternal physiology that may affect future fetal development due to maternal trauma exposure before conception and pregnancy. One such pathway includes changes in maternal HPA axis function that persist through the perinatal period. Childhood maltreatment has been associated with the development of HPA axis hypo- and hyper-activation, which may have important consequences in future pregnancies (Alexander et al., 2018). During pregnancy, maternal and fetal HPA activity is intermingled; as the fetal HPA develops, the placenta secretes placental CRH (pCRH) into maternal circulation, stimulating maternal pituitary ACTH and adrenal glucocorticoid synthesis to increase maternal blood glucose concentrations and meet fetal growth demands (Gangestad et al., 2012). Maternal sensitivity to fetal pCRH may be programmed by preconception early life stress; a recent

study found that childhood traumatic events (but not adulthood or prenatal trauma exposure) predicted steeper rises in pCRH concentrations in the second and third trimester of pregnancy, a signature that may indicate prenatal maternal central glucocorticoid desensitization (Steine et al., 2020). The fetus is largely buffered from circulating maternal glucocorticoids by the action of a placental enzyme, 11β-hydroxysteroid dehydrogenase type 2 (HSD11B2), which converts maternal glucocorticoids into inactive metabolites. However, HSD11B2's buffering efficacy is diminished under conditions of maternal stress, high levels of glucocorticoids, and the presence of maternal inflammation, and epigenetic programming of placental HSD11B2 has been associated with maternal mood disorder and infant neurodevelopment and HPA activity (Appleton et al., 2013; Conradt et al., 2013; Stroud et al., 2016). Thus, the HPA axis a mother brings to pregnancy may be just as important as its activation during pregnancy in shaping the endocrine environment of the developing child.

Another pathway of preconception maternal somatic transmission involves the early life programming of the maternal immune system that influences its function prior to implantation and throughout pregnancy. Early life adversity has been associated with increases in pro-inflammatory signaling in adolescents and adults, both in terms of relative investments in innate vs. adaptive immunity as well as skew towards pro-inflammatory phenotypic expression within leukocyte subsets (Aschbacher et al., 2021; Elwenspoek et al., 2017; Georgiev et al., 2016; Smallwood et al., 2011). The impact of early life adversity on immune system development likely represents adaptive trade-offs in human life history strategy; given limited energetic supply and increased energetic demands due to stress, investments in costly adaptive and/or cell-mediated immunity are diminished (McDade, 2003). Female life history strategies especially may reflect such tradeoffs, given their relatively larger energetic investment in reproduction through the energetic demands of pregnancy and lactation (Abrams & Miller, 2011). Pro-inflammatory immune trade-offs triggered by//related to early life

adversity are especially relevant given the key role maternal immune tolerance plays throughout conception, implantation, and pregnancy. Before implantation, T-regulatory cells, polarized macrophages, dendritic cells, and natural killer cells migrate to the uterine decidua. There, they suppress local inflammatory responses to the fetal allograft as well as repair and remodel tissues necessary for the formation of the maternal-fetal interface and healthy establishment of the placenta (Peixoto et al., 2018; Reyes & Golos, 2018). Differential investment in the immune compartment as well as pro-inflammatory programming due to early life trauma thus could result in placental vascular malperfusion and dysfunction, increasing the risk of preterm birth, the hypertensive disorders of pregnancy, and low birthweight (Burton & Jauniaux, 2018; Ernst, 2018).

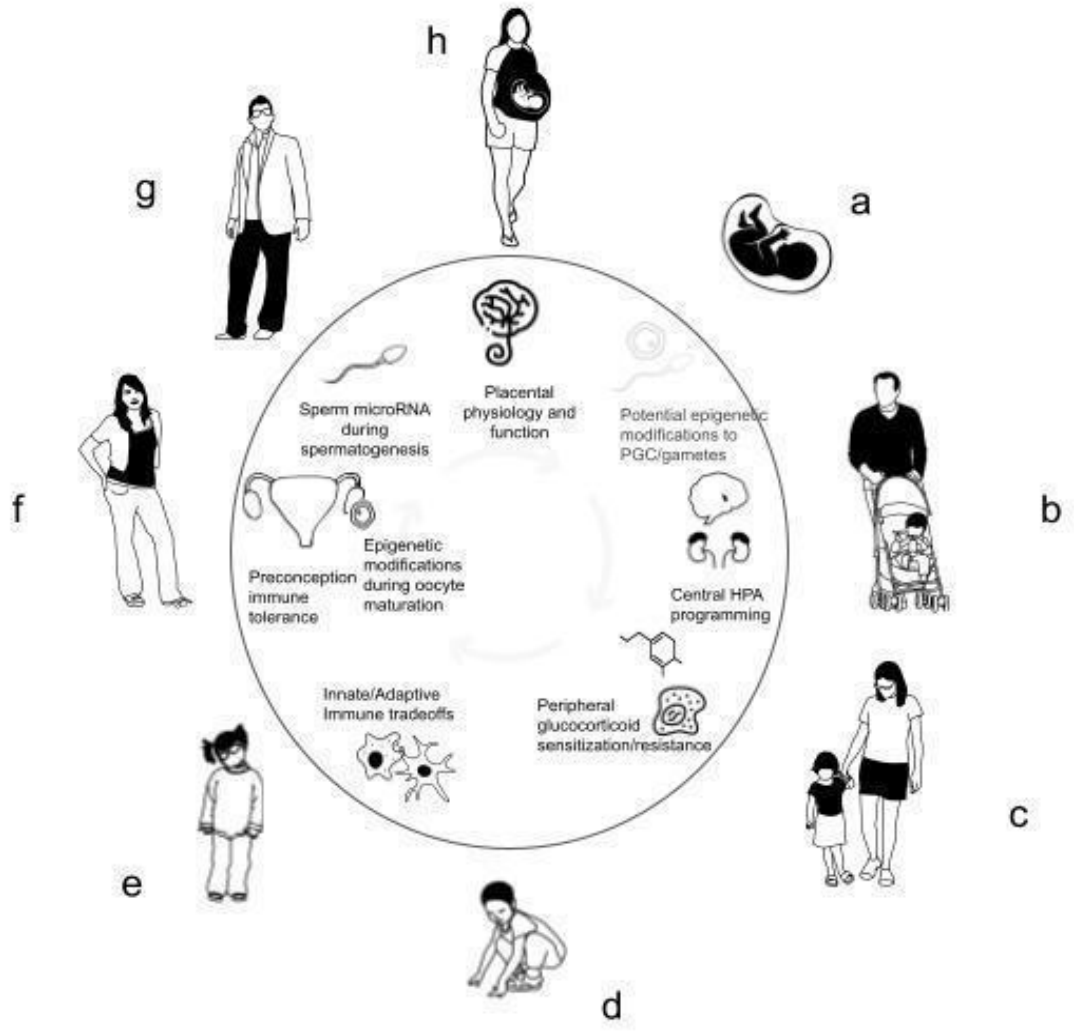


Figure 1. Intergenerational cycles of germline and maternal somatic transmission are depicted. Because the fetal gametes undergo critical periods of development in utero, epigenetic modifications of the germline (a) are theoretically possible, although important caveats apply (thus this caption appears in grey font). Throughout early life, stress exposure is also thought to impact (b) central HPA axis programming and (c) peripheral sensitization and resistance as children shift activational thresholds to maintain homeostasis. In tandem, early life programming of immune system investment (e) sets the stage for alterations to the now adult woman's preconception immune tolerance of the fetal allograft (f) and potential modifications to oocytes prior to ovulation. In (g) stress experienced during preconception period during which spermatogenesis occurs may result in exosomal vesicle transfer of microRNAs to seminal fluid. The environment that F1, the pregnant woman's child experiences—for example through placental physiology at the maternal-fetal interface (h) is thus shaped by the impacts of stress on F0's life course.

Caregiver-child relationship

Sensitive periods of offspring development continue postnatally into adulthood, and caregiver-child relationships structure the developmental environment in ways that bear a lasting impact on offspring psychobiological function (Worthman, 2009). Intergenerational patterns of socially learned parenting behaviors have been posited as a key mechanism of trauma transmission. Multiple longitudinal studies have demonstrated the perpetuation of socially learned harsh parenting and child maltreatment across up to three generations (Bailey et al., 2009; Conger et al., 2009; Michl-Petzing et al., 2019; Simons et al., n.d.). However, such work has also come under critique for stigmatizing parents whose parenting practices may reflect untreated mood disorder or parenting stress related to economic deprivation, intimate partner violence, and state or structural violence rather than poor parenting skills (Michl-Petzing et al., 2019; Sim et al., 2018). Ethnocentric norms of appropriate parenting may especially stigmatize highly trauma-exposed populations, who may be living in poverty, involved in state carceral systems, non-white, identify as a gender or sexual minority, or foreign-born (Asnaani & Hall-Clark, 2017; Raffaetà, 2016; Roberts et al., 2010)). Beyond their direct interactions with the child, the caregivers that make up the 'developmental niche' interact and shape each other's life worlds in ways that matter deeply for children's lives. Family conflict arising from material struggle, system involvement, and power inequalities within the family suffuse the developing child's emotional world and psychophysiology (DeCaro & Worthman, 2007).

Ecosocial inheritance

Despite the emphasis on parenting (especially that of mothers), intergenerationally inherited social structures play a key role in structuring trauma exposure over the life course and across generations. Researchers have demonstrated the intergenerational transmission— although perhaps the word "reproduction" may be more appropriate here— of poverty, discrimination, gender-based

and sexual violence, political violence, incarceration, and community violence (Benner & Kim, 2009; Borgerhoff Mulder et al., 2009; Klungel, 2010; Manduca & Sampson, 2019; Sangalang & Vang, 2017; Vartanian et al., 2007; Zhao et al., 2020). Intergenerational experiences of structural violence— the systematic oppression of subjects and groups of people that results in excess death and suffering— emerges as a force by which durable political-economic and cultural structures (cis-hetero patriarchy, colonialism, structural racism, discrimination by gender, ethnicity, migrant or refugee status) are maintained and reproduced (Farmer, 2004). The structural roots of intergenerational trauma exposure and its consequences for wellbeing increasingly are a focus of contemporary trauma studies, resulting in multidisciplinary shifts in assessment, intervention, and community engagement (Danzer et al., 2016; Mohatt et al., 2014; Ruíz, 2020). Nancy Krieger's development of the term 'embodiment' for social epidemiology — the process by which the social environment is incorporated into the material of the body and thus to pattern population health— has done further conceptual work to knit individual biology into social structure (Krieger, 2005).

Ecosocial approaches emphasize the fundamental causes of trauma exposure, but they also signal the contextual and cultural specificity of how trauma is experienced and embodied. The sociocultural norms that shape traumatic appraisals and their impacts on agency, selfhood, and moral injury in turn shape trauma symptomatology and neurobiology (Bernardi et al., 2019; Zefferman & Mathew, 2020). Such work shines light on why experiences of violence do not universally provoke trauma symptoms and/or shift trajectories of stress reactivity.

Integrative models

Drawing on interacting pathways of somatic and social inheritance, integrative biocultural models of the intergenerational transmission of trauma have emerged from anthropologists, Indigenous studies scholars, and social neuroscientists. Conching and Thayer (2019) suggest an integrative model of historical trauma that accounts for social inheritance of individual risk for

personal trauma exposure via structural oppression and political disenfranchisement of historically marginalized populations— a generationally shared 're-traumatization'. In parallel they integrate the developmental effects of exposure to the intrauterine and postnatal hormonal milieu in response to maternal stress and challenges in the parent-child relationship due to overextended parental material and emotional resources (Conching & Thayer, 2019). Kirmayer, Gone, and Moses (2014) de-emphasize the role of in utero programming in their model, instead taking a bird's-eye view to nest individual HPA axis programming within layers of cultural loss, individual and familial traumatic stress, and political dispossession experienced by Indigenous peoples of the Americas. In doing so, they underscore the differences in the social context that shapes international traumatic re-exposure of Indigenous populations in contrast with commonly studied non-Indigenous populations from which intergenerational trauma concepts are theorized (Kirmayer et al., 2014).

In our integrative model (see Figure 2.) we situate embodied cycles of life course trauma (germline and maternal somatic transmission) within an ecosocial model of the fundamental causes of trauma exposure, as they suffuse through community and individual experience.

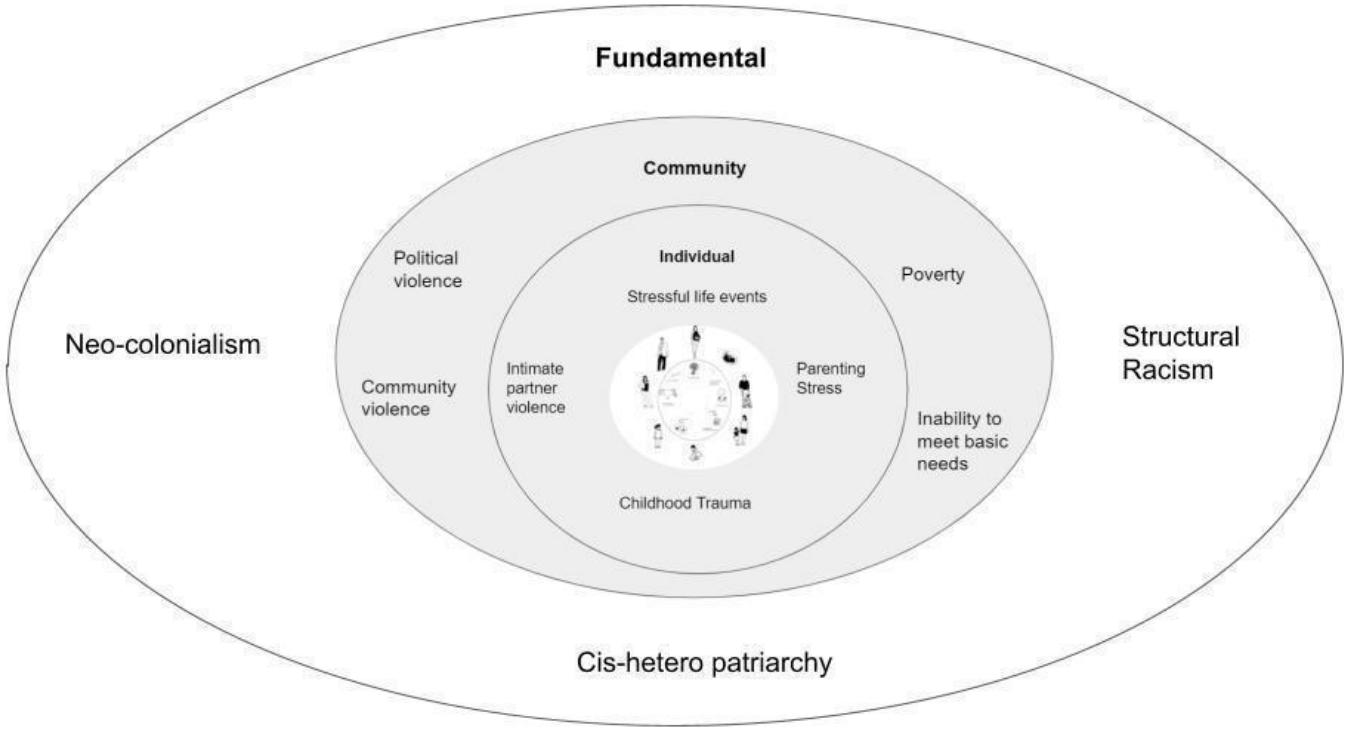


Figure 2: An integrative biosocial model of intergenerational transmission. We nest our previous model of embodied transmission in layers of individual, community, and fundamental causes that activate trauma-related stress responses. At the individual level, stressful life events, parentings stress, childhood trauma, and intimate partner violence represent how most social epigenetic research studies measure the burden of trauma. At the community level (representing for simplicity's sake both the local and nation-state), economic precarity, community violence (e.g., witnessing or being victimized by crime and/or over policing), and political violence experienced as persecution, discrimination, ethnic cleansing, forced flight, warfare, and genocide. The final level of fundamental causes includes macrosocial structures that shape the distribution of power and coercion across lines of race, gender, and neo-colonial exploitation of the Global South and First Nations territories.

Intergenerational effects and DOHaD

The heart of the Developmental Origins of Health and Disease hypothesis is an evolutionary-developmental theory of an offspring's attempts to adapt to the early cues of the postnatal environment, leading to problems associated with mismatch between anticipated and actual environments (Gluckman et al., 2007). Although theory in this area helps form predictions around how and why early life adversity might change offspring developmental programming, especially of the stress response system, its intergenerational aspects are less theorized. In much of the intergenerational trauma literature, the allostatic load model suggests general suboptimal functioning and development following "wear and tear" of the stress response system (Belsky & Pluess, 2009; Danese & McEwen, 2012). The diathesis-stress paradigm followed this view to propose that individual differences in vulnerability to stress underlie patterns of risk and resilience. Concurrently, evolutionary-developmental theories have called for understanding the effects of early life adversity through the lens of life-history theory, suggesting that individual differences in stress responsivity following adversity reflect tradeoffs to optimize fitness across multiple domains and timescales (Ellis & Del Giudice, 2019; Kuzawa & Thayer, 2011). The nonlinear effects of stressful environments and stress-responsivity are further explored in the biological sensitivity to context model, which posits that increased stress-responsivity is beneficial in both highly adverse and highly enriched environments. In an extension of this, the adaptive calibration model suggests multiple 'switch-points' for calibration of the stress response system across development and provides a framework for considering sex differences and reproductive strategies in evolutionary context (Del Giudice et al., 2011).

While scaffolding the development and testing of predictions, DOHaD historically has emphasized the life course of one individual, even as it posits another's (the mother's) as the first environment of exposure. Intergenerational effects that occur outside the direct experience of

offspring pose challenges for classic evolutionary theories of inheritance, particularly those that involve germline transmission. Regarded as Neo-Lamarckism, or the intergenerational transmission of acquired characteristics, epigenetic forms of 'inheritance' have come under expanded models of the Modern Synthesis under 'inclusive inheritance', niche construction, and a broad literature non-genetic parental effect (Laland et al., 2015). Thus, if adaptive calibration models of individual developmental programming call for attention to how and why sensitivity to environmental cues should evolve within individuals, intergenerational effects expand the timescale to include environmental cues experienced by parents, grandparents, and ancestors.

Much of the theory on intergenerational effects at short evolutionary timescales comes from studies of metabolism and intergenerational patterns of nutrition and child development. Kuzawa (2005) frames the ineffectiveness of prenatal supplemental nutrition on child growth by proposing a model of 'phenotypic inertia', whereby the maternal uterine environment (and its nutrient provisioning and buffering) conveys the average information about the nutritional environment as opposed the cues from just prenatal nutritional abundance or short falls. Hence, phenotypic inertia represents the degree to which maternal developmental conditions influence offspring phenotypes in ways that outweigh or interact with the child's own early life environment. Thus, under phenotypic inertia, although current conditions may improve, offspring phenotypes will conservatively reflect 'anticipation' of long-term adverse environmental cues. This view differs from a perpetuation model (such as that proposed by Kirmayer et al 2014), in which stable, politically inscribed forms of adversity emerge from past colonial trauma to re-expose generation after generation to trauma via structural violence.

Methodological challenges in social epigenetics

Beyond the difficulties of measuring multiple generations of traumatic exposure and impact, social epigenomics also must contend with specific methodological challenges inherent to biological and technical confounding. The use of surrogate tissues— such as buccal cells, saliva, or peripheral blood— undermines the functional plausibility of observed epigenetic associations. Because DNA methylation mediates cellular differentiation and because cell-type admixture may be related to outcomes of interest, investigators must be thoughtful about the use of methods to account for cell-type heterogeneity. These range from relatively simple (monocyte/lymphocyte ratio) to more complex reference-based and reference-free deconvolution algorithms (Teschendorff & Relton, 2018). Technical artifacts such as batch effects also must be addressed up front. Finally, DNA methylation has been shown to be under direct genetic control. Gaunt and colleagues (2016) have developed a public catalog of methylated quantitative trait loci (mQTL) using data from the Avon Longitudinal Study of Parents and Children. In a sample of nearly 800, they compared life course epigenome-wide methylation of two maternal and three offspring timepoints with whole genome sequencing. They found that the genetic contribution to methylation was never less than .2, although this varied across the life course, with stronger correlations between mQTL in childhood than in later life. Best practices for controlling for genetic confounding are still developing in the field, with many researchers including no controls, ancestry estimates from genome-wide sequencing, or exploring moderation by polymorphisms in the candidate genes under epigenetic investigation.

Rationale

Research exploring the impact of preconception trauma on offspring outcomes must negotiate the significant biological, conceptual, and methodological challenges outlined above. Despite this thorniness, public perception that trauma can be inherited and passed on through generation continues to grow, suffusing public and mental health interventions for parents, schools, and communities(Müller & Kenney, 2021; Overbeek et al., 2020; Yehuda et al., 2018). This review aims

to clarify the current empirical basis of these beliefs as well as investigate the rationales, mechanisms, and implications for clinical and social policy that the researchers themselves make in their work.

Methods

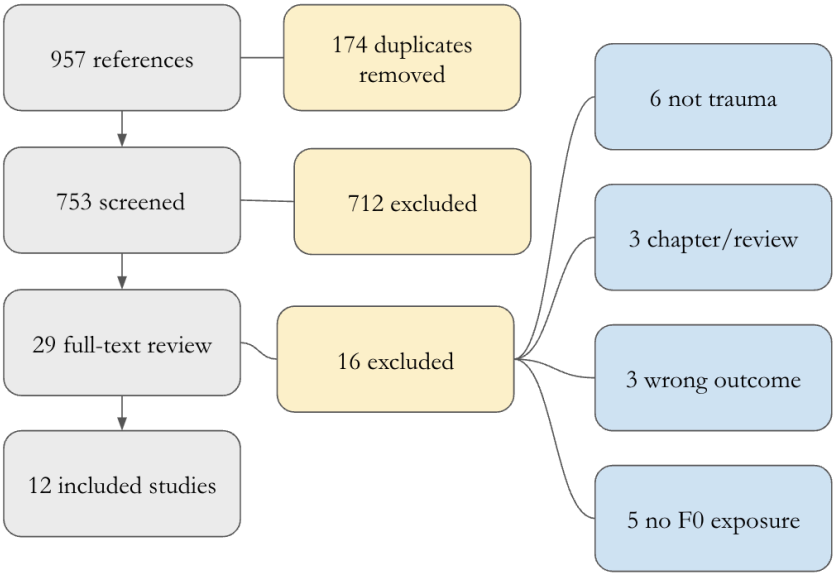
We conducted an extensive scoping review by searching the following databases for relevant studies: MEDLINE ALL, Embase, Web of Science Complete, Scopus, and PubMed. Initial searches were conducted between November 1 and November 15, 2021. Full details of databases searched can be found in Appendix 1. The review objectives and search strategy were submitted on 10/1/2021 to the PROSPERO registry for systematic reviews. The full list of search terms used is available in Appendix 1. Databases were searched by L.M.R under the supervision of an experienced research librarian, Dr. Lori Jahnke. Search strategies and syntax were tailored to each database. Where possible, searches were limited to human samples, and to empirical articles published in English between 1/1/2000 and 11/1/2021. In accord with suggested best practices in systematic review search strategies, search results were confirmed by examining the first 200 results from a Google Scholar search of the keyword list; no novel eligible articles were found (Bramer et al., 2017). Search results were uploaded into Covidence systematic review software, which identified and removed duplicates (Covidence, n.d.).

This scoping review follows the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) statement and checklist (Appendix 2). 927 articles were identified during the search. L.M.R screened titles and abstracts for inclusion and exclusion criteria and identified 753 articles as eligible for full text review. Of these, 29 appeared to meet criteria for inclusion. Studies were eligible if they were empirical studies published in English where 1) preconception F0 trauma exposure (e.g., exposure to stressful life events, intimate partner violence, war trauma, or PTSD symptoms) was measured as the primary predictor of 2) F1 or F2

DNA methylation, histone modification, or gene expression outcomes. See Figure 1 for clarification of generational nomenclature. Studies were excluded if 1) trauma exposure was only measured during the prenatal period of the exposed offspring, 2) non-trauma-focused forms of adversity such as nutritional stress, low socioeconomic position, environmental toxicants, obesity, or smoking in F0 was assessed with no measure of psychological traumatic stress, or 3) only non-epigenetic outcomes (e.g., mental health, endocrine function, growth, developmental outcomes) were assessed in offspring. Twelve studies met full criteria for inclusion (See Figure 3).

L.M.R. extracted the study design, rationale, hypothesized mechanisms of intergenerational trauma transmission, sample size and characteristics, intergenerational trauma exposure conceptualization and measurement, epigenetic outcome conceptualization and measurement, main findings, and clinical or policy implications. Wherever possible, we report standardized coefficients and results of statistical testing. Study quality was assessed by review of the representativeness of samples, assessment of offspring adversity exposure and mental health, measurement of parental mental health, measurement of offspring phenotypes of interest (hypothesized to be mediated by epigenetic programming, e.g., hormone levels, mood disorder), statistical controls for cell-type heterogeneity, correction for multiple testing, and assessment of genetic confounding.

Figure 3. **PRISMA Flowchart**



Results

12 articles met full criteria for inclusion. All were published after 2014; the majority (8) were published after 2020. Six publications concerned samples where F0 was known to have high exposure to violence or traumatic stress, including the Holocaust (4 studies), the Kosovo war (1 study), and high levels of community violence (1 study). One study sampled children known to be experiencing chronic pain, while the remainder (5) were birth cohort studies with no specific or known trauma exposure at enrollment.

Eleven of the studies addressed preconception trauma in F0 and descendant outcomes in F1. A single study examined how prenatal stress experienced by F0 impacted F2 grandchildren (Serpeloni et al. 2017). Offspring ages ranged from infancy (5 studies), late childhood/adolescence (3 studies), to adulthood (4 studies). All but one study was conducted in the global north; one took place in Sao Gonçalo, Brazil. All the studies of Holocaust survivors and offspring (4) used case-control designs, that matched exposed F1 offspring to demographically similar Jewish control adults

whose parents lived in North America during the Holocaust. The rest used observational cohorts recruited in hospital or community settings and modelled predictors through a variety of approaches including trauma exposure threshold, PTSD diagnosis dichotomization, and/or continuous scores of childhood trauma exposure or exposure to childhood adversity (see Table 1).

All but one of the studies used a candidate gene approach to test F0 trauma-related differences at specific CpG sites or regions. One study reported epigenome-wide DNA methylation results only (Serpeloni et al. 2017), and two conducted a combination of EWAS and candidate gene analysis (Hjort et al. 2021; Merrill et al. 2021). The most frequently tested candidate gene was FKBP5 (6 studies), specifically DNA methylation in various sites in intron 7. The next most frequently tested genes were NRC31 (5 studies, all in exon 1F) and BDNF (2 studies). The other target candidate genes (NRC32, HTR3A, SLC6A4, CRH1, SERT, COMT, DRD2, OXTR) were each tested in one study only.

Study rationales

Study rationales emphasized the need to "elucidate mechanisms" of intergenerational transmission of trauma given epidemiological and clinical data suggesting that offspring of trauma-exposed parents are at higher risk of physical and mental health problems. "Prospective intergenerational studies are needed to elucidate the mechanisms for such trauma effects, as well as their functional consequences for vulnerability, adaptation, and resilience." (Bierer et al. 2020, p. 752). Nonetheless, several studies noted the limited ability of epigenetic correlational studies to "speak to the mechanism through which epigenetic alterations are acquired," (Daskalakis et al. 2021, p. 752, see also Christensen 2021, p. 7).

Another common rationale was discovery, either explicitly stated in the introduction or emphasized in the discussion. Eight of the twelve papers reported that they were unaware of other papers that had tested the precise correlations between adult preconception trauma and offspring epigenetic outcomes in their specific study. For example, Grasso and colleagues cited evidence from Yehuda et al. 2016 in humans and a preprint from Klengel et al. 2019 in rhesus macaques, that found correlates of preconception stress with offspring FKBP5 methylation and went on to note that to their knowledge, no previous report had tested direct correlations between parent and offspring methylation of FKBP5. Similar statements of novelty were present in Daskalakis et al. 2021, Christensen et al. 2021, Hjort et al. 2021, Merrill et al. 2021, Ramo-Fernández et al. 2021, Serpeloni et al. 2017, Yehuda et al. 2016, and Yehuda et al. 2014.

Pathways of transmission

The process by which offspring came to acquire epigenetic changes in the wake of preconception trauma was assessed across studies. A common mechanism suggested in all studies was maternal somatic transmission. However, models varied on how preconception traumatic experiences might systematically change a future pregnancy's hormonal milieu. Some studies examined parental PTSD as a potential mediator of preconception trauma's impact on offspring DNAm methylation, although analytic approaches varied in how PTSD was thought to have impacted prenatal physiology or parenting (or both). For example, in their follow-up studies of Holocaust survivors and their offspring, Bierer et al. 2020 and Daskalakis et al. 2021 included F0 PTSD and F1 childhood exposure as covariates (e.g., potential confounders or colliders) rather than mechanisms (via tests of mediation). By contrast, Hjort et al. 2021 hypothesized that F0 PTSD related to preconception war trauma would exert pregnancy-specific effects on offspring. Postnatal effects via the parent-child relationship and/or parenting were seldom assessed (see Yehuda et al.

2016, Yehuda et al. 2014 for exceptions). In addition, while they did not directly evaluate the parent-child relationship (as offspring were newborns at the time of the study), Ramo-Fernández et al. 2021 suggested that alterations to social bonding through epigenetic programming of the oxytocinergic system may underlie intergenerational transmission.

Several papers proffered a pathway involving germline transmission albeit with varying degrees of caution. In their study of the intergenerational transmission of parental adverse childhood events to offspring with chronic pain, Christensen et al. 2021 suggest "heritable epigenetic and behavioral changes" (p. 8). Serpeloni et al. 2017 proposed a quasi-germline effect in their study of grandmaternal prenatal stress and F2 grandchild epigenetic outcomes but were quick to note that such an effect should not be considered trans generationally programmed through the germline unless it persisted to F3. Lastly, germline modifications were more explicitly suggested in Merrill et al. 2021 in their study of paternal ACEs and infant DNAm, drawing on an emergent literature documenting specific epigenetic modification of sperm (small noncoding RNAs, post-meiotic changes) contingent on paternal experience. Given the previously discussed evidence of demethylation during embryogenesis, the caution expressed by these and other authors about interpreting evidence for germline transmission appears warranted.

Notably, most reports pointed to unknown mechanisms, given that correlational results could not precisely specify the process by which epigenetic modifications in offspring were acquired.

Developmental-evolutionary frameworks

Several reports invoked ultimate or evolutionary frameworks to contextualize why biological pathways mediate the intergenerational transmission of trauma. Typically, authors drew on the Developmental Origins of Health and Disease hypothesis to situate the process of preconception

trauma and epigenetic changes in offspring. Evolutionary theories of adaptation or mismatch are nested within DOHaD; alterations to developmental trajectories are thought to reflect either adaptation, such as early life tailoring of the phenotype for success in an adverse environment, or constraint, such as early life suboptimal development given scarce resources (McKerracher et al. 2020). Despite this background, most papers only superficially assessed adaptive or mismatch dynamics, perhaps implicitly supporting diathesis stress or allostatic load models of psychobiology (Belsky and Pluess 2009). In such models, stressful environmental circumstances interact with underlying genetic or biological vulnerability (via developmental processes of sensitization or otherwise) to broadly produce adverse outcomes at individual and population levels. To this end, four studies examined whether intergenerational stress responses varied by 'risk' alleles in FKBP5 and OXTR, with mixed results (see Table 1.).

The minority of studies that did frame their findings as reflecting adaptation or mismatch varied in their interpretations of findings. In Yehuda et al. 2016's influential work identifying increased methylation in site 6 of FKBP5 in survivors of the Holocaust but decreased methylation in their offspring, authors suggested F1 hypomethylation may be an adaptive response to increased glucocorticoid sensitization in trauma-exposed parents (although the effect was not dependent on parental sex and could thus not be attributed to an in-utero effect). They further consider the site dependencies of their findings in an adaptive context. Methylation of bin 2, was not related to ancestral Holocaust exposure. Rather, for carriers of the risk A/T rs1360780 allele, an offspring's own childhood trauma predicted hypomethylation. This site specificity and redundancy was suggested to reflect human developmental capacity to "facilitate maximal stress responsivity and adaptation," (p. 379).

In contrast with these findings, Grasso et al. 2021 found evidence that maternal threat-based ACES predicted increased methylation of intron 7 in infants (DNAm of four sites being reduced to

a single PCA factor). Furthermore, they reported that maternal pregnancy PTSD severity and emotional dysregulation predicted increased DNAm of FKBP5 in CC infants— those carrying the 'protective' allele. Noting these differences, they write "Increased methylation in CC infants may suggest a more efficient feedback loop, a pattern that may be adaptive for infants expecting to be born into an adverse caregiving environment. However, without knowledge of physiological responding in these infants and prospective data, it is difficult to speculate whether this intergenerational pattern reflects increased risk or a short-term protective adaptation" (p. 7).

Finally, Ramo-Fernández and colleagues (2019) addressed the role of intergenerational trauma and epigenetic programming of offspring stress responses as evidence of Neo-Lamarckism. While they did not find any main effect of maternal childhood trauma on infant methylation of OXTR, they noted that maternal and infant OXTR methylation was only correlated in mothers without childhood trauma.

> From an evolutionary neo-Lamarckian point of view, our results suggest that maternal epigenetic adaptations might only be perpetuated across CM− dyads, and not across CM+. In CM− dyads, this transmission may prepare the next generation to deal with stress. However, in CM+ dyads it might not be evolutionary adaptive to transmit the maternal adaptations, which were presumably acquired to deal with severe, detrimental experiences and thus do not provide evolutionary fitness under normal circumstances. (p.7).

Synthesis of offspring epigenetic findings

The study designs varied in terms of how ancestral trauma was measured and what kind of epigenetic outcomes were assessed (e.g., EWAS, candidate gene). The populations sampled were also very different; some studies studied war trauma survivors while others investigated relatively low rates of adverse childhood events in community samples of middle-class populations. However,

it appears that the positive findings from earlier studies of Holocaust survivors have not been replicated in subsequent studies of trauma exposure in other populations. Studies of Holocaust survivors in the US found a pattern where trauma exposure in F0 was associated with F1 hypomethylation of the glucocorticoid receptor NR3C1, and of FKBP5, a co-chaperone protein that modulates GR activity. Furthermore, biological significance of the epigenetic pattern was reflected by increases in gene expression of hypomethylated genes, glucocorticoid sensitivity, basal cortisol levels, and anxiety.

However, this pattern has not replicated in subsequent studies of FKBP5. Indeed, in the original 2014 study from Yehuda and colleagues, trauma exposure itself was not associated with offspring DNAm; rather, only parental PTSD (of both exposed offspring and unexposed controls) was predictive. Although the majority of studies emphasized the potential meaning of significant findings in their analyses (see Ramo-Fernández 2019 for an exception), the main effect of F0 trauma on F1 or F2 epigenomic outcomes was null (Ramo-Fernández 2019, Ramo-Fernández 2021, Merrill et al. 2021), sex-specific (Christensen et al. 2021, Pilkay 2021), trauma subscale- or timing-specific (Grasso et al. 2021, Hjort et al. 2021, Pilkay et al. 2021), or involved novel/unreplicated EWAS results in non HPA-related pathways (Serpeloni et al. 2017, Merrill 2021). To date, no stable alternate pathways have emerged from this work.

Epigenome-wide studies of DNA methylation and gene expression

Four studies conducted epigenome-wide analyses. Their designs and findings were heterogenous and difficult to compare, but overall do not replicate one another. An analysis of genome-wide gene expression found F0 Holocaust exposure was associated with 42 differentially expressed genes, of which none were represented in any other epigenome-wide analysis (Daskalakis et al. 2021). Subsequent gene enrichment and weighted gene co-expression network analyses found

Holocaust-associated DEGs indicating down-regulation of innate immune function and glucocorticoid sensitivity. Hjort et al. 2021 conducted an EWAS in a sample of women who experienced pre-conception sexual violence during the Kosovo war, comparing DNAm in offspring of women with and without 1) current PTSD due to wartime experience and 2) retrospectively reported PTSD during the pregnancy with the offspring. No site-specific or differentially methylated regions survived correction for false discovery rate. In the sole study of paternal preconception trauma (ACEs), an EWAS found eight medium confidence hits in intergenic regions, TEF, HCG4, CMTM2, KLF1, and APOL2 ((Merrill et al., 2021) . The sole study of grandmaternal prenatal trauma, on F2 grandchild outcomes in Brazil, found no association between intimate partner violence in F0 pregnancy and offspring methylation (Serpeloni et al., 2017). However, grandmothers exposed to community or domestic violence had grandchildren with differential DNAm at five CpGs in CORIN, CFTR, SMYD3, BARX1, and intergenic regions after correction for multiple testing.

Table 2. Comparison of EWAS results

Study	Design	Epigenome-wide findings
Daskalakis et al. 2020	Impact of F0 Holocaust exposure, on F1 PBMC gene expression during adulthood.	Gene expression varied at 42 separate genes; WCGNA analysis indicated down regulation of innate immunity and glucocorticoid signaling.
Hjort et al. 2021	Impact of F0 maternal PTSD due to Kosovo war on F2 whole blood DNAm in middle childhood.	None
Merrill et al. 2021	Impact of F0 paternal childhood adverse events on F1 buffy coat DNAm in infancy	DNAm at 8 CpGs (intergenic, HCG4, CMTM2, KLF1, APOL2, TEF)
Serpeloni et al. 2017	Impact of F0 grandmaternal prenatal intimate partner and community violence on F2 grandchild salivary DNAm in early adolescence.	DNAm at 5 sites (intergenic, CORIN, CFTR, SMYD3, BARX1) for community violence only. No significant hits for intimate partner violence.

Candidate gene results

FKBP5

Methylation of FKBP5 intron 7 (five studies) and exon 1F of NRC31 (4 studies) were the most frequently studied candidate genes (see Table 3). Findings yielded no consistent pattern. In all three studies of Holocaust survivors and offspring in the US, exposed offspring had significantly less methylation of FKBP5 in peripheral blood mononuclear cells (although this varied by CpG site, with site 6 being the most replicated). The direction of this effect was reversed in Grasso et al. 2020, a study of childhood trauma and newborn salivary DNAm in an urban US population, where exposed offspring exhibited significantly greater methylation of four sites in FKBP5, reduced to a single factor through PCA. In addition, this finding was specific to the childhood threat-based adversity score of the Childhood Trauma Questionnaire, with no effects for the deprivation subscale, PTSD qualifying events in childhood or adulthood, or total stressful life events. Finally, in their study of childhood trauma and neonatal umbilical cord blood DNAm in an urban German hospital, Ramo-Fernández 2019 found no group differences in the offspring of women who did or did not report experiencing abuse and neglect before the age of 18.

Table 3. Comparison of FKBP5 and NRC31 DNA methylation results

Study	Design	FKBP5 findings
Bierer et al. 2020	Impact of F0 Holocaust exposure on F1 PBMC DNAm in adulthood	Exposed F1 had lower site 6 DNAm than controls (offspring: mean= 64.87%, SE=0.48; control subjects: mean=67.49%, SE=0.93; F=6.26, p= <.05). Maternal age ≤ 11 at exposure associated with less DNAm (F=6.78, df=1, 114, p=0.010) No associations with F0 PTSD or sex of exposed parent.
Daskalakis et. al 2021	Impact of F0 Holocasut exposure on F1 PBMC DNAm in adulthood	Exposed F1 had lower intron 6 FKBP5 DNAm FKBP5 DNAm (β = -.12, p< .001).
Grasso et al. 2020	Impact of F0 childhood trauma on newborns F1 salivary DNAm	CTQ threat-based F0 adversity scores predicted increased F1 DNAm (ß=.23, p< .05). No effect for F0 intimate partner violence, total stressful life events, PTSD qualifying events in childhood or adulthood, and PTSD symptoms. An interaction where PTSD symptoms predicted DNAm in CC but not CT/TT infants (ß = .41, t = 2.38, p = .024, 95 % CI [.01, .04],
Ramo-Fernández et al. 2019	Impact of F0 childhood trauma on F1 umbilical cord blood DNAm in newborns	No associations between F0 childhood trauma and F1 FKBP5 DNAm

Yehuda et al. 2016	Impact of F0 Holocasut exposure on F1 PBMC DNAm in adulthood	Exposed had lower FKBP5 intron 7 DNAm (7.7% difference) (F =.03, p= .034), which held after covarying for offspring childhood trauma and PTSD. The association became non-significant after the inclusion of parental PTSD.
		NRC31 findings
Christensen et al. 2021	Impact of F0 ACEs on F1 gene expression in children with chronic pain.	No associations between F0 ACEs and F1 NRC31 expression.
Daskalakis et al. 2021	Impact of F0 Holocasut exposure on F1 PBMC DNAm in adulthood	Exposed F1 had lower NR3C1 DNAm (β = -.21, p< .001),
Hjort et al. 2021	Impact of F0 maternal PTSD due to Kosovo war on F2 whole blood DNAm in middle childhood.	F1 exposed to prenatal PTSD (secondary to preconception trauma exposure) but not current PTSD had less DNAm in various sites in NRC31 cg07715663 (β = -0.79); cg21209684 (β = -1.88); cg26464411 (β =-3.42)
Ramo-Fernández et al. 2019	Impact of F0 childhood trauma on F1 umbilical cord blood DNAm in newborns	No associations in for DNAm or gene expression of NRC31
Yehuda et al. 2014	Impact of F0 Holocasut exposure on F1 PBMC DNAm in adulthood	There was no main effect of F0 Holocaust exposure (either maternal, paternal, or both) on F1 DNAm.

NRC31

As summarized in Table 3, three of the five studies that examined NRC31 found hypomethylation in exposed offspring, but they defined parental trauma exposure in different ways that make it hard to compare their results. For example, in an early study by Yehuda and colleagues (2014) exposure to traumatic events (the Holocaust) had no effect on DNAm in NRC31; instead, only parental PTSD symptoms, regardless of Holocaust exposure, predicted of DNAm, although effects were opposing depending on the sex of the affected parent; paternal-only PTSD was associated with hypermethylation whereas PTSD of both parents was associated with hypomethylation, with no main effect for maternal-only exposure. By contrast, in an expanded replication sample of Holocaust survivors, Daskalakis et al. 2021 found exposure to traumatic events (Holocaust exposure) was associated with hypomethylation of NR3C1 but did not report whether this also was dependent on parental PTSD status. Because they did not use controls, Hjort et al. 2021 did not report whether exposure to the Kosovo war itself was associated with offspring outcomes. Rather, they contrasted offspring DNAm between mothers who reported current PTSD, mothers who retrospectively reported PTSD during the pregnancy of F1, and mothers without PTSD, finding that only prenatal PTSD was associated with hypomethylation of NR3C1 in middle-childhood aged offspring. Christensen et al. 2021 found no effect of parental ACEs and offspring NRC31 expression (not DNAm) in their Canadian sample. In their study of maternal childhood trauma and newborn NRC31 DNAm in a German sample, Ramo-Fernández et al. 2019 also found no effect.

Other candidate genes

In the same cohort and in the only study to examine the oxytocinergic system, Ramo-Fernández et al. 2021 also found no association between maternal childhood trauma and offspring

methylation or expression of OXTR, the oxytocin receptor. They noted, however, that maternal and infant DNAm of OXTR was associated, but only for mothers who did not experience childhood trauma ($\beta = 0.56$, $p < 0.001$). Due to their focus on children living with chronic pain, Christensen et al. 2021 examined the relationship between parental ACEs and offspring candidate gene expression in DRD2, COMT, and SERT. In unadjusted analyses stratified by sex, they found no effect of parental ACEs on male offspring gene expression. For female children, parent total ACES ($r = -.264$, $p<.05$) and maltreatment ACEs ($r = .272$, $p<.05$) were associated with DRD2 expression and parental household dysfunction was associated with SERT expression ($r=-.232$, $p<.06$). In another study with sex-specific effects, Pilkay et al. 2020 found maternal history of child abuse was associated with increased NF expression (but not methylation) only in male children ($B=.471$, $p=.001$). By contrast, maternal lifetime fear history (e.g., reporting of degree of fear felt during childhood or adult trauma exposures) was inversely associated with expression (but not methylation) of NF this time only in females ($B = -.123$, $p=.004$). Hjort et al. 2021 also examined site-specific methylation in NF (total number of sites unknown) and found that preconception war-related prenatal PTSD was associated with increased DNAm at five CpGs and decreased methylation at one site (see Table 1.).[6]

Phenotypic relevance

Most studies only measured whether ancestral trauma impacted offspring epigenetics and did not explore whether epigenetic changes resulted in changes in gene expression, cellular function, or other more distal mental and physical health outcomes. Those that did investigate whether DNAm functioned as an "architect" of gene expression and HPA axis function (Bierer et al. 2020, Daskalakis et al. 2021, Yehuda et al. 2016, and Yehuda et al. 2014) broadly supported the idea that DNAm acts as a stable repressor of transcription; hypomethylation of HPA axis-relevant genes was

associated with increased gene expression of FKBP5 or NR3C1, increased basal cortisol levels, and increased in vivo and in vitro glucocorticoid sensitivity. Although Daskalakis et al. 2021 did not find associations between Holocaust-related DEGs and cortisol, they noted that Holocaust-associated GRIP2 expression and a WCGNA module mediated 58.84% of the effect between Holocaust exposure and decreased C-reactive protein in exposed offspring ($p<.001$), potentially underscoring anti-inflammatory effects of improved glucocorticoid signaling. Similarly, Hjort et al. 2021 found offspring DNAm at cg12612985 in NR3C1 to be significantly negatively associated with basal cortisol levels.

Other studies examined whether DNAm associated with ancestral stress was in turn associated with offspring mental or physical health. Christensen et al. 2021 found no associations between DNAm and offspring pain or PTSD symptoms. In their EWAS study of grandmaternal prenatal stress and adolescent grandchild DNAm, Serpeloni et al. 2017 found only one of five significant CpGs was associated with youth depression and PTSD in their Brazilian sample.

Clinical or policy relevance

Several authors emphasized the translational potential for intergenerational epigenetic research to advance precision screening through the development of epigenetic biomarkers that were "objective" and "noninvasive" (Christensen et al. 2021). Noting the difficulty in establishing mechanistic causation in social epigenetics, Merrill et al. 2021 suggested that epigenetic 'biomarkers of risk' might still be of practical use for clinicians seeking to identify parents and children in need of support services. Similarly, Yehuda et al. 2016 underlined their findings contribution to "early detection" of individuals impacted by the sequelae of intergenerational trauma. Similarly, several authors suggested their work may contribute to precision therapeutics, or treatments adapted specifically to the psychobiological processes identified in their study. Pilkay et al. 2020 noted that

epigenetic processes may be helpful in identifying "treatment-resistant PTSD" and suggested that "the more we understand about the effects of maternal trauma experience on infant gene regulation, the more opportunities we can identify to intervene for improved quality of life for mothers and their children." (p.3).

Lastly, several authors noted that findings pointed to the need for investment in social policy to address the underlying social determinants of health that likely undergird intergenerational processes. In their study of grandmaternal prenatal stress and community violence, Serpeloni et al. 2017 de-emphasized precision approaches and suggested universal screening for prenatal stress and public health support for healthy nutrition and tobacco cessation. In one of the only studies to suggest that epigenetic approaches to screening and therapy might be misguided, Ramo-Fernández et al. 2019 noted that rather than seeking to elucidate potential biological processes, clinicians and researchers should emphasize the psychosocial factors already known to lead to poor mental health and physical outcomes.

Study Quality

Methods for assessing risk of bias and other constraining factors in observational, non-experimental, or clinical trial research continue to be developed but no standard method currently exists to evaluate study quality (Bero et al. 2018). Best practices in social epigenetic research continue to evolve; consequently, the present evaluation is descriptive, addressing known confounders in epigenomic studies, correction for multiple testing, and phenotypic validation/plausibility of results.

Six of the twelve studies addressed genetic confounding, of which two included genome-wide and/or mQTL analysis. Serpeloni and colleagues (2017) cross referenced all significant EWAS hits with a known mQTL database and found only gene likely to be impacted and did not include it as a confound in follow-up analyses. Similarly, Merrill and colleagues (2021) searched for mQTLs

post-EWAS and controlled for the presence of an mQTL within10 bp of a significant CpG hit in follow up models. The remaining four studies only examined polymorphisms of candidate genes known to impact methylation levels. Eight studies included estimates of cell-type heterogeneity (either through decomposition-derived principal components, lymphocyte-monocyte ratio, or other undescribed methods). All studies that conducted epigenome-wide analyses included controls for multiple testing through moderate to conservative applications of FDR. In both epigenome-wide and candidate gene studies, controls for multiple testing across numerous hypotheses and various CpG sites (e.g., testing multiple subscales of a trauma measure or continuous vs. dichotomized scores) were infrequent. For example, Ramo-Fernández 2021 applied FDR for site-specific multiple testing, but the total number of hypothesis tests and interactions was uncorrected.

Sample representativeness is challenging to operationalize given the emphasis on traumatic exposures in this body of research, and that exposures are not equally distributed within and across populations. Five studies drew on samples with severe war-related trauma exposure (4 from Holocaust survivors and 1 from Kosovo war survivors). The Holocaust survivor samples are notable in that offspring were all white, predominantly middle-class, and college educated US residents. The Kosovo war survivors (an all-female sample) were of more mixed socio-economic backgrounds. War trauma severity was not assessed in these studies, although several examined offspring and parental characteristics such as anxiety, depression, or PTSD symptoms and/or subsequent trauma exposure in offspring.

Six studies drew community samples with varying levels of trauma exposure. For example, in Merrill et al. 2021's study of the APRON cohort in urban Canada (n= 45), fathers endorsed overall low levels of childhood trauma exposure (M= 1, SD = 1.7). Trauma exposure rates were similarly low in two studies of mothers recruited during pregnancy in Ulm, Germany and Pilkay et al.'s study of childhood trauma in a population representative sample of Shelby County, TN. Two community-

based studies reported higher levels of trauma in their samples. Grasso et al. 2021 drew on a low-income, ethnically and racially diverse sample (n=114) from the Atlanta Grady Trauma Project with elevated rates of lifetime trauma exposure (30% meeting criteria for DSM-IV PTSD diagnosis, 57.3% with a PTSD qualifying event in childhood, 79.1% with a PTSD qualifying event in adulthood). Trauma levels were similarly high in Serpeloni et al. 2017's study of community and intimate violence exposure in Sao Goncalo, Brazil (96% exposed to at least one experience of community violence, and 26% reporting high levels of exposure).

Discussion

Overview

This review traces the emergence of a small but growing subfield of preconception trauma and offspring epigenetic changes. Most studies were published within the last five years, perhaps the first fruits from calls for intergenerational cohorts in developmental science in the earlier part of the decade. Sample sizes were relatively small, perhaps due to the constraining effect of expensive epigenetic microarrays and/or sequencing, with the largest study including 201 mother-infant dyads from the CANDLE cohort in Shelby County, TN. The lack of larger, population-representative samples may be due to the emergent nature of the field, but also potentially due to file-drawer effects where null findings are less likely to be submitted or accepted for publication (Simonsohn et al., 2014). Best practices for epigenetic observational studies are still emergent, but none of the reviewed studies included replication analyses, and strategies for addressing technical confounds were inconsistent. With exception of two studies taking place in Sao Paolo, Brazil and Pristina, Kosovo, most research was conducted in Western, high-income countries. Five of the 12 studies examined childhood trauma and maltreatment as the primary predictor of offspring epigenetic changes; the rest examined exposure to preconception political, community, and intimate partner

violence, though none explicitly examined historical trauma or cultural loss. Several studies included covariates meant to account for the shared environment between grandparents or parents and their children, but no studies formally examined mediation via the parent-child relationship, social inequality, or trauma re-exposure.

The influence and cohesiveness of findings of epigenetic and functional changes in HPA axis outcomes in the adult offspring of Holocaust survivors is striking, although generally not replicated in samples examining other kinds of trauma. In three of the Holocaust survivor studies, exposed offspring had lower DNA methylation of site 6 in intron 7 of FKBP5, decreased in vitro and in vivo glucocorticoid sensitivity, and lower levels of basal cortisol. Later replication studies found evidence that these findings were driven by younger age of maternal exposure, pointing to the plausibility of preconception maternal somatic transmission, effects which remained after controlling for offspring postnatal adversity, psychopathology, and the quality of the parent-child relationship. Taken together, these results support a model of the intergenerational transmission of ancestral trauma via epigenetically programmed HPA axis blunting (e.g., loss of diurnal pulsatility, diminished HPA response to stressors) that has been associated with early life adversity and risk of PTSD and other anxiety disorders (Koss & Gunnar, 2018).

However, other studies examining HPA axis candidate genes did not replicate this pattern of findings, either finding an opposing effect (increased methylation and decreased expression of NRC31) and/or no effect. Other candidate genes (OXTR, DRD2, COMT, SERT, BDNF) revealed interesting sex-specific effects and/or unexpected correlations between methylation and expression. For example, Pilkay et al (2020) found expression of brain-derived neurotrophic factor, a molecule with important functions in neural plasticity and memory formation in the cord blood of male (but not female) neonates was elevated in mothers who experienced child abuse. Total lifetime fear, however, was associated with BDNF expression in females, but not males. In neither finding did

expected correlations between expression and methylation (e.g., functioning as a repressor) emerge. Across studies, none of the significant EWAS hits replicated.

Such lack of replication might be due to methodological differences between the studies and qualitative differences in the populations they studied: this handful of papers examines outcomes in newborns as well as middle-aged adults and exposures ranging from genocidal violence to exposure to common stressors such as parental divorce or substance use. Kirmayer et al (2021) note the limitations of drawing on Holocaust survivor experiences to understand intergenerational trauma in First Nations and Indigenous peoples; re-traumatization, political disenfranchisement, and cultural loss shape the experiences of First Nations families in ways very different than those of Holocaust survivors. Whereas Holocaust survivors experienced mass recognition of the genocide perpetrated against them, a reduction of systematic violence in the postwar period, and (at least in the samples studied) limited intergenerational impact on education and income, First Nations peoples live under ongoing conditions of political disenfranchisement, racialization and discrimination, and cultural loss. Indeed, violent experiences that occur during warfare in communities that do not flee oftentimes live on through stigmatization and discrimination against victims, especially those of sexual violence (Clark, 2014).

To capture the key processes that undergird trauma transmission and resistance more accurately, we urge researchers to recruit representative samples from low income, racialized populations and/or lower- and middle-income countries that accurately capture cultural dimensions of trauma and moral injury, as well as to attend to the heritable social structures that shape generational experiences. Such calls for representativeness and attention to social structure are typical from social scientists working in this field. But it is also possible that the promise of epigenetic research as a mechanistic framework for understanding the intergenerational transmission of trauma is inherently flawed, even with the best of methods.

Indeed, several larger studies have questioned the idea that early life epigenetic programming of the HPA axis or genome-wide methylome via traumatic stress reliably occurs. Although they did not examine preconception effects,(Marzi et al., 2018) found limited evidence for the longitudinal impact of early life victimization on variation in DNA methylation across childhood and adolescence in the 2,232 twins drawn from the population-representative E-Risk Longitudinal Study. Despite their use of a twin design and robust measurement of victimization (including self, parent, home-visiting staff, doctor, and child intervention services reports), they found that the few epigenome-wide associations that did emerge failed co-twin analysis and replication in a second dataset. Along with the unreplicated EWAS, their candidate gene analysis of NR3C1, FKBP5, BDNF, AVP, CRHR1, SLC6A4 also failed to find expected associations.

Two recent systematic reviews also have noted the lack of consistency in the early life adversity and DNA methylation literature, and recommended methodological improvements to address sources of heterogeneity by addressing participant confounding characteristics, aggregating data via consortia, using replication datasets, improving measurement of trauma exposure, studying longitudinal epigenetic outcomes, and more thoughtful interpretation of biological effects in surrogate tissues (Cecil et al., 2020; Parade et al., 2021). The idea that better methods are needed to reveal the utility of DNA methylation as a marker of risk is common, but lack of consistency in findings in early life — and in this review, of preconception— traumatic stress should lead researchers and practitioners to question whether epigenetic epidemiology can in good faith continue to argue its ability to elucidate the mechanisms of how 'stress gets under the skin'. It also begs the question of what ends shining a light on proximal mechanisms has achieved for public health and wellbeing.

The papers reviewed here make a case for the need to 'elucidate mechanisms' of preconception trauma to facilitate precision screening, precision therapeutics, and investment in social policy. These goals may be admirable, yet it must be acknowledged that the translation of early life and preconception epigenetics to screening and therapeutic applications remains a distant goal. In terms of policy implications, tying the need for social investments to small epigenetic effects that replicate poorly may inadvertently undermine public faith that evidence of biological embedding can or should justify social resource allocation. Like research on 'risk' and 'protective' alleles in psychiatric epidemiology, researchers and clinicians must openly communicate that no replicable epigenetic signature of intergenerational trauma has emerged in the 20 years in the wake of (Weaver et al., 2004)'s groundbreaking rodent model of early life stress and epigenetic HPA axis programming. If Weaver et al (2004) foregrounded a quarter-century of research on early-life stress and epigenetics, it is possible that Yehuda et al (2016) has had a similar founding effect in preconception trauma and intergenerational transmission.

In the intergenerational trauma transmission (and early life effects) paradigm, epigenetic changes are assumed to be 1) stable, 2) guide phenotypic expression in predictable ways, and 3) predictive of future developmental trajectories. Furthermore, surrogate tissues (cord blood, peripheral blood, epithelial cells) are typically used as proxies of central nervous system function. Research infrastructure and funding limitations render repeated-measures epigenetic designs challenging, and so cross-sectional or single-time-point designs are common, perhaps leading to associations that represent shifts in tissue investment or developmental tempo rather than cellular reprogramming (Roubinov et al., 2021). However, functional and embryonic research has shown DNA methylation to be dynamic across development, circadian rhythms, and immediately following glucocorticoid exposure (Azzi et al., 2014; Luo et al., 2018; Provencal et al., 2020). These findings have begotten advances in the development of epigenetic 'signatures' of glucocorticoid exposure

using in vitro experimentally derived polyepigenetic risk scores that have demonstrated some reliability to detect dexamethasone treatment in vivo, but their predictive value in revealing exposure to naturalistic stressors and of determining risk of psychiatric or anxiety-related disorders is unclear.

Even if epigenetic signatures of intergenerational trauma were detectable, reliable, and clinically scalable, one may ask how these insights would be used to improve clinical practice and public health poses powerful bioethical challenges. For example, several reports indicated that epigenetic screening might improve the tailoring of treatment to treatment-resistant patients with mental health disorders. Data indicate that this kind of precision approach remains in the distant future; moreover, such arguments elide profound social inequalities in access to mental health and psychiatric care that is culturally relevant and acceptable to participants (Dinwiddie et al., 2013). Also unclear is how clinicians might act on a patient epigenetic profile that differs from their current best practices: trauma-informed treatments that target distress tolerance and emotion regulation are likely more justifiably offered in the context of trauma disclosure and a patient's wishes than epigenetic screening (Grabbe & Miller-Karas, 2018; Muscat et al., 2021). In terms of screening children preventively, epigenetic profiling raises real concerns about pathologizing the neurobiology of children living in conditions of socially malleable intergenerational deprivation and risk. The social epigenetic gambit thus requires that epigenetic changes must be durable enough to be a meaningful indicator and therapeutic target, yet reversible enough to avoid determinism in the tradition of hereditarian behavioral genetics.

We conclude with cautionary observations and recommendations. First, we draw attention to reasons researchers should expect epigenetic changes to be durable and transmissible in the light of evolutionary-developmental and social theory. The notion that epigenetic changes might underlie phenotypic inertia is intriguing but given that epigenetics function as a dynamic regulator of development, it is not likely that cross-sectional studies in surrogate tissues would uncover a true

mechanistic effect. Further, study design should aim to distinguish whether phenotypic inertia vs. perpetuation is occurring. Most studies reviewed here implicitly invoked allostatic load or diathesis-stress paradigms while also suggesting offspring changes might reflect adaptation. We caution researchers to be intentional about their use of the word 'adaptive'. The unambiguous detection of an adaptive signature is a canonical problem in evolutionary research, and such language may invest post-hoc speculation about the meaning of significant results with perceived scientific rigor (Reeve & Sherman, 1993). While most researchers who mentioned germline effects were cautious about its plausibility and mentioned multiple pathways of causation, Ramo-Ferndandez et al (2019) suggested that correlations between OXTR methylation in mothers without histories of child abuse and their neonates exemplified "Neo-Lamarckian" inheritance of acquired traits. None of the studies here were designed to answer questions about theories of inclusive inheritance or Neo-Lamarckism, and we encourage more precise use of theory to shape research questions and methods to render findings more interpretable. Engaging theory about the pacing of development, life-history tradeoffs, and sex-specific effects in the context of future reproductive function also could benefit the rigor of research and potentially shift it from deficit frameworks.

Earlier in this paper, we introduced a model of the intersecting pathways by which trauma experienced prior to conception might be embodied across generations. Epigenetic changes are powerful illustrations of embodiment— they render a metaphor into a concrete, material process that can measured and reported. However, the science of epigenetics and the way they are deployed in research to date calls into question whether the notion of epigenetics as embodiment can make good on its promise to reveal how 'stress gets under the skin' to produce health inequality (Geronimus, 2013). As seen here, few studies engage the social and ecological levels of transmission that are likely to be most relevant in predicting health and well-being. Rather than implicating researchers, this lack of attention to social context is symptomatic of limitations in research

practices— funding, measurement methods, research population access— within which researchers must operate (Evans et al., 2021). We encourage future investigators to consider structural violence and investigate the contingency, cultural specificity, and ecological patterning of trauma even as they interrogate the most proximal processes of embodiment in the epigenome.

Limitations

We note several important limitations to this review. First, our inclusion criteria that trauma exposure be clearly measured before conception removed several studies of perinatal trauma where the precise period of exposure was unclear. For example, work examining culturally-informed indices of political violence and trauma in the Democratic Republic of Congo and newborn DNA methylation of HPA axis relevant genes and BDNF could not be included because preconception exposure was difficult to ascertain (Kertes et al., 2017; Rodney & Mulligan, 2014). Second, our decision to include any measure of preconception trauma (PTSD symptoms, adult preconception exposure, early life preconception exposure) may have compared exposures which are too disparate and should be studied separately. Finally, our decision to include offspring of any age (ranging from infant to mid-life) might also have occluded age-specific patterns. Nonetheless, given the small number of studies in total, we believe this early review to be a useful contribution to an emerging literature and a call for more thoughtful framing of how trauma is expected to be transmitted, and what offspring epigenetic research specifically has to offer families and communities in the wake of trauma.

Table 1. Summary and Main Findings

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Bierer 2020	187, 147 offspring of Holocaust survivors and 40 Jewish controls F1 age: 51 (1.3) F1 sex: 45% female F0 age: not reported F0 sex: 50% female New York City, USA *This study is a replication/expansion of Yehuda 2016	Preconception Holocaust survival Maternal PTSD dx, age at exposure (before or after age 11) Paternal PTSD dx, age at exposure (before or after age 11)	Tissue F1 PBMC Epigenetics ➔ Site 6 of FKBP5 intron 7 DNAm (pyrosequencing) ➔ FKBP5 gene expression Phenotypic measures ➔ In vivo glucocorticoid sensitivity via dexamethasone suppression ➔ In vitro lymphocyte glucocorticoid sensitivity via lysozyme suppression	Covariates: age, sex, study wave, batch, genotype, psychotropic medication use, childhood trauma, lifetime psychiatric dx, lymphocyte/monocyte ratio Exposed F1 had lower site 6 DNAm than controls (offspring: mean= 64.87%, SE=0.48; control subjects: mean=67.49%, SE=0.93; F=6.26, p= <.05). DNAm was associated with increased anxiety (r = 0.169, p< .05) but not childhood trauma, PTSD, parental bonding, or depression. This effect was driven by childhood maternal (not paternal or maternal adult) exposure). Site 6 DNAm was not associated with maternal or paternal PTSD FKBP5 expression was higher in exposed F1(control: mean=0.78, SE=0.15; exposed: mean=1.13, SE=0.06; F=6.82, p< .05); this association did not hold after controlling for F1 anxiety. Site 6 FKBP5 DNAm was negatively associated with basal cortisol (r = - .308, df = 100, p<.05, and r= -.364, p<.001) and dexamethasone suppression of cortisol (r= -.287, p<.001) with weaker glucocorticoid sensitivity in lymphocytes $IC_{50\text{-}DEX}$ (r = 0.22, p< .05, r = 0.33, p< .05).

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Daskalakis 2021	96, 79 Holocaust survivor offspring and 17 Jewish controls in New York City, NY	Preconception Holocaust survival Maternal PTSD[1], age at	Tissue F1 PBMC Epigenetics	Covariates: F1 age, sex, depression, anxiety, childhood trauma, blood pressure, lymphocyte/monocyte ratio Genome-wide analysis found 42 differentially expressed genes in

F1 age: 56 (.9) F1 sex: 68% female F0 age: not reported F0 sex: 50% female	exposure (continuous) Paternal PTSD[1] age at exposure (continuous)	➔ Genome-wide gene expression (Illumina Beadchip 12v.4) ➔ DNAm in NR3C1 exon 1F ➔ DNAm in FKBP5 intron 7 Phenotypic measures ➔ In vitro lymphocyte glucocorticoid sensitivity via lysozyme suppression ➔ Basal cortisol, CRP, ALP, T3, total cholesterol, hgA1c, free thyroxine, gamma-glutamyl transferase, glucose, insulin, TSH, triglycerides	exposed F1 (logFC ranging from - 1.61 to .35, all p<.0001). Most (36/42) were downregulated. F0 PTSD and age at exposure did not significantly correlate with exposed F1 DEGs; the highest correlation between exposure associated DEGs was with younger maternal age at exposure (rho r = −0.10, rrho r = −0.11, n.s.) Weighted co-expression gene network analysis found 1 of 52 potential modules differed significantly between exposed F1 and controls Gene set enrichment analyses indicated down-regulation of innate immune function and glucocorticoid sensitivity. Holocaust exposure related DEGs were not associated with basal cortisol. Exposed F1 had lower NR3C1 DNAm (β = -.21, p< .001), FKBP5 DNAm (β = -.12, p< .001), ALP (β = -.12, p< .001), CRP (β = -.12, p< .001) and T3 (β = -.06, p< .001)). No other associations with plasma biomarkers were found. GRIP2 expression mediated 73.13% of the effect of exposure on CRP after correction for FDR (p<.001). The WCGNA module associated with Holocaust exposure mediated 58.84% of the effect on CRP (p<.001).

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Christensen 2021	86 children and 41 parents recruited from pediatric chronic pain clinics in Calgary, Canada F1 age: 14 (2.3) F1 sex: 64% female F0 age 45 (6.0) F0 sex: 93% female	Parental ACEs (Maltreatment, Household dysfunction) continuous	Tissue F1 saliva Epigenetics ➔ COMT, DRD2, NRC31, SERT gene expression Phenotypic measures ➔ Child Pain Interference ➔ Child PTSD	Covariates: none Expression levels were significantly different by sex, and so all analyses stratified on child sex. For female children, parent total ACES (r= -.264, p<.05) and maltreatment ACEs (r= .272, p<.05) were associated with DRD2 expression. Parental household dysfunction was associated with SERT expression (r=-.232, p<.06). For male children, there were no associations between parental ACEs and gene expression. Parental ACEs were not associated with child pain or PTSD for boys or girls.
Grasso 2020	114 women and newborns recruited at urban prenatal clinics in Hartford, CT. F1 age: 39 (1.1) weeks gestation F1 sex: 44% female F0 age: 27 (5.2) F0 sex: 100% female	Maternal childhood trauma (threat, deprivation, physical violence subscale and chronicity continuous scores) Maternal PTSD	Tissue F1 saliva Epigenetics ➔ DNAm at 4 sites in intron 7 of FKBP5 (reduced to 1 factor through PCA) ➔ rs1360780 genotype	Covariates: None CTQ threat-based adversity scores predicted increased F1 DNAm (ß=.23, p< .05), with no interaction by genotype. F0 PTSD had no main effect on F1 DNAm, but F0 PTSD severity and F1 genotype interacted such that maternal PTSD predicted DNAm in CC infants (ß=.41, p=<.05), but not CT/TT infants. CTQ deprivation, PTSD qualifying events, total stressful life events, physical violence, trauma chronicity, and maternal emotion regulation were not associated with DNAm.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Hjort 2021	118 women who experienced sexual violence during the Kosovo war and 120 offspring in Pristina, Kosovo F1 age: 12.66 (not reported) F1 sex: 45% female F0 age: 43(5.8) F0 sex:100% female	Preconception war-related PTSD (during pregnancy and current, dichotomized)	Tissue F1 whole blood Epigenetics ➔ EWAS (EPIC microarray) ➔ DNAm at candidate genes HTR3A, SLC6A4, FKBP5, BDNF, NRC31, NRC32 (# of sites not reported) ➔ Genotyping (array) Phenotypic measures ➔ Basal serum cortisol ➔ Infant sleep duration and quality (parent-report)	Covariates: F0 age, F1 age, F1 sex, batch, estimated cell composition No significant epigenome-wide site-specific or differentially methylated regions between F1 of mothers with and without current or pregnancy PTSD. Multiple significant site-specific candidate gene associations with prenatal only PTSD (without FDR), all p< .05). HTR3A cg12612985 (β = -0.78) SLC6A4 cg12612985 (β = -0.55) OXTR cg17036624 (β = -2.00); cg03710862 (β = -0.73). cg14483142 (β =-0.98) FKBP5 cg09268536 (β = 1.21), BDNF cg18595174 (β = -1.69); cg20340655 (β =0.68) cg15688670 (β = 1.86) cg04481212 (β = .69) cg04106006 (β = 2.31) cg10022526 (β =.83) NRC31 cg07715663 (β = -0.79); cg21209684 (β = -1.88). cg26464411 (β =-3.42) NRC32 cg1315799 (β = -2.11); cg13373360 (β = 1.33) Basal cortisol was positively correlated with (all p<.05, β not reported): cg12612985 (HTR3A), and cg15688670 and cg04481212 (BDNF). DNAm at cg12612985 (NR3C1) was significantly negatively associated with cortisol.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Merrill 2021	45 fathers and infants drawn from the Alberta Pregnancy Outcomes and Nutrition Study in Alberta, Canada F1 age: 13 (1.0) weeks F1 sex: 51% female F0 age: 34 (4.3) F0 sex: 100% male	Paternal ACES (continuous and dichotomized)	Tissue F1 buffy coat Epigenetics ➔ EWAS (450k) ➔ Candidate genes: DNAm at 332 CpG sites previously associated with early adversity Phenotypic measures ➔ Infant sleep quality ➔ Child externalizing and internalizing symptoms at age 3.	Covariates: infant genotype, cell-type PCA, infant sex, infant age, infant ethnicity EWAS found eight medium confidence CpG site-specific associations for either continuous or dichotomized paternal ACES (all p<.0001). Intergenic: cg12030301 (β_c = −0.04, β_d = −.02); cg13615516 (β_c = −0.08; β_d =−.05); cg02380750 (β_c = -.1, β_d =-.05) HCG4 cg13688808 (β_c =−0.05, β_d = -.02) CMTM2 cg00049664 (β_c = .05, β_d =.01) KLF1 cg23505145 (β_c = -.01, β_d = -.06) APOL2 cg10543947 (β_c = 0.19, β_d =.07) TEF cg26297819 (β_c = .04, β_d =.02) No associations with candidate sites were found. Gene enrichment analysis found meiotic chromosome segregation to be significantly (FDR<.05, p<.0001) associated with EWAS results. Infant sleep (Adj R^2= 0.20, p<.001), maternal postpartum depression (Adj R^2=0.10, p<.05), and paternal BMI at 3 months (Adj R^2 0.18, p<.05), and F1 attention/hyperactivity at 3 years (Adj R^2= 0.08, p<.05), significantly correlated to total paternal ACEs.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Pilkay 2020	201 mothers and newborns drawn from the CANDLE study, a representative birth cohort of Shelby County TN F1 age: 39 (1.0) weeks gestation F1 sex: 45% female F0 age: 26.7 (5.1) F0 sex: 100% female	Childhood and adult trauma exposure, (continuous and dichotomized)	Tissue F1 Umbilical cord blood Epigenetics ➔ DNAm BDNF at cg16257091, cg27351358 (microarray) ➔ BDNF gene expression	Covariates: F1 sex, F1 race, cell composition In combined sex analyses, none of the maternal trauma history variables were associated with BDNF DNAm or expression. In sex-stratified analyses, history of child abuse was associated with increased BDNF expression in male newborns (M = 6.44, SD = .59, p= .001) B = .471. Lifetime fear history (including child and adult traumatic events) was associated with BDNF DNAm in males (B = .004, p= .001), but not BDNF expression. Lifetime fear history was not associated with BDNF DNAm in females, but it was associated with expression ((B = −.123, p= .004). Fear history meditated the effect between child abuse and BDNF expression in females (B = −.22, bootstrap CI [−.52–−.01], R^2 = .19), but not males.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Ramo-Fernández 2019	117 mothers and 113 infants recruited at the time of delivery in an urban hospital in Ulm, Germany F1 age: 40(1.1) weeks gestation F1 sex: 44% female F0 age: (33) (4.2) F0 sex: 100% female	Childhood trauma (dichotomized)	Tissue F1 umbilical cord blood, F0 PBMC Epigenetics ➔ DNAm in NR3C1 exon 1F, FKBP5 intron 7, CRH1 promoter ➔ FKBP5 rs1360780 genotype ➔ NR3C1, FKBP5, CRH1 gene expression	Covariates: F1 gestational age, F1 sex, lymphocyte to monocyte ratio, smoking during pregnancy No associations between F0 and F1 DNAm. No associations between F0 childhood trauma and F1 gene expression or DNAm. No associations between F1 DNAm and gene expression, and no interaction by F1 genotype.
Ramo-Fernández 2021	117 mothers and 113 infants recruited at the time of delivery in an urban hospital in Ulm, Germany F1 age: 40(1.1) weeks gestation F1 sex: 44% female F0 age: (33) (4.2) F0 sex: 100% female		Tissue F1 umbilical cord blood, F0 PBMC Epigenetics ➔ DNAm of OXTR in exons 1, 2, and 3, and introns 1 and 2. ➔ OXTR expression ➔ OXTR rs53576, rs2254298 and OXT rs2740210 genotype.	Covariates: F1 gestational age, F1 sex, lymphocyte to monocyte ratio, smoking during pregnancy No associations between F0 childhood trauma and F1 OXTR DNAm. F0 mean DNAm and F1 mean DNAm were only positively associated for women without childhood trauma history ($\beta = 0.56$, $p < 0.001$). F0 CpG2 was positively associated with F1 DNAm at the same site ($\beta = 0.27$, $p = 0.008$). This association was moderated by maternal childhood trauma history ($\beta = -0.36$, $p = 0.02$). No associations between F0 childhood trauma history, F1genotype, or F1 OXTR expression.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Serpeloni 2017	121 teenage youth and their maternal grandmothers from a low-income urban community with high levels of community violence in Sao Gonçalo, Brazil F2 age: 14(2) F2 sex: 54% female F0 age: 64(8.2) F0 sex: 100% female	Intimate partner violence (IPV) and community or domestic violence (CDV) during pregnancy with F1.	Tissue F2 saliva Epigenetics ➔ EWAS (450k) Phenotypic measures ➔ F2 depression and PTSD	Covariates: F2 sex and age F0 IPV was not associated with F2 DNAm. F0 CDV was associated with DNAm at 5 CpGs: CORIN cg 23275840 (logFC -.30, adjP< .05) CFTR cg21212505 (logFC .33, adjP< .05) SMYD3 cg24478129 (logFC .88, adjP<.05) BARX1 cg05385163(logFC.17, adjP<.050) Intergenic (chromosome 7) cg2668463 (logFC .22, adjP<.05) Only the site in CFTR was positively associated with youth depression (r=.21, p<.05) and PTSD (r=.20, p<.05). Enrichment analyses found overrepresentation of CORIN and CFTR.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Yehuda 2016	Holocaust survivors (n = 32), their adult offspring (n = 22), and demographically comparable parent (n = 8) and offspring (n =9) control subjects, respectively in New York City, NY. F1 age: 46 (8) F1 sex: 72.7% female F0 age: 78 (5.2) F0 sex: 37.5% female	Preconception Holocaust exposure Parental PTSD	Tissue F1 PBMC Epigenetics ➔ DNAm of FKBP5 intron 7 at six CpG sites, binned by proximity into three regions (pyrosequencing) ➔ FKBP5 rs1360780 genotype Phenotypic measures ➔ F1 Salivary cortisol diurnal curve ➔ F1 depression, anxiety, and PTSD symptoms F1 Adversity ➔ Offspring childhood trauma (CTQ)	Covariates: F0 PTSD, F1 depression and anxiety symptoms, F1 age, F1gender Lower intron 7 DNAm (7.7% difference) was observed at bin 3/site 6 in Holocaust offspring than comparison subjects (F =.03, p= .034), which held after covarying for offspring childhood trauma and PTSD. The association became non-significant after the inclusion of parental PTSD. F0 intron 7 bin 3/site 6 DNAm was correlated with F1 DNAm at the same site (r= .441, p= .010). This association was primarily driven by the Holocaust-exposed families (r= .569, p=.005) for Holocaust-exposed compared with (r =.370, ns) control subjects, and not related to genotype. A linear regression confirmed that F0 Holocaust exposure predicted F1 bin 3/site 6 DNAm (β = 2.368, p= .034), including after controlling for F0 bin 3 DNAm (β =2.418, p= .022). Intron 7 FKBP5 average DNAm was negatively correlated with wake-up cortisol (r=- 2.630, p= .005), controlling for age, gender, and current mood/anxiety disorder). There was no association with pm cortisol.

Study	Sample characteristics	Ancestral Exposure	Offspring Outcome	Main findings
Yehuda 2014	95 participants of which a majority had a parent with Holocaust exposure and "a small group of controls" in New York City NY. F1 age:58(7) F1 sex: 70% female F0 age: not reported F0 sex: 50% female	Preconception Holocaust exposure Parental PTSD	Tissue F1 PBMC Epigenetics ➔ Mean DNA DNAm of 39 CpG sites in exon 1F of the GR. ➔ $GR\text{-}1_F$ expression Phenotypic measures ➔ Basal serum cortisol ➔ Dexamethasone suppression test F1 Adversity ➔ Offspring childhood trauma (CTQ)	Covariates: F1 age, sex, smoking, cell type estimate There was no main effect of F0 Holocaust exposure (either maternal, paternal, or both) on F1 DNAm. There was a significant interaction of maternal and paternal PTSD on mean GR-1F promoter DNAm ($F=5.97$, $p=0.02$) (Figure 1). Post hoc analyses found the effect of paternal PTSD was moderated by maternal PTSD, such that F1 with paternal PTSD had higher GR1F DNAm, but F1 with maternal and paternal PTSD had less DNAm ($t=3.49$, $p<.05$). GR-1F DNAm was negatively correlated with GR-1F expression (percent DNAm: $r=-.346$, $p=.003$; number of methylated sites ($r=-.361$, $p=0.002$). Lower GR-1F DNAm was associated with greater cortisol decline following dexamethasone administration ($r=-.249$, $p=.03$).

Chapter 1 Appendix

Search Strategy

Databases	**Keywords**	**Limit**
MEDLINE ALL, Embase, Web of Science Complete, Scopus, and PubMed	Maternal, paternal, early life, childhood, trauma, adversity, violence, intergenerational, multigenerational, transgenerational, stress, child, infant, offspring epigenetic, DNA methylation, gene expression, mRNA	Article Human English 1/1/2000 - 11/1/2021

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Interlude 2

Error

Despite being told it would be better for my mental health, I have not taken Outlook off my phone or silenced its notifications and so I am awakened late at night by a panicked email.

Reviewers want to know how we know all the traumas in the paper were "interpersonal". But wasn't there a kid bitten by a dog in that sample? Will we have to reanalyze? Can you look it up ASAP?

My eyes are dazzled by the data set as I bring up the file. The main file has a count of every trauma, and most researchers just use the summary score variable as a predictor in their regression models. But my file has the item-level data, and I can see exactly which two, or three, or four terrible things happened to the fifty souls in our study

I run frequencies. Everything looks good. Everything is an interpersonal trauma. I sort the file by the summary score. There is a child with only one trauma. I am scared. I was certain we had removed the dog-bite kid.

The child with only trauma only has item 32 marked. This item is for "Other trauma not listed here." In the notes, it says the only trauma this four-year-old had experienced was being separated from his mother at the border and incarcerated without her for a period of three months.

I write the PI back.

We're good.

Chapter 2: Intergenerational trauma and resilience in postwar Guatemala

Note on data sources/collection

In the introduction to this dissertation, I provide an overview of my original study design and plan. The following chapter presents ethnographic and qualitative data from the fieldwork I was able to accomplish prior to leaving the field in March 2020 at the declaration of the COVID-19 pandemic. Because it was gathered for a study I did not end up completing, I do not present or go deep into all the data I gathered during my fieldwork. The table below highlights what data was gathered, and what data is presented in the following chapter.

Table 1. Data overview

Data source	Participants	Status
Biological measures - Hair cortisol - Anthropometry - Salivary DNA	Tri-generational study participants	Collected, not analyzed. I have no plans to analyze these data at present.
Psychometric measures - Trauma indices - Mental health symptoms - Child socioemotional development	Tri-generational study participants	Presented and discussed briefly in Chapter 2, but these are not the focus
Life History Interviews	Tri-generational study participants (grandmothers and mothers)	Collected, analyzed and presented in Chapter 2
Demographic data	Tri-generational study participants	Collected and basic components (income, age, education) presented in Chapter 2.
Focus Groups	Community members and stakeholders	Collected and used as background/contextual material for Chapter 2. Not formally analyzed or written up, plans to do so in future work.
Key informant interviews	Community members and stakeholders	Collected and used for background/contextual material for Chapter 2. Not formally analyzed; no plans to do so in future work as these were not recorded.
Ethnographic fieldnotes	Community members I engaged with in daily activities	Collected and used for background/contextual material for Chapter 2, plans for more formal analysis in future work.

Preface

On a warm, bright day in the summer of 2017, I guided a group of predominantly Maya women in a small community in rural Guatemala in a drawing exercise meant to help them express the hopes and dreams they have for their children. I am there to conduct pilot research for my dissertation project exploring intergenerational trauma and resilience in Nueva Esperanza Chaculá, one of several "repatriation villages" founded by former refugees of the Guatemalan Civil War, a genocidal conflict that has deeply impacted the lives of all the women in the room and indeed everyone in the surrounding borderlands in highland Guatemala. Hoping to help overcome their (and my own) shyness, I tried using a technique I had read about in Jennifer Hirsch's work on intergenerational relationships and hopes in Mexican-American immigrant communities (Hirsch & Philbin 2016). On large sheets of paper, I drew a horizontal line. I asked the four women in the room to draw a picture of their own life, and on the other side of it, the life they hoped their children will have.

I wasn't quite sure what I had hoped for, but as the session went on, I found myself worried that the exercise was not revealing very much I had not already learned from having worked in the community for several year as a volunteer midwifery trainer. The lives women drew for themselves and their children were linked by flowers and signs of nature, and while their "side' emphasized their homes, gardens, and children, the side representing their hopes for their children were very similar, features suns, flowers, and sports, and school. The drawings were cheerful, and women described hoping that their children would be able to enjoy education, exercise, natural beauty, and have large families and colorful homes of their own someday, most indicating that they would prefer it if their children could stay in the community and raise their grandchildren there.

Figure 1. **Focus group drawings**

Only one of the drawings stood out to me, drawing made by a woman I had known for several years, Doña Cheli. Unlike the other mothers, Doña Cheli focused only on her side of the drawing, using gray pencil and no colors. She drew herself encased in a box with no roof, a wiggle on the ground indicating grass or rough terrain. She drew her hair as if it was blown, and while she made eyes, arms, and fingers for herself, she drew no mouth or feet. As we talked, her young son Jairo came up and drew a heart next to his mother, giving her a small yellow mouth. I joked with Cheli that the amateur psychoanalyst in me was curious about her choices in self-representation—but she told me they were intentional. She had meant to represent herself as unable to speak, unable to flee, and trapped.

Figure 2. Cheli's drawing

The next day, I went to her house to talk more, bringing with me the packet of questionnaires and trauma indices I had prepared for my biosocial research study on intergenerational trauma and mental health in mothers and grandmothers in the community. Cheli endorsed experiencing many war-related traumatic events, but by far the most painful part of her story was her recounting of the dehumanization she felt when she was denied the ability to learn to read by her father. There was no item for this in my packet and so I simply coded it under "other". Her body rigid with emotion, she told me of her father's refusal to let her go to even primary school. "My father told me women who learn to write just write letters to boyfriends. That's what he said. Just to boyfriends." She began to weep, describing feelings of being stupid, left behind, and worthless. "Once I was older, I knew that great need in myself, the need to be able to read and write. I went and got school myself. I was so happy when I learned there were teachers for adults, I would work all day and go to school at night. I went and got it myself."

In this chapter, I argue that what Cheli described—her subjective experiences of gendered discrimination and violence—help illuminate how ongoing structural violence in the wake of the Guatemalan Civil War continues to impact the lives of survivors of the war and their descendants today. As such, I advance the argument that, in contrast with biosocial and psychological models of

intergenerational trauma transmission that emphasize behavioral or biological inheritance, intergenerational trauma is better understood as an ongoing process of social reproduction and perpetuation. This chapter links scholarship on intergenerational trauma, the continuum of violence against women and Maya peoples in Guatemala, and anthropological theories of subjectivity in revealing processes of intergenerational transmission and resistance. In it, I analyze how life histories of women in Nueva Esperanza Chaculá reveal the sociopolitical embeddedness and recapitulation of intergenerationally shared experiences of trauma and loss—but also agency, hope, and resilience—from past to present.

Introduction

Intergenerational trauma is understood as process of cumulative wounding transmitted from one generation to another; as a process, it has been used to understand increased rates of mental and physical health in the descendants of Holocaust survivors and First Nations and other Indigenous communities who experience high rates of depression and suicide (Kirmayer et al 2014). Despite its historic connection to discourses of cultural and collective loss and dispossession, research on intergenerational trauma tends locate it within the bodies and behaviors of individuals who are characterized as having poor parenting, inadequate knowledge, or 'vulnerable' biologies (Cerdeña & Rivera, 2021). Influenced by a series of seminal studies of the mental health of Holocaust survivors, much biosocial and psychological research on intergenerational trauma has failed to attend to the recent and ongoing experiences of violence and loss in the Latin America and other non-Western, white, and/or middle-class populations (Sangalang & Vang 2017).

Against the landscape of Central America's broader role as a Cold War battleground, the Guatemalan counter-insurgent war is a conflict known for the extraordinary brutality and terror inflicted against Maya peoples viewed as both sub-human and subversive by a series of military

dictatorships. Covertly supported and overtly unacknowledged by the U.S. and international community until the peak of violence under dictator Rios Montt between 1981-1982, it is also known as the Silent Holocaust. The signing of the peace accords in 1996 heralded a supposed return to democracy, justice, extension of human rights to the Maya, who comprised a majority of the 200,000 people murdered during the conflict. For many of the 150,000 who had fled into refuge in camps in the bordering Mexican states of Chiapas, Campeche, and Quintana Roo, the signing of the peace also heralded the potential for the chance to return. Nearly 15,000 of the 40,000 UNHCR recognized refugees would eventually repatriate to lands purchased by the Guatemalan government.

In the years following the conflict, several researchers investigated its effects on the mental health of those who lived through its violence. In a survey of Guatemalans living in refugee camps in Chiapas four years after the signing of the Peace Accords, Sabin and colleagues (2003) found that a majority met diagnostic criteria for PTSD, with an average of 8.3 traumatic events experienced or witnessed, and high rates of anxiety (54.45%) and depression (38.8%). In a follow up study of Maya who repatriated to Guatemala, they found that rates of PTSD (8.9%) and anxiety (17.3%) dropped significantly, with depression prevalence increasing to 47.8% ((Sabin et al., 2006). More recent epidemiological work has sought to identify the ongoing prevalence of mental health disorders related to trauma exposure 20 years after the war's official conclusion. In a nationally representative sample, Puac-Polanco and colleagues (Puac-Polanco et al., 2015) found that individuals who endorsed experiencing a war-related traumatic stressor had 4.3 adjusted odds of alcohol use disorder and 4.0 odds of PTSD, suggesting generational recovery. However, risk of PTSD, depression, and alcohol use disorder were also higher among Maya and low-income Guatemalans than among the general population, irrespective of previous war-trauma exposure, signaling the re-perpetuation of violence exposure and distress in Maya communities.

The postwar cultural anthropology of Guatemala has been deeply invested in the structural roots of the war and the psychological effects of its brutality and aftermath (Burrell & Moodie, 2015; Hale, 1997). National ambivalence in recognizing the genocide through memorials, collective mourning and repair, inclusion in school curricula as well as the ongoing presence of mass graves and massacre sites in Maya communities instills ongoing cultures of fear, mistrust, and dread (Cecile Rousseau & Drapeau, 1998; Cécile Rousseau et al., 2005). Anthropologists Beatriz Manz, Patricia Foxen and others have documented the violence of everyday life in Guatemala vis a vis the murder of public officials, gender-based violence and femicide, pervasive and racialized economic inequality, and criminal impunity of organized crime (Bellino, 2014; Clouser, 2009; Dudgeon & Inhorn, 2004; Foxen, 2010; Manz, 2008; McSweeney et al., 2017). Many link the fundamental causes of ongoing violence in Guatemala to global neocolonial exploitation of the state, evinced most literally in the 1954 U.S - backed coup d'etat that ended Guatemala's progressive revolution and entrenched autocratic military rule and civil war in the country (Schlesinger & Kinzer, 2005). Guatemala's contemporary structural adjustment policies and regressive taxation schemes are facilitated by a European-descended agribusiness-owning elite that disenfranchises the rural citizenry and creates and maintains the highest level of unequal income distribution (inflation-corrected Gini coefficient) in Central America (Benson et al., 2008; Garcimartín et al., 2021).

The insufficiency of subsistence agriculture, limited job opportunities, inadequate access to healthcare, and barriers to education have resulted in the expansion of labor migration and stagnation of local development (Ferling, 2014). Remittances may be a necessary strategy for families which nonetheless exacts embodied costs (e.g., diminished child nutrition and growth) for the families who become indebted to coyotes and anxiously hope their loved ones make safe passage and find work (Davis & Brazil, 2016). Rather than seeing their loved ones killed or disappeared by paramilitaries, Guatemalan Maya mourn loved ones lost to preventable illness, the dangerous and

inhumane conditions of migration, murder related to organized crime, human, and narco traffic, or execution to silence community activism and resistance of criminal impunity (Briscoe et al., 2010; Hall-Clifford, 2020).

As during the genocide, such experiences do not affect all Guatemalans equally—they are patterned by the intersections of gender and indigeneity. Rape, torture, and the sexual enslavement of Maya women were strategic tools of state violence during the war, echoed in contemporary high rates of femicide and gender-based violence.[6] (Torres 2005). Accordingly, there is an extensive literature on the gendered dimensions of the political, economic, and historical forces that shape experiences of violence and forms of resistance in Guatemala (Nelson 1999, Nelson 2009). This work highlights tandem processes of dehumanization and racialization in which a Ladino ruling class comprised primarily of European descendants cast Maya as backward, subhuman, and undeserving of human rights, political representation, or even life itself.

The work of sociologist Cecilia Menjívar in eastern and highland Guatemala links high rates of femicide and intimate partner violence to a discriminatory legal system that reflects enduring legacies of power inequality (Menjívar 2011; Menjívar & Walsh 2017). In Enduring Violence, Menjívar traces the co-constitution of symbolic, political, and gender violence in lives of Ladina women in the less-studied eastern region of Guatemala. Although situated above Maya women in Guatemala's racialized sociopolitical hierarchy, she reveals how the normalization of violence against Ladina women too is a process rooted in centuries of neocolonial exploitation in Latin America, rather than a series of individual, interpersonal experiences. In applied work, the intergenerational and embodied consequences of the intersection of racialization and patriarchy have been explored by anthropologist Heather Wehr and others in their work on Maya Guatemala women's autonomy

[6] The overall homicide rate in Guatemala has been declining from a high of around 30 per 100,000 to about 17 today; femicides peaked at over 700 in 2009 (Asmann and Jones 2021).

in decision-making about child nutrition and adolescent reproductive health (Wehr & Tum, 2013; Wehr et al 2014). In each case, they find that patriarchal norms shape Maya women's ability to influence decision making to promote intergenerational wellbeing—the decision to allocate family resources to feed a child, or indeed the ability to plan and prevent pregnancy at all.

Subjectivities, violence, and hope

In The Anatomy of Loneliness, Ozawa-de Silva (2021) traces the history of subjectivity within anthropological thought as means of understanding the social construction and phenomenology of inner lifeworlds and emotional experience, on in her words, "first-person experience and the internal structures of body and mind that shape experience" (p.22). She links the rise of theories of subjectivity as a response to the totalizing aspects of Bourdieu's concept of habitus—or the set of learned expectations, affordances, and possibilities that shape our 'predisposition' to action or feeling—as well as anthropologist Clifford Geertz's view of human cognition as inextricable from a web encultured symbolic representation (Bourdieu, 1977; Geertz 1973). Subjectivity thus offers a framework for understanding both the encultured as well as the vivid, individual, and creative aspects of human interior life. Ozawa- de Silva highlights the "janus-like" aspects of subjectivity; it is a theory of subjectification that attends to the processes by which we become subjects and define the contours of our selfhood in response to political economy, history, and culture. At the same time, subjectivity highlights the individually felt, configured, and sometimes transformative nature of our internal emotional experience.

I argue here that subjectivity is a promising lens to use to analyze the processes that shape intergenerational trauma. First, subjectivity asks us to attend to the internal expectations for self and society that an individual carries, and the social processes that shape the internal scaffolding in which we place our selfhood. While Western psychiatric paradigms of trauma emphasize that it arises as a mental 'wounding' in the face of overwhelming fear, it does little to theorize what kind of threats to

the self are most likely to provoke a sense of the self as ripped away from the realm of what was once though possible or acceptable (Lester 2013). Viewed through this lens, intergenerational trauma might be understood shared experience of violent disruptions of the self from an expected way of being in the world. This draws our attention to how expectations for the self are created between generations, and how lived experiences might re-rupture our 'tethering' to society through the inheritance of structural violence. As such, it reorients us away from the inheritance of innate vulnerability towards the inheritance of social disruption.

Another strength to subjectivity as a framework for analyzing intergenerational trauma is its attention to the emotional and expressed experience of such ruptures. The paradox of why certain people fail to develop post-traumatic stress symptoms after life-threatening events while others develop these symptoms after what seem to be less lethal incidents has been addressed by scholars of trauma and moral injury (Luhrmann 2006; Zefferman & Matthew 2020). Moral injury is "profound and persistent psychological distress that people may develop when their moral expectations and beliefs are violated by their own or other people's actions." (Mjolendik et al 2022, p. 1). Moral injury has especially been invoked to explain the feelings of guilt and self-loathing that emerge when individuals are forced to carry out or enact behavior that they find repugnant or wrong; the ongoing or lived aspect of moral injury thus also asks us to think about processes and lifeways rather than isolated events. The circumstances that provoke such injury might differ across individuals and cultures—therefore anthropologists and social psychologists find it useful—but it may also differ across generations. Thus, intergenerational trauma may indeed be shared experiences of moral injury, but different expectations for the self and society between grandparents, parents, and children might also the path and likelihood that such injuries take.

Finally, subjectivity provides a means of more richly understanding intergenerational processes of resilience. Many definitions of resilience have been used by psychologists and anthropologists; I

ground myself in the material sense of Catherine Panter-Brick's definition, "a process harness resources needed to sustain wellbeing" but also consider the internal resources one might harness that are illegible to the eye (Panter-Brick & Eggerman 2017). That is, if subjectivity asks us to understand traumatic processes as the lived experience of rupture from encultured expectations for self and others, it also sheds light on the internal remodelling of those expectations, notions of selfhood as processes of resilience. I choose not to classify my participants into 'resilient' or 'vulnerable'. By turns, every participant is revealed as vulnerable and resilient in this study. Rather, I explore the ways their narratives reveal their subjective experience of socially inherited structural violence and their agency in enacting desires for pleasure, satisfaction and care, and the futures they hope for themselves and the ones they love.

Background: Nueva Esperanza Chaculá

Nueva Esperanza Chaculá— referred to in this chapter as just Chaculá, as its inhabitants do— is a community of about 2000 former refugees of the Guatemalan Civil War and their descendants located about half an hour from the Mexican border in northwestern Guatemala. A deep exploration of the history of the 30-year counterinsurgency and genocide that they fled is beyond the scope of this chapter and is better served by extant robust Guatemalan scholarship as well as historical and anthropological scholarship from the Global North (Falla, 2021; Manz, 2008; Nelson, 1999; Nimatuj & Alicia, 2005). Briefly, Guatemala underwent a revolution between 1941 and 1954 that led to the rise of populist leftist administrations, culminating in the democratic election of Jacobo Arbenz Guzmán. Then as now, Guatemala was marked by the dramatic political and economic inequity stemming from its colonization by the Spanish in the mid 16th century. After declaring independence from Spain in 1810, Guatemala maintained emigration treaties that privileged ongoing Spanish migration to Guatemala, which surged through the 1920's. Spanish-descended landowners claimed and appropriated land that dispossessed much of the indigenous

Maya population; prior to 1944, approximately 2% of the population controlled 72% of all arable land (Trefzger, 2002).

Following the economic unrest of World War II, Guatemala underwent a pro-democracy revolution in 1944 that ousted dictator Jorge Ubico and resulted in the first years of democracy Guatemala had known since colonization, known as the Ten Years of Spring. The heart of the revolutionary politics centered on agrarian land reforms to return cultivable acreage to the majority peasant population (Ferreira, 2012). In 1952, democratically elected president Jacobo Árbenz enacted Decree 900, which expropriated half a million hectares of arable uncultivated land from landowners and distributed them to approximately 100,000 resident Ladino and Maya families (Gleijeses, 1989). In response to a perceived leftist threat at the rise of the Cold War as well as the behest of the United Fruit Company, in 1954, Árbenz was deposed by a U.S.-backed coup known as CIA operation PBSuccess (Streeter, 2000).

The decades of brutal military rule that followed enacted systematic repressive violence against insurgent leftist groups of self-organized Maya and Ladino peasants, activists, and guerilla militants. By the 1980's the insurgency was largely organized under the Marxist Guatemalan Nationalist Revolutionary Unity (UNRG) guerilla, with strongholds in the mountainous western highlands. In 1982, a military coup installed General Efraín Ríos Montt to power; the most severe crimes against humanity of the war would occur in the years between 1982 and 1990. Montt dissolved the legislature and installed martial law, convicting and disappearing dissidents in a tribunal system that grew to structure the authoritarian surveillance and violence of daily life within Guatemala. Military death squads were supplemented by fratricidal Civil Defense Patrols, to which villagers were forcibly conscripted and compelled to participate in violence against their neighbors and kin (Remijnse, 2001). The violence resulted in the forced migration of approximately a million and murder of more than 200,000 people, largely the Maya peasantry (Commission for Historical

Clarification, 2012) . The brutal, de-humanizing tactics of the counterinsurgency emphasized the disruption of kin networks, gender-based and sexual violence, and the use of public torture and execution as means of terror (González, 2012).

The violence of the 1980's included whole-village massacres in areas perceived to be controlled by the UNRG (Menchu, 2010). Steinberg and colleagues' ((Steinberg et al., 2006) maps of documented massacres show their concentration in departments of Quiché and Huehuetengango, affecting K'iché, Akateko, Ixil, Q'anjob'al, and Chuj populations among other ethnolinguistic Maya groups (see Figure 1). Many villagers who experienced or heard of massacres nearby fled into the dense mountainous forests of the area; those who lived in areas relatively close to the border with Mexico fled there in hopes of finding asylum. A minority of these refugees (approximately 46,000) would come to be recognized by the United Nations High Commissioner for Refugees and settle in camps throughout the Mexican state of Chiapas (Fagen & Yudelman, 2001). About 300[7] of these refugees would become the founding families of Nueva Esperanza Chaculá, one of two early repatriation efforts in which the Guatemalan government provided land grants and organized resettlement to those expelled by violence.

[7] Figures as to the founding families and residents of Chaculá vary, depending on the source. The numbers I heard from Chaculenses were closer to 200 and may reflect the conflation of "founding family" with "socio". Not everyone who came to Chaculá ultimately was granted sociedad in the cooperative.

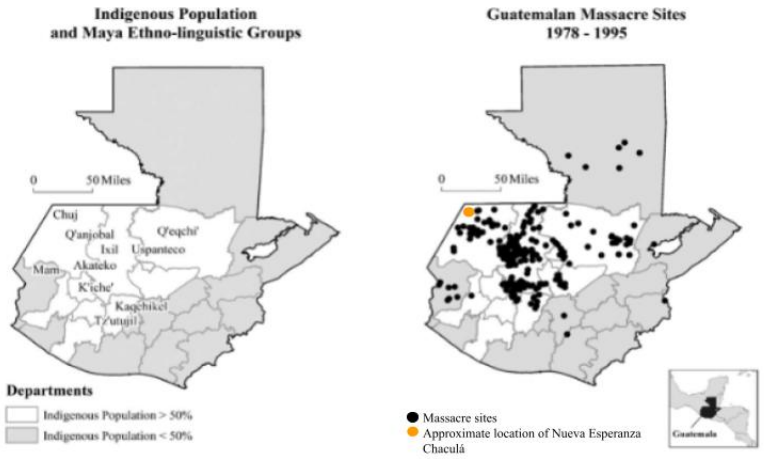

Figure 3. Location of Chaculá. Adapted from (Steinberg et al 2006, p. 65).

Known as retornados, Chaculá was founded on January 12, 1994, on the expropriated finca of an absentee landowner about twenty minutes from the border with Chiapas in the municipality of Nentón. The founding families of Chaculá (and their current residents) reflect the ethnolinguistic diversity of the communities who lived through the repression and violence of the conflict. Most of the families in Chaculá identify as Chuj, Mam, Popti, or Jakaltek Maya but about a third identify as Spanish-speaking Ladino, or as expressed in interviews sin idioma, "without language". After their experience of exile, most Chaculenses are conversationally fluent in Spanish, and the second generation of children born in the camps and raised in the community are heritage Maya language speakers but primarily communicate in Spanish. The 1994 repatriation occurred before the official end of the armed conflict and signing of Peace Accords in 1996; the process of return— the land-grant for the community and initial infrastructural support— itself was part of efforts of the Guatemalan government's efforts to re-gain global legitimacy following international condemnation of the genocide.

Accordingly, many Chaculenses feared reprisal and violence upon repatriation. At its founding, Chaculá was supported by multiple NGOs— UNHCR, Witness for Peace, The Rigoberta

Menchú Foundation, SEIBA International, Planned Parenthood and others, although they have all largely ceased working in the community. Connie Vanderhyden was one of the American human rights accompaniers sent by Witness for Peace to help ensure the safety of and document any human rights violations against the community. She went on to found a small NGO, KGAP, and it remains one of the few active nonprofits working in the community today. Connie would become my mother-in-law, my suegra, and in 2012 I began joining the annual delegation to Chaculá, working as an interpreter for a grassroots funded midwifery training program supported by the KGAP. My ties to the community are rooted in this relationship, one that is resonant within the themes of kinship and the roles of daughters-in-law that this chapter explores.

Chaculá was founded as a cooperative, originally conceived as a lumber enterprise Cooperative Los Pinos, referred to simply as la cooperativa. Cooperative membership involved shared land and resource ownership distributed through sociedad. Each founding family nominated a head socio who would vote on any collective agreements, negotiate collective labor duties, and receive and distribute any cooperative profits. Equal numbers of men and women were nominated as socios, to codify gender equality into its political structure, but only one child can inherit the title and voting rights. Land was not individually titled; families could receive homes and other goods funded by NGOs, but these were negotiated to be equal between socios, and not transferable. Furthermore, the original sociedad involved a start-up investment of 500 GTQ, something some of the poorest families were unable to provide, and they became avecindados, members of the community without cooperative ownership or representation but who retained communal social rights. Rules around land entitlement and transfer have long been political touchpoints in Chaculá. Currently, community members cannot sell their homes, small agricultural plots, or wood-collecting rights to 'outsiders', but they are permitted to do so within the community. Over time, this process has led to the accumulation of cooperative land by a small number of families, as other liquidate

assets to raise capital for medical expenses, funding the immigration of a family member, or sending a child to school.

La cooperativa is headed by a socio-elected junta and has numerous subcommittees and projects meant to raise collective funds. These include a dairy and an agrotourism program. Chaculá's physical construction is different from the surrounding villages and reflects the centralized planning and influence of the Global North NGOs that funded its construction. Unlike the spidery, circular pathways and roads of neighboring Chuj villages, Chaculá is a grid of uniform gravel roads, separated into five barrios. At its inception, socios each received an equal sized village plot, cement-block house, and access to water, wood-collecting rights in the forest, and agricultural plots about a kilometer away. During la cooperativa's founding, each nuclear family nominated a socio to represent their interests in the cooperative; socios in turn decided that sociedad could only be inherited by one of the socios children, not all, and that this new socio would have to negotiate the sharing of family assets between siblings. Being a socio involves certain duties. A small start-up investment several hundred quetzales was required of each of the founding families, and socios must either provide or pay for labor in the trabajos comunales, community building projects such as the digging of drainage and building of roads. Those who could not give their part risked losing their socio status and land rights, although they would be allowed to remain in their homes as residents of the community or avecindados.

Originally, equal number of men and women were nominated as socios, but the scheme to inherit only one child and require manual labor has favored the ongoing participation of sons and able-bodied men. For example, I spoke to several women who reported losing socio status when their husbands left them, and they were unable to keep up their labor duties. With several children to choose from and a tendency for women to co-reside with their husbands after marriage, a similar bias towards inheriting sons is also present. Over the 25 years since its founding, questions about

how children inherit and maintain the cooperative structure have strained village political relations. A key feature of these disputes is the distinction between cooperative and individual property. The current legal status of the land agreement in Chaculá stipulates that while Chaculenses (of socio status or not) may sell their home or land to other members of the community internally they have no rights to formally entitle their land and sell it to outsiders. Many families choose simply to build additional small homes on their family plot to accommodate offspring and their families, while others lobby for the ability to own and sell their lands that their children may inherit financially and allow them to move to new parts of Guatemala or to emigrate.

Figure 4. Arial comparison of Chaculá (A) and Yalambojoch (B)

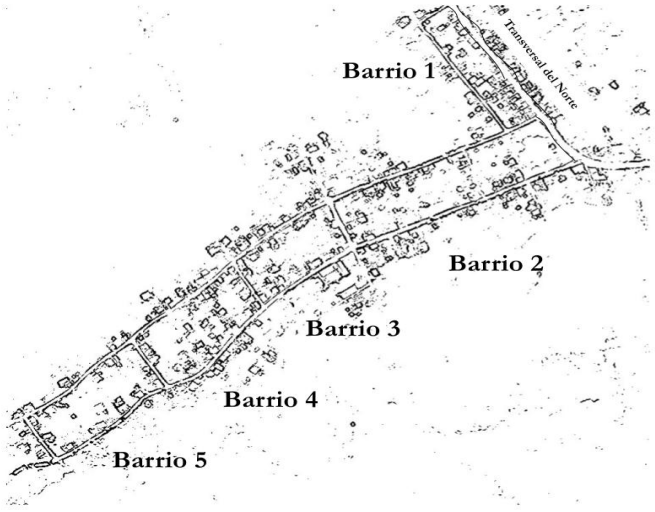

Figure 5. Map of Chaculá
Each of the community's five barrios or neighborhoods are indicated on the map. These form the community subunits that provide representation to COCODE and some communal labor (e.g., public safety). Barrio 1 is the wealthiest and is made up primarily of Jakaltek and Ladino residents. Barrio 2 is made of a similar mix of ethnicities, with some Mam families. Barrios 3 and 4 are mixed-ethnicity with Mam, Q'anjobal, Jakaltek, and Ladino residents. Barrio 5 is considered the poorest barrio and is made up primarily of Chuj residents, who make up much of the surrounding municipality.

the actual politico-legal organization of the village conforms to a top-down participatory governance system known as COCODE (McNulty, 2015). In this system, locally elected community councils are responsible for day-to-day administration of the community including safety, maintenance of law and order, and coordination of basic services such as the national health post, access to water, and electricity. Unlike la cooperativa, COCODE functions as a direct democracy, in which all community members are invited to collective meetings to discuss initiatives and collectively vote on community rules, ordinances, fines, and penalties. COCODE also forms Chaculá's link to the broader systems of governance in Guatemala. While many anthropological perspectives on Maya community organization, systems of mutual aid, direct democracy, and indeed the CODODE

systems emphasizes their resistance of neoliberal citizenship (Hale, 1999), the perspective that emerges from Chaculenses themselves is one of awareness of growing socio-economic inequality and stratification stemming from how la cooperativa was founded and its evolution over time.

Socio status and relationships are important determinants of material wellbeing and political power; they enable access to land use for agriculture, home-building or expansion, and influence in the decisions made by la cooperativa. Despite this, in my fieldwork, I often inquired about who the socio living in a particular household was and was surprised to that often it was no one. I do not remember the first time someone joked and asked if I was looking for a socio—or a sucio. Sucio translates to "dirty". It was invoked oftentimes as a joke in everyday language in groups where either no one had a close socio relationship (e.g., their sociedad had been taken away or belonged to a family member who didn't closely support them) or when talking with a new kind of avecindado—literally "neighbors" —people who had moved to Chaculá but had no blood kin claim to any socio. Such avecindados could be people who married into the community, or more often domestic or agricultural workers who lived with socios and their families and cared for their children, washed their clothes, cleaned their homes, and harvested their fields.

The low wages and limited opportunities for women of the region make domestic labor (including live-in domestic labor) a viable lifeway between and within many communities in Guatemala and in Central America. And despite the disinvestment, poverty, and precarity that many Chaculenses live in, relative to neighboring communities Chaculá's high levels of primary and increasingly secondary education and Spanish proficiency mean that an emergent group of higher-earning Chaculenses can afford to pay for domestic labor in their own homes. These new avecindadas of Chaculá came from nearby villages and would often have distal ties to community members and socio families. Community members and leaders told me the population of domestic

laborers had crept up over the years, and once Chaculenses began emigrating to the U.S. and sending wages back, the cash and informal labor economy had expanded quickly.

Unlike other communities of the region where even low school fees are a barrier to education, especially for women and girls, fees from primary through middle school are covered by an NGO-funded lottery system run by the community schoolteachers. Each family can nominate a child for a scholarship, and increasingly fund scholars' attendance in high schools/occupational training programs. While the lottery system is imperfect, and many families report that they were never able to receive support, a large proportion of children attend school at least through sixth grade. After schooling, men and boys immigrate frequently to Mexico or the United States, while women tend to remain in the community or make shorter migrations to Mexico where they oftentimes carry dual citizenship and the liberty to work. Those who can obtain training as teachers, accountants, or nurses tend leave the community to find employment in the surrounding region.

As a multi-ethnic community, Maya cultural norms and practices vary across the different families. For example, post-marital residence patterns and family living arrangements generally favor multiple-family housing on the same plot of land, but patterns of patrilocal, matrilocal, or neolocal residence after partnerships depend on family resources and contexts (Ensor, 2013). At its founding, the majority of Chaculenses were Catholic, and a large two-story church with a bell-tower is located at the entrance of the village. Those who practice Maya religions do so within their own homes and I only rarely observed people engaging in these practices and no organized Maya religious worship site exists in the community. While most families describe having ties to Catholicism at some point in the past, a proliferation of Evangelical Protestant churches have cropped up in the last ten years, which vary from Seventh Day Adventists, Charismatic, Baptist, Jehovah's Witness, and various other denominations.

Maya identity, language, and cultural practices are complex and layered in Chaculá, but at a glance it may appear that its culture is more Ladinized relative to the surrounding communities. Less than a mile up the road in Aguatcate or Yalambojoch, the morning announcements that blare across community loudspeakers are in Chuj, and wear most women wear their traditional dress of a corte skirt, backstrap-loop woven colorful faja girdle, and complex, symbolically rich huipiles. By contrast, Spanish is widely spoken in Chaculá; all announcements and communal business are conducted in Spanish, and Maya languages are primarily spoken in homes and families. While elder generations continue to wear corte and huipiles, it is rare to see women under the age of 40 (unless they are from surrounding towns) dressed in traditional Maya dress. Instead, they wear leggings, pencil skirts, and colorful blouses that echo the silhouette of traditional dress, but which are procured from the weekly visit of a salesman who brings recycled clothing from the United States and sells it by the quetzal.

During a visit to Yalambojoch, a local midwife Ana told me the family conflict over her niece attending high school in Chaculá, where the perception is that education is higher quality. "Her mother told her she could go but begged her not to give up her corte and dress like those girls." Ana expresses fears that others have shared with me about Chaculá—that it is a place of potential cultural incompatibility or contamination—but also that its residents think of themselves as better than their neighbors.[8] Indeed, part of the reason that Chaculá is perceived as Ladino is also because many people who identify as Ladino live there, but strong political and cultural connections to Maya identity are commonly expressed by the Chaculenses. Rather than a rejection of Maya identity, the linguistic and sartorial differences seem to be related to the afterlife of the cultural mixing that happened in refuge and contemporary economic conditions that erode traditional Maya lifeways. For

[8] These conflicts can be deadly; Aguacate and Chaculá have a long-standing dispute over water rights and many community members told me stories that they feared their neighbors had contaminated the water supply on purpose

example, Chaculense multi-ethnic marriages have resulted in new mixed-language families where new parents might prefer to communicate in Spanish and teach Spanish to their children, even as their grandparents speak to them in Chuj or Popti. The reduction in traditional dress is oftentimes attributed to the high cost of hand-woven clothing and relative abundance of cheap Western-style clothing. Distance from natal communities (e.g., Mam, Q'anjobal, or Quiché communities are located more than a day's travel away) means that markets for one's own traditional dress are harder to come by, becoming prized heirlooms or special occasion dress rather than everyday wear.

As in other areas of highland Guatemala, machismo suffuses gender norms, and women's freedom to engage in premarital sex, deny unwanted marital sex, and access birth control is limited by patriarchal norms around women's modesty, purity, marriageability, and subservience to male physical, reproductive, and economic power (Nelson, 1999). Family decision making about financial investments and decisions remains with men, even in families where the socia is a woman. A shift from agricultural subsistence to U.S.-based immigration has shifted these power dynamics. While in some senses granting women new autonomy, surveillance of their activities by other family members against untoward behavior or suspected infidelity maintains entrenched patriarchal norms (Taylor et al., 2006). Women who achieve higher levels of education and professional employment as nurses or teachers, or who build successful businesses, describe feelings of increased autonomy. However, they also report that these activities also come with costs; increased surveillance, partner jealousy, and gossip about their movement in the world and potential for infidelity or impropriety are also part of their everyday lives.

Methods

Data Collection

Ten months of non-consecutive in-person ethnographic research took place between 2017 and 2020[9]. Broadly, data collection took the form of an ethnography of everyday family life in Chaculá with a nested semi-structured psychobiological study of intergenerational trauma in grandmother-daughter-grandchild triads. Data sources and collection methods are reviewed in Table 2 below. Living with a host family in the community, I shared in everyday family life (meal preparation, childcare, shopping, household tasks) in a woman-headed, tri-generational household while also conducting participant observation at community meetings, the primary school and reproductive health/prenatal care clinics held in Chaculá and the surrounding villages of Yalambojoch and Bulej. I also conducted formal in-person key informant interviews to learn about the community's history, current challenges, local models and mechanisms of intergenerational trauma, and stakeholder priorities for the research goals and aims. I held four focus groups to help develop and culturally adapt the questionnaire set and interview guide for a nested psychobiological study of intergenerational trauma in grandmother-daughter-grandchild triads. Semi-structured interviews for the psychobiological study began after formal community review and approval in December 2019.

Key informant interviews

Thirteen key informant interviews were conducted in-person and lasted between 1 and 5 hours, some occurring over multiple days. Key informants included current and former leaders of the cooperative junta directiva, the local COCODE community council, leaders of the local primary and secondary schools, health clinic coordinators and health promoters, feminist activists, traditional healers, and midwives. These interviews were semi-structured and tailored to the perspective of each informant, but broadly covered the political, economic, and social histories and realities of

[9] I conducted several interviews over Zoom in the fall of 2020 to continue data collection but found that these were ineffective and technically difficult. None of those interviews are included in this chapter. I also excluded one field site interview of a woman who had severe difficulties speaking and whom I also struggled to comprehend.

Chaculenses and the impacts of psychological trauma on health, wellbeing, and child development. These interviews were not recorded. Notes were taken on paper (including quotations) throughout the interview and written up in daily fieldnotes. I systematically reviewed key informant interviews and fieldnotes to construct a composite history of the community, characterize its social and political organization, and better understand the sociopolitical dynamics of family life. For stakeholders in the local governance, schools, and clinic, these interviews also served as a means of understanding community priorities and needs for the research. Key informants were selected for their leadership roles and specialist knowledge of the community.

Focus Groups

Four focus groups were held during the research period. Two occurred during preliminary research visits: one group of primary school teachers and one group of four local mothers and grandmothers. These exploratory groups were guided to reflect on local concepts and mechanisms of intergenerational trauma, and how trauma has impacted families, children, and the community. The group with mothers also completed drawings of the lives they lived and those that they hoped their children could live. These focus groups were recorded and transcribed by a professional transcriptionist. I systematically reviewed the transcripts, drawings, and my notes from the sessions to characterize the cultural context of the community and to develop open-ended questions for the semi-structured psychological interview with grandmother-daughter dyads.

Two focus groups of five different grandmothers and mothers were conducted in October 2019 prior to the beginning of the semi-structured interviews to assess the appropriateness and validity of the questionnaires included in the final research interview. These focus groups were not recorded. During these focus groups, I read each item of each questionnaire and encourage participants to discuss what they thought it meant and how they might express the idea in their own

words. I took notes throughout the session and reviewed these with my research assistant to make changes to the final set of questionnaires and constituent items to be included in the research interview.

Grandmaternal-maternal-grandchild interviews

Semi-structured life history interviews broken into three different interview sessions were conducted with 28 grandmaternal-maternal-grandchild triads. Grandchild data were collected via maternal report, and grandmothers reported on the early lives of their adult daughters. 18 families provided full interview data, with the remaining ten either providing maternal-only (n=9) or grandmaternal-only reports (n=1). Recruitment was conducted via announcements at community meetings as well as an announcement transmitted over the community loudspeaker system; interested participants contacted me or my research assistant directly via telephone or by stopping by my home or office. Interviews were conducted by me in Spanish in a small private research office in the community. Inclusion criteria included biological mothers of any age with children aged 3-9 who spoke Spanish. Mothers were asked to share my contact information with their own biological mothers and to request they contact me directly if they wished to participate. All participants engaged in an informed consent process that was repeated during any follow-up interviews. I consulted with community stakeholders (teachers, healthcare workers, COCDE members) to determine what an acceptable compensation would be, and it was agreed 70 GTQ (10 USD)[10] for the full set of three interviews was appropriate. Interviews took between one and ten hours and occurred over several days. Interviews were recorded and transcribed. All questions were read aloud to participants and visual aids were used to assist in answering with Likert-like scales. All study

[10] An additional 30GTQ was paid to mothers who brought their child in for the biological samples collection visit, not described in this chapter.

procedures were approved by the village council (COCODE) and ratified by the community in a large public meeting as well as approved the Institutional Review Board at Emory University.

Table 2: Data Sources

	n	Description
Key informant interviews	**13**	Ethnographic key informant interviews were conducted to learn about community history, political-economic structure, social concerns, and understandings of how collective trauma has impacted the community. Key informants included current and former cooperative leaders, primary and secondary school directors, coordinators of the local health clinic, feminist activists, midwives, and traditional healers.
Focus Groups	**4**	Two focus groups were held in summer of 2017 to learn about local understandings of intergenerational trauma and discuss and receive feedback about research aims and goals. The first focus group included mothers and grandmothers, and the second focus group was composed of local teachers and healthcare workers. Two focus groups were held October of 2019 with 5 local grandmothers and mothers to assess the quality of translations and local understanding of measure items in the research interview.
Grandmother and daughter interviews	**28**	Semi-structured interviews were conducted with grandmother-mother-child triads where mothers reporting on themselves, and their young children and grandmothers reported on themselves and their daughters.
Participant observation		Two visits in 2017 and 2018 to conduct midwifery education with KGAP, lasting approximately two weeks each. One month of pilot research conducted in the summer of 2017. Three days of prenatal visits, approximately 10 patients per day. Four community-wide meetings and one emergency community meeting. Eight months of everyday experiences in the community and daily field note taking and weekly field note organization and analysis.

Descriptive data analysis

Demographic, trauma and symptom inventories were entered into an encrypted REDCap database and descriptive statistics and item frequencies were assessed in R statistical software. Notes taken ?during discussion after the life stressor and war trauma items were consulted to aid in additional description.

Qualitative data analysis

Qualitative data was analyzed in several stages. Only the life history section of the interview was recorded and transcribed. I conducted all coding myself in the original Spanish language; quotations here are my own translations. My coding was guided by the approach for thematic analysis suggested by Braun & Clarke (2006), which reflexively acknowledges the role and goals of the coder even as codes and themes 'emerge' from the data. As a first step, I conducted open coding of transcripts using MAXQDA software. Throughout open coding I kept code memos and notes highlighting linkages between the expressed processes of intergenerational transmission. The first pass of coding produced 228 individual content-related codes. I then reviewed these codes and grouped them into related areas, generating a candidate list of themes. Returning to my transcripts, I conducted a second pass of transcripts, to determine whether the candidate themes fit the data, whether there were missing aspects or codes, or whether a theme would be better broken into two separate themes. I then produced a refined final list of themes. I provide a sample codebook, full list

of themes, and overview of the theme generation process in the Appendix[11]. Finally, in order to focus on intergenerational processes rather than consider each individual account separately, I placed each mother-daughter transcript pair side by side and considered how the themes identified in each of their accounts reflected their shared experiences in ethnographic and life history context.

During my reading, I asked myself— which narratives evince these themes? Which resist them? Where do agency, resilience, and desire reveal themselves in narratives and what can they tell us about intergenerational trauma and embodiment? Following this analysis, I selected interview excerpts and ethnographic vignettes that illuminated these processes, which are also presented below.

Representativeness of the sample and positionality of the author

I wish to note here I do not (and would not) claim that the women[12] who came to speak with me in this study are representative of the experiences or mental health in the community as a whole. Each of their experiences matter and reveal important processes that undergird the inheritance of structural violence and the social nature of resilience. I have strong reason to believe that the women who came to talk to me may have done so out of a desire to talk about the challenges they had lived through. This was revealed in things they told me, but also in the communal understanding of my purpose in Chaculá. Women occasionally came to my research office without wanting to sit for the full interview, simply wanting to talk about the problems of life they experienced, concerns they had for their children, or unmet physical or medical health needs. Early in the fieldwork, an elder woman came to my office and asked if she could talk to me about how sad seeing her husband with his new wife made her. She spoke about her feelings of romantic

[11] As Rachel says, a 'firehose' of data. Many of the themes would be meaningful to go into in future work, and I'm excited to continue working with this material. The more abbreviated approach here is meant to capture the elements I thought most relevant to questions of intergenerational transmission.

[12] I say 'women' here because I explicitly asked the gender identity of my participants. All identified as cisgender women, so I use this throughout the text.

rejection, and the pain of seeing him dancing with his new partner at community festivals. After she had finished, she smiled and thanked me, saying. "Ya me desahoge." Ahogarse means to drown; desahogarse literally means to "undrown" but is an idiomatic term for unburdening oneself or venting. Its metaphoric links to suffocation remain vivid for me. The women who came to my study, for many reasons, came seeking a space where they would not be ahogadas or silenced. They sought a space they could breathe and speak— or even scream. The data presented in this chapter should be understood as resulting from participants enacting those desires.

Women also showed up for my study because social connections to human rights and NGO workers are known to be advantageous, and I provided knowledge and material resources[13] within my means to all my study participants (as well as any other community members who asked). This included things like researching U.S. detention contact procedures and numbers to locate lost family members, assisting community members as migrant family members travelled from the border to their ultimate destinations in South Carolina and Florida[14], providing transportation, patient advocacy at doctor's visits, and researching and coordinating support for many social needs with high administrative barriers, such as obtaining documentation of Mexican citizenship or accessing state services. My interlocutors made good use of the knowledge capital and mobility that I brought, and their motivations for speaking with me undoubtedly included this.

My positionality also influenced what kind of information and experiences my interlocutors shared with me. I am a light-skinned Puerto Rican woman with American citizenship and

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[13] By "material resources" here I mean car-rides, shared meals, use of my telephone and computer, and childcare. Outside of participant reimbursement, I did not provide personal cash to participants. I have and continue to financially support close friends in the community who did not participate in the study when they have need, such as for educational, healthcare-related, or legal expenses.

[14] Most migrants from Chaculá get picked up at the border in Texas and driven to Atlanta, where they will find their own way to the restaurants, construction companies, meat processing plants, and farms in South Carolina and Florida where they find employment. The transfer in Atlanta is dangerous; sometimes people get extorted and dumped by coyotes, and knowledge of the city, its neighborhoods, how to move around, as well as my social contacts was highly desired by participants.

postgraduate education, a member of what economist Richard Florida calls the "creative class" in postindustrial societies (Florida, 2019). My socioeconomic position is marked by transnational mobility, relative economic security, and most importantly a perceived entitlement to creatively produce knowledge and innovations as the primary means to my sustenance. The women who spoke to me were very aware that we inhabited different lifeworlds and privileges, and I acknowledge what they said exists as what they chose to say, to me, in that room.

Along with informing the exchange of information in our interviews, my positionality inflects my analysis. I come from a similar culture of machismo and have lived cycles of family violence tied to women's economic and political power. Regardless of this lived experience, my academic analytic lens is rooted in a lifetime of education in white feminism, what Mariana Ortega diagnoses as a kind of "loving, knowing ignorance" (Ortega, 2006). My own whiteness, Americanness, Westernness, and privilege mean that my interpretation of my interlocutors is biased by a culturally-specific sense of what female empowerment can and should look like (Mahmood, 2011).

Results

Psychometric measures and trauma indices

Descriptive Statistics

The table below provides descriptive statistics of the measures used in the study for the mothers (n = 27) and grandmothers (n = 19) who completed the interview and questionnaires. For scale and demographic data, means and standard deviations are presented. For traumatic event count data, means and ranges are provided.

Table 3. Descriptive, Trauma, and Psychometric Statistics

Measure	**Grandmothers n=19**	**Mothers n=27**	**Children n= 27**
Demographic characteristics			
Age	48 (8.2)	30 (3.2)	4.8 (2.3)
Monthly Income	911.1(714.9) GTQ 118.5 (92.8) USD[15]	1418.0 (1108.8) GTQ 184.34 (143.82) USD	n/a
Years of education	2.6	6.0	n/a
Trauma Exposure			
War Trauma (Harvard Trauma Questionnaire) 46	8.1 (0-19)	n/a	n/a
Life Stressors (Life Stressors Checklist Revised) 34	16.1 (12 - 21)	15.1 (4-24)	n/a
Childhood Trauma (Traumatic Events Screening Inventory, Parent-Report-Revised) 24	n/a	n/a	4.0 (0-7)

[15] I provide estimates based on current exchange rates for GTQ to USD to help interpret the relative poverty participants lived in. The weakness of the Guatemalan quetzal does not translate to lower prices for goods in everyday life. At the time of writing, a factory-farmed chicken in Chaculá costs around 30 GTQ, a packet of crackers cost about 4 GTQ, and a roundtrip bus ride to the largest municipality (Nentón) cost around 25 GTQ. While few people pay formal rent, life is still very expensive relative to income.

Depression Symptoms (Hopkins Symptom Checklist)	2.4 (.8)	2.3 (1.0)	n/a
Anxiety Symptoms (Hopkins Symptom Checklist 25)	2.3 (.8)	2.5 (.9)	n/a
Child Socioemotional Difficulties (Strengths and Difficulties Questionnaire)	n/a	n/a	14.67 (4.4)

Grandmaternal and maternal trauma and mental health symptoms

Grandmaternal and maternal anxiety and depression symptoms were assessed with the Hopkins Symptom Checklist - 25 (HSCL-25) which has been adapted and used successfully in previous studies of trauma exposure and mental health in Guatemalan refugees (Mollica et al., 2004; Sabin et al., 2003; Smith et al., 2009). The HSCL-25 is a 25-item measure of depressive and anxiety symptoms. Participants are asked to rate the frequency of symptoms on a Likert-like scale ranging from 0 (never) to 4 (almost always). Although the convention cutoff indicative of likely DSM-5 mental health disorder (e.g., clinical depression, generalized anxiety disorder) is 1.7, appropriate cutoffs vary cross culturally, oftentimes quite widely (Haroz et al., 2016; Ichikawa et al., 2006). HSLC-25 depression subscale cutoff scores of 2.25, for example, have been found to be appropriate in Afghan women, whereas 1.5 has been found to be more sensitive/specific for Indian women (Ventevogel et al., 2007). I am unaware of any studies that have systematically assessed appropriate cutoffs for Guatemalan and/or Maya women. Both the mean depression and anxiety subscales for grandmothers were above most reported cutoffs available in the literature.

Grandmaternal war trauma

Grandmaternal war trauma and grandmaternal/maternal anxiety and depression symptoms were assessed with the Harvard Trauma Questionnaire (HTQ) and the Hopkins Symptom Checklist - 25 (HSCL-25) respectively. Both measures have been adapted and used in previous studies of

trauma exposure and mental health in Guatemalan refugees (Mollica et al., 2004; Sabin et al., 2003; Smith et al., 2009). The HTQ is a 46-item questionnaire assessing multiple forms of directly experienced and witnessed traumatic events related to war. All but one of the grandmothers endorsed experiencing traumatic events during the war.

The most frequently endorsed items included the lived impacts of flight, such as not having a place to live (38%), lacking food or water due to the conflict (43%), and having to flee on short notice from their home for fear that they would be killed (43%). Sixty-six percent of grandmothers described either witnessing torture, sexual assault, gross bodily harm, or murder, describing the practice of communal burning of people in churches or public execution of villagers who refused to identify guerilla members or provide information on the guerilla. Only one participant described experiencing violence from the guerilla, who abducted, repeatedly raped, forced labor from and brainwashed her over a series of months prior to her eventual escape. Several accounts revealed witnessing of the documented and profoundly dehumanizing tactics of the military and paramilitary forces, including witnessing of villagers murdered via anal rape with poles and rifles, the display and desecration of mutilated bodies, and the disembowelment of pregnant women and murder of infants.

Grandmaternal and maternal life course trauma

Grandmaternal and maternal life-course trauma (not including trauma experienced during the war) was assessed with an adapted version of the Life Stressors Checklist Revised, which has successfully been used in Latin American populations, and which has been adapted and used previously with Guatemalan and Central American migrants in the United States (Humphreys et al., 2011; River et al., 2019; Wolfe et al., 1997). Following focus groups and pilot research, two items were added to this typically 32-item measure of common stressful life events. The first assessed community level censure and expulsion: “Have you ever experienced severe problems within your

community, such as being expelled, sanctioned, or systematically shunned?" The second assessed severe marital problems that did not culminate in divorce: "Have you ever experienced severe marital problems— such as disagreements with your in-laws or infidelity— that were bad enough that either your or your husband was kicked out of the house and had to find somewhere else to live?".

Many of the most frequently experienced stressors were shared by grandmothers and mothers. Eighty-six percent of mothers and 79% of grandmothers endorsed experiencing severe economic problems that constrained their ability to provide for basic needs. Nearly equal numbers (mothers= 75%, grandmothers = 79%) endorsed witnessing family violence before the age of 16 and experiencing physical abuse from a family member or intimate partner. Similar numbers of mothers (75%) and grandmothers (77%) endorsed experiencing emotional abuse from a family member or intimate partner.

Eighty-six percent of mothers (vs. 74% of grandmothers) reported experiencing severe marital problems. Interviews with mothers revealed ongoing challenges related to maintaining relationships in the face of infidelity, transnational immigration, conflicts with in-laws, and conflicts over ability to work outside the home. While these issues were also present in grandmaternal interviews, the proximity of these conflicts within different life-stages and the sexual or reproductive expectations mothers faced relative to grandmothers may have influenced different patterns of endorsement and memory. Similarly, some of the highest rates of endorsement for grandmothers were related to their experiences of aging and caregiving for elders, with greater numbers of them reporting experiencing severe illness (77%) and caring from someone with severe illness (77%).

Grandchild trauma exposure and socioemotional development

Traumatic events experienced by the grandchild were assessed via maternal report on the Traumatic Events Screening Inventory Parent-Report Revised (TESI-PRR) a measure of 24 stressful

events commonly experienced by children which has been successfully adapted for Spanish-speaking Latinx immigrant parent report in the U.S. (Griffin, 2021; Hagan et al., 2015).

Child socioemotional development was assessed via maternal report on the Strengths and Difficulties Questionnaire (SDQ), a 25 item Likert-like scale of emotional problems, conduct problems, hyperactivity-inattention, peer problems, and prosocial behavior, with the five problem-focused scales yielding a difficulties total score (R. Goodman, 2001). To my knowledge, Guatemalan and/or Maya population SDQ norms have not been published, but the SDQ is widely used in Latinx immigrant populations, including to assess the mental health of Guatemalan children detained in the U.S. Mexico border (MacLean et al., 2019). According to the cutoff scores recommended in the scoring manual, the mean SDQ total difficulty problems score of 14.67 was within the borderline (14/15) range for mental health problems in children. However, it is important to note the relationships between SDQ scores and 'caseness' of mental health problems vary by population, language, ethnicity, and culture (A. Goodman et al., 2012) . For example, in Mongolian school-aged children, researchers found 16/17 to be an appropriate cutoff score to screen for mental health problems; by contrast 11/12 was found to be predictive in Finnish children (Aoki et al., 2021; Borg et al., 2014).

Qualitative and ethnographic results

In this section, I explore the results of thematic and ethnographic analysis. As noted in the methods section, the analysis here is not meant to present a tabulation of common themes, but rather critically appraise the ways trauma experienced as intimate or personal was tied to structural violence, and link grandmaternal experiences of the war to daughter and grandchild experiences of precarity and violence following repatriation. Throughout, I highlight the ways my interlocutors' subjectivities negotiate desires that are both shaped by lifeworlds as well as constitutive of the lifeworlds they desire for themselves and their loved ones.

Forming partnerships: continuity and change

Grandmothers and daughters described the formation of partnerships as key moments of transformation that bore lasting generational impacts. While many community members do elect to get married in formal religious ceremonies, it is also common to juntarse or "get together" with a partner, a form of common-law marriage in which the couple take up residence together; when the pair are younger and lack property or housing of their own, this typically involves the young couple residing in the man's home with his parents, siblings and other family members, e.g. a patrilocal-multiple family household arrangement. Other couples with more means may rent a property within the community from a willing community member, or indeed leave the community altogether and form new households outside of Chaculá. As noted above, historical and contemporary Maya norms for post marital residence take many forms, but a general trend towards patrilocal residence or neo locality (as opposed to residing with one's maternal kin, or matrilocality) was described by most participants. Still, several participants had settled marital households on the same plot as their own mothers or co-resided with them after unpartnered pregnancies or leaving a partnership.

Many participants described informal marriage processes with the idiom of being "taken" (él me llevó) or leaving with someone (me fui con él). When this partnership occurred before women had left their natal homes— usually due to being still in middle or high school—they typically relocated to the home of their partners. Such relocation meant public recognition of loss of their virginity, and women oftentimes experienced rejection and stigmatization from their families should the partnership fail, and they wish to return home. Imelda, a 24-year-old mother of one, had formed one such partnership when she was taken by a schoolteacher to live with him as his wife at the age of 12. She wept as she recalled the experience, grieving the sexual exploitation she had experienced, but was most upset when describing her sister's insistence that she stay with him as she had brought shame on the family by seducing him. The transition to becoming a nuera (daughter-in-law) in the

home of one's in-laws for women with limited power was repeatedly described as emotionally humiliating and physically demanding. Nueras were expected to take on increased duties of agricultural work, cleaning the home, doing laundry, and cooking for in-laws as well as their husbands and own children.

Grandmothers experienced significant upheaval during the wartime period that shaped the ways in which they came to form partnerships, oftentimes obliged by pregnancy or forced sex. Those that described pre-war marriage systems revealed the embeddedness of kin support and considerations of sustainability and exchange. Gloria and her husband Pablo wed several years before armed conflict came to their Huehuetenango village. When I asked Pablo why he had wanted to court Gloria, he responded. "I knew her mother. Her mother was very clever. They had a small piece of land and lots of kids but were doing very well. Her mother knew how to do many things, I thought she had probably taught Gloria, and that she would be clever too." When I turned to Gloria and asked her the same question, she smiled and with humor in her eyes replied simply. "Well, he had a lot of corn."

Pablo and Gloria's marriage remained stable—though not without conflicts over Gloria's work outside the home as a midwife— throughout their daughter Atena's life. Their mutual support of Atena allowed her to become one of the first college educated women from their natal community. Bucking patrilocal marital traditions, they also permitted her to build a home and live with them on their plot, even after Atena's marriage to Josue, a man from Jacaltenango. Atena's co-residence with Gloria has in turn allowed her to build a valuable midwifery and reproductive health practice. Atena's nieces— the children of her brothers who traditionally have a right to live on the family plot and who live a few meters away— come to her home daily and take care of her young children and perform her domestic labor. This network of support allows Atena to work outside the home, traveling throughout Huehuetenango and Nentón to neighboring villages and communities to

provide prenatal and midwifery care. Her two sons, 8 and 4, have been raised relatively buffered from the root causes of child trauma: material insufficiency, inadequate support for education and healthcare, loss of loved ones, and violence in the home.

By contrast, other grandmothers described how the loss of maternal support and war-time disruptions removed many of the support networks that could buffer a new wife from a hostile marriage. Remembering the loss of her mother at age 8, Azucena describes being abandoned by her father and being forced to work throughout childhood and adolescence. "They say it was a heart attack, they say it was anemia, they say it was a cough. But I had no one to be with, even in the time of war. I grew up with people [not family]. Sadness. My life was pure sadness, because if I didn't work, I didn't eat. I didn't clean myself, I didn't brush my hair. I was abandoned…It was then as it is now." She fled to the refugee camps at 17 when war violence became overwhelming. Having already survived sexual assault by an extended family member at 12, she was again raped by paramilitaries during her flight. She met her husband Arturo in the camps, and while the first years of their marriage were relatively happy, she reported escalating physical and sexual violence throughout her pregnancies and the early years of her children's lives. After her return to the community, her husband was named family socio. He left her for another woman in the community, and while she still can reside in the home built for them by the Rigoberta Menchú Foundation, she has lost all access to land for cultivation. She and her daughter Reyna live together in the home, along with Reyna's three children. Together with Reyna's wage labor as an in-home domestic worker for a wealthier family in the village, they subsist off Azucena's bricolage of bread and tortilla making, taking in clothes for washing, and selling lunchtime foods at the local school.

Like her mother, Reyna's account of her life emphasized the material deprivation, emotional rejection, and mistreatment she experienced growing up. Reyna described feelings of intense emotional pain of being seen as a burden and source of discord within the family, leading to a

suicide attempt in adolescence. Azucena tied Reyna's struggles to the emotional distress she experienced as her pregnancy with Reyna was the product of marital rape. "Honestly, I never wanted her," Azucena admitted in her interview, blunt in affect but weeping. Reyna felt this rejection deeply, and to escape extensive maltreatment from both of her parents, became pregnant with her first child and joined the family of a young man in the village who lacked material means to support them. He accused her of infidelity and rejected paternity of the child, forcing Reyna to move back in with her mother with her young daughter Deisy. In the interim, Arturo had left Azucena and begun a new family with a woman a few blocks away in the village.

Reyna's life with Azucena continued to be emotionally challenging for them both. Without income from Arturo or food from their family plot, both Azucena and Reyna had to seek work. Reyna eventually left Deisy with Azucena and emigrated to Mexico as a domestic laborer, narrowly escaping being forced to work in a brothel. She met her second partner Chuy here, and they began a life together cleaning hotels in Cancún. Their marriage was filled with conflict; Reyna described a cycle of intensely emotional fights that frequently culminated in physical violence. These conflicts became worse after the birth of their two children, Preciosa and Ezekiel. She describes Preciosa's exposure to these episodes as a two-year-old.

> I was cooking and he came in, he was on his phone. I wanted to know who he was on his phone with. I was always jealous, I'm like that. He was talking with other women. So, I asked him, I confronted him. Preciosa was with me, she was little, maybe two or three, it was before Ezekiel was born. He got so angry at me for asking, he began throwing pots and pans everywhere. Everything, everything. He made such a mess of the food. I was so angry. He hit me and choked me, screaming and crying. I know that he loved me, but he was so altered, he would get so angry so quickly. I was like that too. After a while he got so tired of fighting, he lay down on the bed. I took a knife from the kitchen, and I sat on his chest. I put the knife at his neck, and I told him to kill me. Kill me, kill me. And he said no, I will never kill you, you can kill me. Just kill me, Reyna. You have to kill me. And Preciosa came

> and she said please don't kill my daddy. Don't kill my daddy. And then I put the knife on the ground, and I lay down on the ground and I wept, and so did he.

Working in humid Cancun with limited access to insect repellent, Reyna was infected with Zika during her pregnancy with Ezekiel and he was born with severe microcephaly. Ezekiel's disability proved to be the breaking point in the relationship. Unable to cope with the emotional distress of their son's disability, Chuy separated from Reyna, and she and the children were forced to return to Guatemala and move back to Azucena's home again. A year ago, after being diagnosed with advanced cervical cancer, Reyna underwent a hysterectomy in the local public hospital. "Who will want me now," she said, signaling the importance of women's reproduction as a key feature of partnership formation and maintenance.

The series of events that link Azucena and Reyna's subjective experiences of social rejection, maltreatment, and constrained agency have gone on to shape the emotional worlds of Deisy, Preciosa and Ezekiel. Their shared experience of domestic and sexual violence is not intergenerationally transmitted through hormones or cytokines. Neither is lack of knowledge about 'proper parenting'. Instead, their distress and risk of re-exposure to trauma is deeply linked to limited options for seeking alternative social, romantic, and economic arrangements in their lives. Even within these limitations, the desire of interlocutors to negotiate, bricolage, and hope for change is vivid.

Simona, like Reyna, describes her teenage pregnancy as a liberatory and strategic choice made among limited options. Growing up in a home filled with family violence, she describes her outrage at the unpredictable and arbitrary nature of her father's physical abuse and unwillingness to humanely explain what he wanted and how he wished her to respond.

> I didn't understand that, that part, why he hit us so much. If we didn't want to do him a favor or something. Sometimes he would say "Bring me my sandals!" or "Bring me my shoes!". Sometimes we'd say, "In a little bit." Then when we'd look up, he would be there,

ready to hit us. That is how it was. My mom wouldn't get involved, because it was the same for her, he hit her so much. He beat her so much. We were little, we couldn't defend her. [He hit her] so much.

I lived that childhood. That's why when I was 16, I got together with my husband, I just couldn't live that way anymore. Not anymore. I had boyfriends. I had secret boyfriends, because if my father found out, he'd hit me. Everything was hitting. He never talked, he never sat us down and said, "Honey, this is wrong," or, "Sweetheart, this is how you should do things,". No. Him and his belt. I had boyfriends secretly. That's why I got with my husband at 16, I felt I had freed myself from all that.

I didn't want him to hit me. The day I left, he hit me with a machete. I got so scared, because I thought he had cut me badly and everything, but no.

Interviewer: Did you feel like you were leaving your family for someone who would love and treat you the way you wanted?

No. Maybe he really did love me. How can I explain it to you? Maybe it was just so I could be free. All I wanted was to get out of there. I don't know. Maybe in my head I was thinking, "Who can I stay with, who will take me? I want to escape. I don't want to be here anymore. Not anymore." Sometimes, because I didn't want to be at home, I would stay at my friends' houses all day and only come home late at night.

I think that's why I got with my husband so young, because I was already pregnant when I moved in with him. It's like I got pregnant— how can I explain it to you, just between the two of us— it's like I only got pregnant so that I could be free.

Simona, Reyna, and Azucena lived cycles of violence that many biosocial paradigms would locate in their capacity to form appropriate relationships. Attachment-based theories on the intergenerational transmission of domestic violence can locate damage within their way of loving and relating. Attachment-based hypotheses of the intergenerational transmission of domestic violence include the idea that girls who are maltreated as children go on to have pathological

romantic attachment styles that naturalize violence and resulting in the seeking out of violent relationships and of increased risk of perpetuating violence against their children (Madigan et al., 2012; Zeanah & Zeanah, 1989). These vignettes serve as a counterpoint to this suggestion. Reyna and Azucena's story is very different than Gloria and Atena's, but this is not because either daughter discarded better partner choices in favor of violent ones. Each made the best decision available to her with the resources (their bodies, reproductive capacities, labor capacities, land) at the time, a manifestation of resilience that might also show up as vulnerability in a dataset. Simona's vivid clarity about her active decision to seek out pregnancy as a means of seeking freedom is richly illustrative of an insurgent sense of self. Simona seemed to think that I would judge her for getting pregnant on purpose at 15—she was aware that I had come to the community as a worker in reproductive rights/maternal health—but she was deliberate in her account, looking me full in the eyes and speaking with dignity and clarity.

Bargaining for better relationships

Participant narratives also evoked the different ways in which women resisted the intersecting forces of patriarchy, economic exploitation, and limited subsistence strategies. These strategies were prismatic: some imbued suffering with meaning through religious conversions and transformations, others invested in the education of daughters and themselves to create economic opportunity and autonomy, others formed strategic partnerships with well-resourced men and invested in fulfilling gendered expectations.

One of the most cohesive family narratives of resistance through the re-establishment of maternal ties and financial independence came from daughter Lorena and her mother Valentina. I had known Valentina for several years before beginning fieldwork. An active participant in various local Maya feminist organizations, Valentina was a fierce advocate for women's rights and vocal about discrimination within the community against Mam people like herself. Valentina fled her

homelands in San Marcos in her early teens, after her village was invaded and terrorized by government paramilitary forces. She lived some of the most brutal experiences of the war, including witnessing torture and murder, surviving rape, and a lengthy and dangerous forced flight through the wilderness into Mexico. Like many of the grandmothers I spoke with, Valentina viewed her indigeneity as both the source of genocidal violence against her as well as a font of resilience, meaning, and resistance to be transmitted to her children. She attributed these attitudes to trainings and support received from feminist NGOs in refuge, for whom she ultimately became employed as a community worker. She married in Campamento Carolina, Chiapas, not long after her arrival and there she gave birth to three boys and two daughters, the eldest being Lorena. ??She, her husband, and children all came to Chaculá when Lorena was six, and Valentina was named as family socia. However, more than any of the traumas she experienced in the war, Valentina indicated the seminal trauma of her life was the unsolved murder of her youngest daughter Flor, who was killed at 16 in the city of Huehuetengo where she had gone to attend high school.

Like Valentina, Lorena also describes her indigeneity as a source of pride, but finds herself slightly more able to express herself in Spanish and doesn't wear traje as daily wear, preferring the leggings and stretchy skirts that most women of her generation wear for comfort and ease. Like many participants, Lorena suffered difficulties in the beginning of her marriage, describing overwork and maltreatment as a nuera in her mother-in-law's house. Her husband's alcohol abuse led to increasingly desperate finances and during her second pregnancy, Lorena left her husband and moved in with her mother.

Unlike Azucena and Reyna, Valentina and Lorena had access to significant amounts of land due to Valentina's sociedad. Valentina settled the pregnant Lorena in a small home on the family plot and supported her business ventures. Lorena began working, bringing in cash income from a small business cooking school lunch foods and opening a small dry goods store. These activities

were enabled by their land subsistence; Lorena and Valentina can eat their own corn and beans for most of the year and raise chickens and vegetables. Along with increased subsistence and NGO contracts, Valentina runs a small chicken-frying stand in the town square, sells weavings, and makes money providing limpias and other traditional Maya treatments for the relief of emotional and physical ailments. Together, their bricolage of subsistence and wage labor is sufficient to support Valentina and her husband and Lorena and her children.

Lorena directly credits her economic autonomy for her increased ability to resist violence and demand different treatment from her husband, something biological anthropologists and economists refer to as "bargaining power" (Lowes, 2016; Mulder & Rauch, 2009). Like other women I interviewed, she described resorting to direct confrontation in the face of threatened violence. After her husband came home to try to repair the relationship, he began criticizing her work, accusing her of talking to other men and being a bad mother and wife. Like so many of my interlocutors, Lorena is a gifted storyteller and orator. She provided an almost screenplay-like recreation of her confrontation of her husband, demanding that her domestic labor be compensated either with money or substituted with his own.

> One time, he tried to hit me. "You're always worried about what you're selling. You don't take care of us anymore!" He gets up with that look like he wants to hit me, he pushed me, and I almost fell. My son started crying and my brother came in. "Don't even think about hitting my sister," he says, I get up and I tell him, "If you don't want me to work, I will sit down right here." I'll tell it to him to this day. "If you want me to take care of you, you want me to iron your clothes just the way you like, you want me to make the food right, I will sit right down in this house. I won't work, I will sit right down here but I will want money," I tell him.
>
> It's not like I'm rude to my husband, but I work…I am so grateful to my mom, because of her NGO work, she's had opportunities. Sometimes my mom will tell me, "Between you and me, daughter, we are going to put food on the table." I even have money left over. I buy

> my own clothes and get things for the kids. I tell my husband, "I will sit right down, but I want money right here in front of me, because you are lucky, I give you as much as I do," that's what I tell him.
>
> Sometimes I want a kilo of sugar, sometimes he isn't working, and I'll go buy it and maybe get myself a little soap. I want a chicken breast, I buy it. I tell my husband, "I will sit myself right down in this house, but I want money here." He tells me, "Where do you want me to get money from? You want me to start robbing people?" I reply, "That's why I have to help you." That's how it happened. Later he was embarrassed, and he says to me, "Forgive me honey, don't be mad at me." I was really upset. It was like three discussions like that in the end.
>
> Afterwards he changed and when I would get home from working— sometimes I would leave all the dishes and stuff on the table—and when I get home, there he would be, washing dishes in the kitchen. [laughter]

Lorena's entire body was animated during this monologue. Every time she said "I want money" she tapped the table in front of her forcefully. Her pleasure in recounting her victory was reflected in her smiles, mirth, and her masterful use of her voice and body to convey the unfolding drama. Self-fashioning herself as the protagonist of her own life, she took evident pride in her bravery and cleverness—her ability to become the one with "a lot of corn", as it were, while her husband washed the dishes she left behind. In this moment in time, she emerges as glitteringly resilient, but I also think of the costs she has paid and that her children have paid to arrive here. Like many other mothers who described domestic violence, her children were almost always witnesses to her conflicts with her husband and in-laws. While her brother arrives in the story to protect her, in the rest of her interview she described his bitter resentment of her relocation to the family plot. Women's matrilocal post-marital residence threatens male inheritance of land, and many of my participants reported that their brothers sometimes violently rejected their attempts to settle near their mothers.

Ways of caring

Mutual understanding through emotional intimacy, care, and rational resolution of conflict was a way many families enacted their desires for peace despite constrained material and emotional resources. One of the ways this was described most frequently was with the value of talking, hablando de las cosas. Mothers and grandmothers described the importance of talking and coming to understanding rather than hitting children, partners, or nueras out of anger. While many described feeling that their children could test the edge of their tempers, few endorsed the idea that physical punishment was generally acceptable or that children didn't benefit from gentle, compassionate, discursive approaches. Similar ideas around how to foster a good family life and maintain connections with romantic partners and other families were also described by my interlocutors. Emotional intimacy and connection also extended to non-human aspects of the world, such as emotional connections with the Holy Spirit or with Maya gods, as well as the pleasures of connection and harmony with the land itself. Cultural anthropologists call this "intersubjectivity' and are often most concerned about its possibility between themselves and the people they study (Hollan & Throop, 2008). But intersubjectivity— a sense of shared visceral feelings, moral orientations, and lifeworlds— was described as an important part of the everyday pleasures of sociality in Chaculá as well.

The challenges of everyday life in Chaculá have consequences for harnessing the material and emotional resources for talking, relating and loving. Clarita, a Chuj mother of two describes the way her husband Mario's periodic absences to work in Mexico have impacted her and her children. Her seven-year-old son Jairo is especially affected, throwing lengthy tantrums and lashing out at Clarita in the weeks after a visit from Mario has come to an end. These tantrums unnerve her deeply, especially because Jairo behaves well with his father and expresses that only his father loves him during his absences and refuses to obey her. She describes a time Jairo went through his room,

throwing everything out of the window, except the things that his father had bought him, until only a few sandals, books, and toys were left. Clarita described how humiliating his rejection of her authority and identification with his missing father— when she spent all her time caring for him and his sister— overwhelmed her emotionally, resulting in her sometimes spanking him, a decision she felt deeply ambivalent about. When I asked her how she wanted to treat her children, she responded:

> Clarita: I want to treat them the same way my mother would treat me, loving them, educating them. My grandfather would say, "There's bread in one hand and a belt in the other. Which one are you going to choose." Back in the day it was like that. It's different now. Now you have to love them, you have to talk to them the right way, it's no good to hit kids.
>
> Interviewer: Why do you think things have changed?
>
> Clarita: Maybe because we know that kids actually understand more than we thought, or maybe it's because once they're grown up, they'll understand. You give them so much love, and they hold on to all the love you give them as they grow up. But when they're big they'll do whatever they want anyway. Maybe our grandparents were right, I don't know.
>
> Sometimes you have your weaknesses, because kids misbehave, and you don't know how to guide them and so sometimes you end up hitting them and it doesn't have to be that way. I'm very nervous, it's hard for me to see that sometimes. Sometimes I don't even understand why I hit them. My husband will say to me, "My love, you have to control your temper, because with kids it can't just be hitting them and hitting them, you have to talk to them." He's really calm. When Jairo was little, he was always here and he'd never hit him, he would just lovingly tell him, "Don't do this because it's wrong." He would explain things to him.

Clarita's reflections here are especially poignant to me because I had also interviewed her several years earlier during pilot fieldwork. It was her first separation from Mario, and she had been

deeply sad and emotional in her interview. When I had asked her what the hardest part of her life was, she responded that she felt ugly, and was certain Mario would abandon her for someone else while he was away. She breastfed her one-year-old daughter throughout the interview, even as she wept profusely, and I thought about Maria Tapias' work on embodiment and Bolivian cultural beliefs transmission of maternal emotion through breastmilk, mirrored in the biological anthropology of maternal stress and breast milk composition (Miller, 2017; Tapias, 2006). Clarita expressed no beliefs that her sorrow would reach little Perla through her milk. She breastfed her continuously in the same way most of the women in the community continue to provide on-demand breastfeeding and carry their young children as normative forms of childcare.

When I asked Carlita how her relationship with Mario was going, remembering that she had struggled so much during the early years of separation, her body visibly relaxed. She responded:

> How are we now? We're so good, we get along so well, we talk a lot. Even if he isn't here, I feel something deep inside me that what he tells me is true. I feel like he loves me the way I love him. We get along really well. I feel like I couldn't have found a better man. I feel like we are happy. He changed a lot, for the better. Maybe we don't have a lot of stuff, there are no fancy things in our house. But I'm happy with what we've managed to get. Having love between us is the most important thing. We talk every day, we talk about all of our stuff, everything that happens to us. Things that don't matter, we're always talking about them, we laugh and joke all the time. It's hard because sometimes you just get to thinking that you just want them to be here, you want to hug him, kiss him, and you can't. That part is hard.

Clarita's desires for emotional intimacy and pleasure, physical affection, and recognition that familial love is more important than material wealth was reflected in the other interviews I conducted in Chaculá and in my observations of everyday life there. Many participants described the importance of a fluidly intermingled sense of physical and emotional life for creating peaceful, joyful families. Almost all the participants I interviewed experienced and practiced extended co-sleeping, breastfeeding, and child-carrying. Several who resided with their mothers and who didn't

have current partners co-slept, with grandmother, mother, and grandchildren in the same bed. These features of Maya childcare have been documented in many different ethnographies and are not novel to report, although their near-universal persistence despite other cultural changes is notable. Still, I find it interesting that, particularly in biological anthropology and cross-cultural psychology, the idea that Maya emotional attachments are mediated through physical, rather than verbal, proximity (Cristia et al., 2019). While I think there are important insights from this research, it can also occlude the goals and desires of Maya caregivers. I would not generalize from my findings in Chaculá that all Maya mothers feel the same way about the importance of emotional intimacy, talking, and compassionate conflict resolution with children and family, but I would suggest that my findings add nuance to the diversity of how we talk about and essentialize what Guatemalan mothers think of as good mothering and good partnering.

Hablando se entiende: to talk is to understand

The importance of talking and holding respectful space for multiple viewpoints (sometimes for hours) is also reflected in communal processes of governance and conflict resolution in Chaculá. General meetings are held monthly to discuss community initiatives and problems, and all community members (socio or not) are able to attend in the large communal sala in the center of the village. Coming from an increasingly unrepresentative republic in the United States, experiencing these decision-making processes was the greatest form of culture shock I experienced (and continue to experience) every time I visit Chaculá.[16] One night, a motorcycle with a man on a bullhorn came roaring past the house. "Emergency meeting of the water committee called at 3 o'clock tomorrow" a man exclaimed as he rolled past. My research assistant and I were surprised, what kind of emergency does a water committee have after all?

[16] For example, during my fieldwork year, the community had agreed to a strict 9 pm curfew. No one was allowed in or out of the main entrance, which was chained off, and anyone seen walking in the streets after 9 pm was threatened with a fine.

When we attended the meeting the next day, the stakes of the conflict became clear. Normally each neighborhood in Chaculá had to nominate an individual who would serve on the security committee (a kind of informal local police force) each year. The neighborhoods had a system where each household on the street took turns providing the volunteer so that the work was shared equitably, as the position was unpaid and required volunteers to do nightly patrols and manage crowds and rowdy people at festivals. A man in barrio dos named Juan had refused, and the meeting had been called by the water committee because it was determined that if Juan refused to do his part for the community, then the community ought not provide one of its essential and free services to him, this being the provision of water.

That year's assistant mayor Don Abelardo described Juan's case. Juan had felt that he had contributed significantly to the construction of Chaculá, but because his elderly father had decided to inherit his brother as socio of the family, he bristled at ongoing labor duties to the community without receiving political representation in la cooperativa. His nomination for the security force had been the last straw. Don Abelardo explained that Juan had been invited to the meeting to present his case but had thus far refused to come. The community was to decide what to do about it.

This decision-making process— like many other communal justice and political decisions— involved people taking up a microphone and talking at length. Some advocated that Juan be expelled entirely from the community for his lack collaboración. Others responded that such severity was inhumane and suggested that cutting off his water would be sufficient punishment. Still others noted the life-giving qualities of water, suggesting it was a denial of a basic human right. "A man can live for a month without food, but without water he will die in days," said an elder woman, Dona Telma, to murmurs of agreement. One man suggested that the matter was one to be dealt with by Juan's family and that even discussing this in public was disrespectful and wasteful of everyone's time. In response, Juan's father spoke at length about his lack of understanding about why Juan still resented

his choice to designate his brother as socio, describing what he felt was a just land-sharing agreement between the brothers.

After several hours of open discussion, Don Abelardo held a vote to either cut off Juan's water or expel him. Only a few people raised their hands in favor of expulsion. Don Abelardo then asked everyone to come with him to Juan's house to cut the water tube that led to Juan's home as a communal act. "If I go myself, he will say I decided to do this on my own. I need you all to come with me to show it is the will of the people." The hundred or so of us walked from the sala towards Juan's house. As we arrived Juan's brother, the new socio, emerged in front of the crowd and declared that he would pay for someone to cover Juan's duties. Satisfied, the crowd dispersed, and with it the mandate for Juan's punishment.

While I experienced this episode as stressful, fearing the potential for violence and was worried for Juan's safety, many people told me afterwards that they were sure no harm would come to Juan, and that his brother just needed the public shaming to urge him to pay. The practice of extended public discussion, participation in justice, and strong norms around collective rights and duties are not unique to Chaculá. Many other Maya villages of the region share these practices. Chaculá's relative gender egalitarianism and the role of women in public life and decision-making, however, may be somewhat different than surrounding communities. Although not the subject of my research, future work might explore how the political-economic aspirations for collectivism, unity, and gender equity in Chaculá influence women's perceptions of their ability to talk and show forms of care and negotiation in their intimate lives as well.

Discussion

The psychometric data and qualitative Chaculense narratives reveal how patterns of violence lived in intimate life are rooted in the ecological inheritance of structural violence: deep histories ongoing of colonial exploitation, land loss, and exploitation that have resulted in the economic

scarcity and political disempowerment of poor and Maya communities in Guatemala. The study finds that—at least in the subsample of women who volunteered for research—high levels of war trauma in grandmothers are joined by high levels of traumatic life event exposure in their daughters, and to some extent grandchildren. Grandmothers endorsed an average of 8 war-related traumatic events, rates like earlier work by Sabin and colleagues (2006) conducted in refugee camps in Chiapas. This suggests that grandmother's memories and/or experiences least like those populations that reported on their experiences nearly twenty years ago, and the nature of their experiences is like those of other testimonios and evidentiary accounts.

Daughters reported a similar average number of stressful life events (15.1) than grandmothers (16.1), despite being younger and having less time to 'accrue' events. This is notable from the perspective of theories of subjectivity—similar events may have been perceived to be or reported to me as stressors by daughters that were not perceived as (or considered appropriate or safe to report) by their mothers. Shifting senses of what loving vs. abusive treatment and behavior are, proximity to painful experiences in youth, and expanded senses of women's role in society may have shaped this higher rate of endorsement. Simultaneously, the world daughters experience is different than that of their mothers. Increased rates of migration and family separation, narco and gang violence, and intense pressure surrounding land tenure and postmarital residence create challenges, losses, and conflicts in daughters' lives. While the version of the Life Stressors Checklist Revised was adapted in this stud to include two additional items, I based it off the version I had previously used as a researcher in a trauma-treatment clinic in the United States with Latinx immigrant families. I was struck that the community sample in Chaculá endorsed stressful life events at a similar rate as this clinical sample in the U.S., which endorse an average of about 12 events (Hagan et al 2017). Average rates of child trauma on the same questionnaire in my sample (4.0), compared with a U.S. clinical sample (5.98), seem to somewhat lower, although the age range of the

children in Chaculá is greater and this renders direct comparison not particularly useful. That both grandmother and mothers should express high rates of anxiety and depression symptoms (averages above multiple cross-cultural cut points) on the Hopkins Symptom Checklist is not surprising, given these experiences.

The picture that emerges from the quantitative data emphasizes deficit and damage, suggesting not only that intergenerational trauma is being transmitted, but potentially amplified across generations. But this reductive and stigmatizing view of intergenerational trauma is subverted by the narratives and subjectivities articulated by the women themselves. Unlike symptom inventories, the ethnographic data and in depth interviews help show how women in this study contend with and resist these forces by harnessing a wide array of resources—the reproductive capacities of their bodies, self-fashioning as protagonists through storytelling, collaborative labor bricolage, using maternal post-marital residence as a means of buffering relationship conflicts and increasing bargaining power, or indeed drawing in multiple resources to invest in their education as professionals.

This interplay among constrained agency, enactment of desire, cultural resources to promote wellbeing, and "talent for life" exhibited by people resisting structural violence is well-trodden ground within the medical anthropology of structural violence, trauma and resilience (Eggerman & Panter-Brick, 2010; Scheper-Hughes, 2008). Attention to intergenerational subjectivities as revealed in these interviews helps provide new avenues of insight beyond domination and resistance narratives. Rather than a simple 'tactic of the oppressed', Simona's self-fashioning as an insurgent pregnant adolescent crystallizes her awareness of the patriarchal structures that constrain her and celebrates the creative, resistant, and subversive womanhood she embodies. Subjectivity offers us a lens on such forms of discursive resilience—the harnessing of a narrative strategy to sustain a sense of a purposeful self. Such forms of resilience can go on to (re)shape loved one's lives, one's children,

parents, kin, and friends. This perspective thus both opens a view of what resilience can look like as well as help expand our self of the directions intergenerational transmission can take. Future work might consider how grandmaternal lifeworlds have been shaped by their daughters' experiences, or how shifting childcare norms alter one's perspective on what was right or wrong about one's own upbringing

Early in my graduate studies, I had coffee with an anthropologist who worked in Guatemala and spoke about my project. "No one is going to talk to you about the war," he said, in a tired voice. The feeling that Maya subjectivities are marked by silence or unknowability is recurrent in anthropology of Guatemala. In her comparative analysis of Chaculá[17] and "La Victoria", a different repatriation community, Rousseau and colleagues (2001) describe Chaculá as place where "history is built around silence, a silence that speaks of suffering" and suggest that the community's acute feeling of weakness, vulnerability and isolation has steered the young people of Esperanza toward a strategy of turning inward, avoidance and a return to points of reference predating the trauma,"(p. 160). I have re-read this piece many times, wondering if the silent 'young people' who were the interlocutors to its authors are now the grandmothers in my own study. Nelson's (2009) exploration of a culture of mistrust, apprehension, and fear of duplicity in postwar Guatemala similarly underlines a sense of limited intersubjectivity and mutual understanding. By contrast, the interviews in this study were largely characterized by a sense of intimacy, power over self-narrative, and probing curiosity of my expectations and thoughts during the discussion. A lens of subjectivity highlights the recurrent value that women placed on intersubjectivity and the sharing of inner life with those they love. While this may be a generational difference, these desires were also present in the narratives of grandmothers, signaling processes of healing and transformation rather than damage and deficit.

[17] It is referred to as "La Esperanza"in this piece.

Finally, attention to moral injury shifts emphasis away from ideas of the 'innately traumatic' and development of static 'universal' traumatic stressor inventories, but rather ask us to attend to the lived experience of violated norms and beliefs. This experiential focus, I believe, returns agency and humanity to the individual, rather than particularizing trauma into unconscious 'transmission' that is viral or infectious in aspect. Biological mechanisms of traumatic transmission were rarely described in this study; few of my interlocutors believed that they had inherited trauma through the body, through hormones, epigenetics, or bad parenting. Some local cultural model of intergenerational transmission may exist that was not shared with me—in one of my interviews, an interlocutor mentioned that "some say it can happen, but I don't think it does"—but not clear shared beliefs around these phenomena emerged. When I have discussed my findings to Chaculenses, their interest centers on strategies for managing land inheritance and avecindados as future generations grow and the land becomes over-populated. The idea that their intimate struggles, experiences of loss, separation, and conflict are related to diminishing means of local subsistence and rising community inequality is not revelatory to them, although it may be meaningful to scholars of intergenerational trauma.

Beyond its theoretical aspects, ethnographic approaches to intergenerational trauma that emphasize participant subjectivity may have applied value to mental health professionals who treat communities affected by legacies of trauma. In the one sense it does so by reorienting the causes, presentations, and potential treatments of trauma towards the social. In the other, it provides an important case example of the kinds of traumatic histories a large portion immigrant populations may carry with them, but that are not legible to mental health practitioners. In the United States, Maya peoples are subsumed under the umbrella term "Latinx". This process occludes their unique political and cultural histories in ways that impact the cultural sensitivity of mental health practitioners. Understanding the deep histories of loss, racism, dehumanization, and dispossession

that Guatemalan Maya bring to clinics in San Francisco or Fort Lauderdale is essential to understanding the traumatic disruptions and social 'tethers' that might confer healing to their patients. By the same token, I also wish to signal the multiplicity of cultural scripts the Chaculenses drew upon to construct inner lifeworlds. In particular, the diversity of spiritual beliefs[18] lived in Chaculá means that clinicians should not imagine all their Guatemalan clients use temescals, want to wear traje, honor Maya gods, are Catholic, or do 'indigenous rituals' for healing. Some do, and many do not. Rather than seek to essentialize or 'encylopedize' the cultural beliefs of one group or another, an anthropology of intergenerational trauma helps shed light on the shared processes by which harm is socially perpetrated and inscribed but individually felt.

It is important that I acknowledge limitations to this study. My decision to interview only Spanish speakers restricted the kinds of people I was able to interview. Although most members of the community speak Spanish, it is possible that some—especially elder—people were unable to participate due to this requirement. Given that Spanish speaking indexes educational privilege and opportunity, this bias likely excluded those members living in greatest need in the community. Another important limitation is the expectations that participants may have had given my status as American and association with a human rights NGO. It is possible that they felt narratives or attitudes might be more congenial to me—such as feminist, anti-violent, or less-capitalist ideologies. Finally, another key limitation is my exclusion of men from the study. To some extent, this choice reifies women as containers of trauma, nationhood, and oppression. In future work I hope to meaningfully integrate male family members and draw in their perspectives.

[18] Though religious themes were not extensively explored here, I find many clinicians are surprised to hear that many Maya Guatemalans practice Evangelical Christianity. Those that are 'culturally aware' oftentimes conflate Maya identity with traditional Maya cosmology and healing practices, which may or may not be the case.

Within anthropology, projects that illustrate previous insights (rather than provoke novel theoretical engagements) are often given short shrift as derivative or 'applied',[19] and my work here names and resists this tendency. Ultimately, I believe this work should center the experiences, needs, and hopes of the Chaculenses themselves. I look forward to the opportunity to present it to them, and for the reflections of past, present, self and community the opportunity will bring us.

[19] I have always struggled with the pressure for my engagement with Chaculenses to reveal something radically new about the fundamental nature of intergenerational experiences of violence and the forms resilience takes on in the subjective experiences of the oppressed. It has always seemed incredibly extractive, as if I needed their stories to count for more than what they did. As we joked often in my graduate seminars, a cynical goal of our grant proposals was to demonstrate how we would score esoteric theoretical points on the backs of the poor.

Chapter 2 Appendix

Full list of themes and subthemes

Forming partnerships: continuity and change

This theme was included in the final chapter write up. It refers to the processes by which women formed romantic relationships and started families, and the conflicts and challenges they experienced during these times. It also encompasses the subsistence and land tenure challenges that women experienced during relationships. This umbrella theme was developed from the more content-based subthemes below.

Timing of marriage

Family planning

Perinatal complications

Land tenure and postmarital residence:

In-law conflicts

Sibling conflicts

Division of labor/marriage as subsistence

Bargaining for better relationships

This theme was included in the final chapter write up. It refers to ways in which women expressed their desires for better relationships and reflected on cycles of violence that they had experienced. It also encompasses the strategies women used to bargain or transform their relationships, especially the relationship of subsistence, lend tenure, and financial independence as a means of bargaining for better treatment.

Cycles of intimate partner violence

Male alcohol and substance use

Infidelity

Financial independence is emotional wellbeing

Ways of caring

This theme is related to "bargaining for better relationships' in that it expresses desired ways of relating, but rather than attending to cycles of violence, it encompasses the expressed desires of women for what constitutes a good relationship either with the family, partner, or children.

Hablando se entiende

Cariñoso relationships

Understanding vs. not understanding people's intentions

Changing identities

This theme was not presented in detail in the chapter but is fertile ground for future work. It relates to expressions of identity, belonging, and cultural change. These codes were attached to statements about folk or spirit healing, traditional Maya values or beliefs, other religious (primarily Evangelical) beliefs including conversion stories, ideas around identity as Ladina for those women who did not have a distinct Maya ethnic group but who also identified as being Indigenous (usually grounded in self-phenotyping and ideas around race, skin color, height etc.). I also included codes around the diffusion of global culture into Chaculá—music, movies, fashion, social media, and other ways in which U.S. based Latinx, white, Black, and other cultures (e.g., lots of Korean soap operas!) influenced attitudes around romance, beauty, and relationships. Additionally, specific attitudes around the role of NGOs and feminist organizations in changing women's attitudes around reproduction, women's empowerment, environmental justice, and other themes were included here. I also included a related subtheme, comments about the political future and changes in Chaculá, as these seemed to be relevant to a sense of local identity. There is absolutely another chapter. (or three) nested in this theme!

Traditional Maya Identity

Religious experiences and transformations

Ladina Identity

Influence of global culture

Influence of feminist/NGO/activist organizations

Chaculá political changes/cooperative governance

Hopes for the future

This theme was not presented in the chapter but may be integrated in a future revision. It deals with the expressed hopes that women had for their children at each generation. They tended to cluster around a desire for children to have adequate nutrition and development from a growth perspective and highlighted many women's' hope that their child be able to get secondary (and in some cases, even university) education. Moral development—the desire that their children should grow up to be good people—was also frequently expressed as a desire. Though "explaining things to children" was included to some extent in "Ways of Caring", I also included it here as it signaled the process by which parents felt children ought to be brought up, e.g., through talking. Discussions of corporal punishment (guilt around giving it, beliefs that it is necessary, using threats of violence) are also included in this theme.

Childcare norms

Education

Nutrition

Moral development

Explaining things to children

Corporal punishment

Overcoming challenges

This umbrella theme is not included in the chapter, although aspects of it are woven through. It refers to the strategies that women used to overcome challenges, whether these be through external or internal resources. I would like to further develop this theme for a resilience and strategies focused chapter/article in the future. Codes here include internal processes, such as prayer, narrative, and 'manifestation', something I think about like intention setting or goal setting, as well as external processes of labor bricolage, "hustling", calling on extended networks for support, and investing in one's own embodied capital. I include 'early life in Chaculá' here because the narrative of the poverty and limited resources the community had and the role of collective action in bringing it together is a kind of meta-narrative of challenges overcome.

Labor bricolage

Spiritual manifestations

Maternal education

Early life in Chaculá/building the community

Subsistence bricolage

Painful emotions

This umbrella theme was not explicitly included in the chapter, although aspects of it are woven through certain narratives that are presented. It refers to explicit mention of emotional or physical pain, and the contexts that surround those experiences of pain, sadness, loneliness, or despair. A distinct subtheme around menstruation, birth, and pregnancy emerged here and so I pulled it out from "illness".

Family secrets

Loss and death

Never having been loved

Fear of menstruation/birth/pregnancy

War time experiences

Sexual trauma

Illness and disability

Meaning and pleasure

This theme was not presented in the chapter. It refers to experiences of meaning, pleasure, and fulfilment that participants described. Many of these centered around experiences in nature, including during agricultural labor or other 'non-recreational times". Many women also expressed the pleasure in being alone, and enjoying rare periods of rest and respite in their busy, very social lives.

Experiences in nature

Childhood memories of happiness with extended kin

Being alone

Rest

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Interlude 3

Exclusion Criteria I

Today I interviewed a grandmother who could barely speak. I didn't write her name down correctly on the consent form because I couldn't understand it. She spoke Spanish but was nearly toothless. Not only toothless, but her lips were also puffy and distended and it seemed like speaking at all was painful to her. She was almost unintelligible. I half-heartedly recorded the interview but wish I hadn't.

It was the fastest interview I have completed. We were done in about 45 minutes. It was entirely flat.

She didn't know her age. Eighty, I think, she said, although she was 1 year old when the war hit its peak. It can't be I said, let's try to figure it out together. I am going to ask her daughter to see if I can learn better. She is the only woman with very gray hair.

She showed me an angry red scar on her belly, a belly so hard and distended for a moment I worried she might have had a tumor removed that had recurred. Later my RA told me it was her gall bladder.

The interview was affectively flat, blunted. She did not acknowledge many traumas, and it was the first truly flat TESI for the child, I think. Zero traumas. Her main trauma, the most painful experience that she had ever felt, was the loss of her youngest son, 1 of 8. He left and never came

back. He didn't call. A week or so ago, her daughter had explained this a bit in her own interview. "Maybe he felt like we didn't treat his wife well enough, I don't know. We see him on Facebook, but he won't talk to us."

Her symptom inventory was more vivid, but because of the difficulty in acknowledging traumas, the PCL5 felt like it was in vain. I did some rephrasing as I was worried about her comprehension.

When I got to the item about negative convictions about self, I rephrased. "¿Usted siente que es mala? ¿Que es una persona mala?"

Do you think you're bad? Do you think you're a bad person?

The amber whites of her eyes became radiant with tears. She nodded mutely and wept. I was unable to stop from weeping as well. I told her I did not think she was a bad person. I told her that her love was still with her child, it still reached him even if she couldn't speak to him. I felt I was a bad person for coming there and asking a nearly mute woman to tell me her story, knowing I could not understand it, knowing that her data would be excluded for poor validity.

Later in the night, I texted a friend who is a clinical psychologist in the U.S. about it, wondering if I had said the right thing. I wrote "I told her that her love is still with him." But I forgot in my emotion that my friend hadn't spoken to her own abusive father in years. I'm sorry, I wrote. "His love is still with me," she wrote back.

Exclusion Criteria II

Towards the end of March, before I leave, Bettina tells me again about the day that they found Yaminette (Yaminette who crawls into my bed, who re-arranges all my makeup, who sternly tells her playmates that they must ask permission before coming into my room). Yaminette was abandoned in the woods by her birth mother shortly after birth. Her mother had an affair while her husband was in the U.S. and she had gotten pregnant, but her husband decided to come back sooner than expected. So her mother bore her in the woods outside the village and left her there covered lightly in soil. She was found several hours later by someone looking for firewood. They brought her to Bettina, who had always wanted a daughter, and who had enough money to tile her floors, drive a car, and have WIFI in her house.

In the years before the study, Bettina had asked me for help in deciding when to tell Yaminette that she was adopted. Her biological mother lived less than half a mile away in the village, and children had begun to tell her that Bettina was not her real mother. I told her I couldn't tell her what to do but looked up resources and children's books on adoption and shared them with her. I realized that there was no book that would explain how to tell a child that their mother had buried them in a shallow grave in the woods after birth.

I do not know it yet, but I am about to leave the field. It is March of 2020, right at the eve of the COVID-19 pandemic, and in a few days they will close the border behind me after I cross into Chiapas. I will leave the pieces of hair, signed consent forms, and tubes of

spit in the locked cabinet (the one to which only I have the key) for good. Instead, to achieve my doctorate, I will analyze the epigenetics of the placentas of 400 women in Memphis Tennessee. I will not see Bettina or Yaminette again for years.

That night, watching TV with Guillermo, Bettina asks me if she and Yaminette can be part of the study. I hesitate and say no, she isn't your biological daughter. Why does she have to be my biological daughter, Bettina asks.

I stutter. I say something about prenatal programming, about maternal stress hormones crossing the placenta, suffusing the milk, although I feel ashamed because I know Bettina had longed to breastfeed Yaminette.

Then Bettina tells me again about the night they found Yaminette, covered in loam. She was not buried really; she was covered in earth. She was left there with her placenta still attached, Bettina tells me, and this detail breaks my heart. I wonder if that's why she survived, Bettina says, I wonder if it kept her warm.

Chapter 3: No evidence for placental epigenetic aging as a biomarker of structural racism and life-course stressors

Introduction

Racial inequity in maternal-fetal morbidity and mortality is a chilling manifestation of the embodied consequences of racism in the United States today. In searching for proximal mechanisms underlying the maternal-fetal complications that Black and other racialized groups experience at disproportionate rates— risk of preterm birth, the hypertensive disorders of pregnancy, and low birthweight— research has begun to unravel how socioeconomic inequality and life-course stress alters placental function and mediates adverse perinatal and infant outcomes (J. D. Johnson & Louis, 2020; Longtine & Nelson, 2011; Thayer & Kuzawa, 2014). However, the racialized dimensions of lived experiences of inequality and stress have received less systematic scrutiny (Butler, Rivera and Agbai, in prep). This gap is striking given well-established racial inequality in maternal-fetal outcomes implicated by placental dysfunction (Matoba et al., 2021). Neighborhood-level racialized residential segregation and racialized economic inequality directly impact a fetus' prenatal exposure to concentrated poverty, trauma and environmental toxins along with substandard nutrition and access to health care services (Chambers et al., 2019; Gapen et al., 2011; Krieger et al., 2015). An emphasis on individual level factors, however, masks effects of neighborhood-level racialized policies and practices that have been shown to explain racial health inequities more accurately because they heavily influence neighborhood divestment and constrain the behavioral agency of families living there (Bonilla-Silva, 1997; Wallace et al., 2015). In this study, we apply a structural

approach to understanding the impact of racialized inequality and life course stressors on placental function through the lens of epigenetic 'weathering' of the placenta.

Arline Geronimus' notion of 'weathering' has stimulated research using epigenetic phenotyping of the placenta to test for epigenetic age acceleration (Geronimus, 1992; Workalemahu et al., 2021). Geronimus uses 'weathering' as a metaphor to describe increased wear and tear or allostatic load— activation of stress responses, untreated illness, and overwork that result in hypertension, metabolic disorders, and inflammation— that Black and other racialized groups experience in racially unequal societies. Similarly, the placenta undergoes wear and tear through its normal life cycle, a process that may be captured by algorithms for processes of epigenetic aging. Characterizing placental dysfunction can be methodologically challenging due to the need for time intensive histological analysis; epigenetic approaches have bridged the need for complex phenotyping by exploring the role of gene expression and regulation in placental pathophysiology. Briefly, placental epigenetic age algorithms harness large-scale DNA methylation microarray data from placental samples to train algorithms that predict gestational age in healthy pregnancies. Differences between predicted and actual placental age have been associated with preeclampsia, maternal dyslipidemia, and fetal growth (Mayne et al., 2017; Shrestha et al., 2019; Tekola-Ayele et al., 2019).

A Developmental Perspective on Placental Aging

The placenta represents the interface between a pregnant person and their fetus— a key site of intergenerational communication through which a parent provides information about their life course development as well as the quality of the external environment to their unborn child (Gravlee, 2009). Relative to the human lifespan, the placenta is a short-lived organ that nevertheless

undergoes a carefully timed developmental process. Five to six days after fertilization, the human blastocyst forms an outer layer of cells known as the trophectoderm that will eventually form the fetal placenta. At the same time, the maternal uterine lining begins a process called decidualization; the endometrium changes its structure and forms a secretory layer that prepares for implantation. The trophectoderm contacts and penetrates the decidua, and a specialized cells (extra villous trophoblasts) invade the decidua and restructure the ruptures maternal vasculature, bringing the maternal blood exchange gases.in direct contact with the developing structure of the placenta to allow for nutrient and gas exchange. The maternal immune system modulates penetration by the trophoblast, allowing it to invade the maternal tissues while maintaining a careful balance of maternal tolerance and protection. Maternal decidual macrophages and other immune cells aid in the remodeling of the spiral arteries, the uterine vasculature that enables a steady supply of nutrients and oxygen to the fetus. As pregnancy progresses, trophoblast layers will expand and form a network of villous trees whose increasing surface area reflects the needs of the growing fetus. Placental growth slows at the 36th week of pregnancy, although it maintains some proliferative capacity until it undergoes a series of immune and endocrine changes that result in the onset of labor (Fox, 1997).

The timing of labor may be related to normal processes of aging or senescence in the chorionic villi, fetal membranes, and maternal decidua, and these changes may be related to DNA methylation profiles in these tissues (Bianco-Miotto et al, 2016; Sultana et al, 2018). The planned arrest of the proliferative cycle, senescence is a normal feature of post-mitotic cell function. Planned senescence across all body tissues is an important mechanism to suppress tumor formation and remove cells that have acquired DNA damage through environmental insults. Senescent cells cease proliferating and can secrete pro-inflammatory cytokines that recruit immune cells to eliminate them. Placental cells undergo a similar process, which may be an important trigger of labor and parturition (Menon et al, 2016). Dysregulation or acceleration of the normal developmental process

of the placenta may be implicated in adverse perinatal outcomes: for example, excess early fibrin deposition in the spiral arteries may diminish their diameter, a feature known as maternal vascular malperfusion, which has been associated with hypertensive disorders of pregnancy, restricted fetal growth, and preterm labor (Ernst, 2018; Guller et al 2007). Thus, placental accelerated aging (PAA) may have origins not only in the early stages of pregnancy, but also from ongoing exposure to maternal oxidative stress and inflammation throughout gestation

The root causes of placental accelerated aging are an area of active research, but epidemiologic studies have linked stressful life events with many adverse perinatal outcomes of placental origin. Both subjective and physiological stress have been related to maternal vascular malperfusion and/or histological changes in the placenta, yet the epigenetic features that regulate these processes remain an area of active research (Bustamante Helfrich et al., 2017; Ernst, 2018). In early work in this vein, Mayne and colleagues (2017) developed an accurate predictor of gestational age using a 62 CpG site DNA methylation score and found that this epigenetic score predicted ages in pre-eclamptic pregnancies that were significantly 'older' than the actual gestational age of the fetus. Tekola-Ayele and colleagues (2019) used this same score to and found that placental accelerated aging predicted sex-specific patterns of fetal growth. Later work has found racial disparities in PAA and associations with cardiometabolic health (Workalemahu et al 2020).

Although we are not aware of studies that have examined potential roles of structural racism, life-course stress, and DNAm in PAA, several studies have examined PAA through the shortening of telomeres, repetitive sequences of DNA that protect the ends of eukaryotic chromosomes from degradation and regulate cellular senescence (Blackburn, Epel & Lin, 2015). For example, Jones and colleagues (2017) found racial disparities in placental telomere length and evidence that maternal adverse childhood events predict shorter telomere lengths ((Jones et al., 2019). Although not conducted in the context of pregnancy, a recent study invoking Geronimus' weathering hypothesis

mirrors our interest in examining the interplay of structural and individual factors on accelerated aging and racial health inequality. In a community sample of 494 Black adults, Simons and colleagues (2020) found that neighborhood quality, education, income, and racial discrimination predicted accelerated aging using the GriMaternal age DNAm calculator—and that these associations were not mediated by health risk behaviors.

Structural racism

Structural racism has been defined as "the totality of ways in which societies foster racial discrimination through mutually reinforcing systems" such as housing, access to healthcare, nutritious food, and criminal justice involvement (Bailey et al., 2017). We posit structural racism as the upstream or fundamental cause of the adverse environmental conditions that impact women's life course reproductive health, including intergenerational trauma and historical oppression (Phelan & Link, 2013). Rather than emphasizing individual-level experiences or behaviors, a structural approach implicates the macro-level policies, ideologies, and practices that undergird adverse perinatal outcomes in Black women. An emerging body of social epidemiology has begun to operationalize measures of structural racism to understand how they shape lived experiences of institutionalized harm (vis a vis obstetric violence, mass incarceration, and police brutality (Davis, 2019; Duarte et al., 2020; Salas-Hernández et al., 2022), as well as embodied in racialized patterns of adverse maternal-infant outcomes. While multiple measures of structural racism exist and are in use, in this paper we have operationalized structural racism via the Index of Concentration at the Extremes—a census-tract level measure of racialized residential and income inequality (Massey, 1996).

Intersectional methods for social epigenetics: stratification vs. interaction

First described by critical race legal scholar Kimberlé Crenshaw, intersectionality emphasizes that experience, identity, and embodiment cannot be disentangled as additive effects of race gender,

or class (Crenshaw, 2017). Rather, life at the intersection of each of these identities structures experiences, developmental histories, and trajectories of embodied risk and resilience (Bowleg, 2012). The appropriate translation of intersectional methods to quantitative population health research is a subject of robust debate, yet an appropriate modeling strategy should reflect the research question, goals for communication of findings, and limitations to data. Bauer (2014) notes that the metaphorical use of the word "interaction" poses a common challenge both in qualitative research and the use of interaction terms in linear and logistic regression.

Like other users of quantitative approaches, we elect to implement an intersectional approach by stratifying the sample between Black and white women rather than using an interaction term to model the differential impact of structural racism and stressful life experiences (Salas-Hernández et al., 2022). This choice limits our ability to identify whether racialized group differences are statistically significant. We justify this choice on two grounds. The first is methodological. Where group-based confounding of the outcome and covariates is likely, interaction terms formally have been shown to result in biased estimates (Buckley et al., 2017). This problem can be addressed by including interaction terms for each item in the model of the grouping variable. Other research examining life-course stress, racial inequities, and pregnancy-specific stress physiology has found racialized differences in exposures, covariates, and outcomes that would suggest stratification or adjusted product term interaction to be appropriate for the present study. For example, Schreier and colleagues (2015) found that early childhood trauma predicted higher hair cortisol in a cohort of 180 pregnant women, but that trauma rates and hair cortisol varied by race/ethnicity. Following stratification, they found that early childhood trauma predicted hair cortisol only in Black women. A second reason we elect to stratify as opposed to create an interaction term is theoretical. Moving along a scale of racialized deprivation and privilege means different things to individuals in different social positions. A white woman living in a predominantly white, wealthy community brings a

different generational history to that position, and experiences daily life differently there than a neighboring Black woman might. Stratification accommodates potential non-comparability of the actual processes and relations of interest in each group. It also emphasizes that the factors that impact Black maternal health do not need to be directly compared to a white, 'healthy' reference group to take on meaning and public health importance.

Structural Racism and Maternal-infant health

Sociologist Douglas Massey developed the Index of Concentration at the Extremes (ICE) to highlight the self-reinforcing effects of rising spatial polarization of wealth and racial segregation (Massey, 1996). Responding to Massey's call, social epidemiologists such as Nancy Krieger and others have used the ICE to demonstrate the key role of spatial polarization in mediating racial inequities in health otherwise attributed to intrinsic racial behavioral or genetic vulnerability. In a seminal study of more than a million births in California between 2011-2012, Chambers and colleagues (2019) found that women living in the lowest quintile of racialized income inequality had 1.31 odds of preterm birth and 1.71 odds of infant mortality relative to the most privileged quintile. The developmental dimensions of structural racism and their implication for future reproductive health were explored by Shrimali and colleagues (2020), who found childhood exposure to racialized income inequality was associated with 1.12 times the relative risk of giving birth preterm in adulthood. In that study as in others, adjustment for ICE measures significantly attenuated observed Black/white and/or Hispanic/white preterm birth racial inequities oftentimes attributed to individual maternal factors. Furthermore, the intergenerational consequences of structural racism may extend beyond the maternal body and into the history of the neighborhood itself. Another study examining the relationship between historic redlining of NYC neighborhoods showed that historically favorable 'green-lining' was indeed related to lower pre-term birth risk, but only in those neighborhoods that currently contain most wealthy whites (Krieger et al., 2020). A recent systematic

review found that racialized income inequality (as opposed to income inequality or segregation) was especially associated with poorer maternal-infant outcomes (Larrabee Sonderlund et al., 2022).

How structural racism influences exposure to life stressors—particularly during the prenatal period—has emerged as an important research priority for the NIMH and American Psychiatric Association, which in 2021 formed a task force on identifying and dismantling the effects of racism on trauma exposure and psychiatric risk (Wills, 2021). While stressful life event and trauma exposure in pregnancy is known to raise risk of maternal-infant morbidity and mortality, the research into links between stressor exposure, structural racism, and maternal-infant health is still nascent. In their conceptual model and review, Anglin and colleagues (2021) note that Black Americans experience greater childhood trauma and perinatal complications than white counterparts and suggest that inequality experienced from multiple overlapping social determinants of health may contribute to Black/white inequity in psychosis risk. Furthermore, the same trauma indices may be embodied differently by women experiencing overlapping forms of structural violence. In two studies that assessed prenatal hair cortisol, trauma, childhood maltreatment and prenatal stressful life events were associated with higher hair cortisol levels only in pregnant participants who identified as Black (Schreier et al., 2016, 2015).

The developmental timing of exposure to adversity (e.g., in early life or during pregnancy) has been shown to leave lasting imprints on maternal physiology relevant to placental function and future infant outcomes (Aschbacher et al., 2021; Steine et al., 2020). Given these effects, we examine how ICE_{race} (segregation) and $ICE_{race*income}$ (racialized income inequality) and stressors experienced during childhood, before becoming pregnant, and during the pregnancy itself. We hypothesize that the life course stress and structural racism measures will predict placental age acceleration, but that the inclusion of structural racism in the model will attenuate the impact of stressors as it addresses the fundamental racialized causes of lived experiences of inequality and trauma. We further

hypothesize that the effect size of structural racism on placental age acceleration will be greater among Black participants.

Methods

Participants and procedures

Participants were drawn from the CANDLE (Conditions Affecting Neurocognitive Development and Learning in Early Childhood) Study cohort, a community birth cohort recruited during the first trimester of pregnancy in participating hospitals in Shelby County, TN and led by the Urban Child Institute (UCI) and University of Tennessee Health Science Center (UTHSC) (Sontag-Padilla et al., 2015). 1503 participants in their second trimester of pregnancy were recruited between 2006 and 2010 from participating community prenatal clinics via media campaigns and clinic-flyers. Women were considered eligible for enrollment if they resided in Shelby County, were between 16 and 18 weeks of gestation, were between ages 16-40 years, had a low-risk singleton pregnancy,[20] spoke English, and were planning to deliver at one of five study medical centers[21]. Relative to the population in Shelby County, CANDLE mothers were more likely to identify as Black or non-Hispanic white than any other race/ethnicity, have higher incomes, and higher levels of education (Sontag-Padilla et al., 2015). All participants provided informed consent prior to enrollment.

Demographic data were collected in an in-person interview during the second trimester visit by trained research assistants and study staff. Life-course trauma was assessed via an in-person interview during the third trimester (Traumatic Life Events Questionnaire) and a phone interview

[20] Low risk pregnancy was defined by UTHSC as lacking a diagnosis of gestational diabetes, hypertension, iron-deficiency anemia, insulin-dependent diabetes, cardiovascular, endocrine, renal, or collagen disease, ruptured or prolapsed membranes, oligohydramnios, placenta previa, HIV, or major fetal anomaly.

[21] Baptist Memorial Hospital—Memphis; Methodist Le Bonheur Germantown Hospital, Germantown,; Regional Medical Center, Memphis; Saint Francis Hospital—Bartlett; Saint Francis Hospital—Memphis.

(Prenatal Stressful Life Events, Adverse Childhood Events) during the 8th study wave (8 years postpartum). Labor, delivery and birth outcomes were assessed from medical records abstraction by a trained obstetric nurse.

Placental samples were collected within 15 minutes of delivery by a trained obstetric nurse, who dissected a 2 x .5 x .5 cm rectangular prism of tissue from the placental parenchyma, which was then cut into four .5 cm cubes and placed in a 50 mL tube with 20 ml of RNAlater, refrigerated at 4° C overnight, and then frozen individually at -80°C with fresh RNAlater the following day. Fetal villous tissue was manually dissected from the maternal decidua, placed in additional RNALater, and stored at -°80 C prior to DNA extraction at the Kobor lab at UBC. [22]

Sample Processing and Array Data Generation

Extracted genomic DNA samples were purified, bisulfite-converted using EZ-96 DNA Methylation kits (Zymo Research, Irvine, CA), whole-genome amplified, hybridized to the 850K arrays, and were scanned by a fluorescent scanner (Illumina). The intensity of fluorescence from the raw IDAT files were read in the R programming environment (R 3.5) where the level of DNA methylation for each targeted CpG site was estimated as a β value, a number between 0 and 1 (0 = no methylation, 1 = fully methylated).

Placental DNA methylation was quantified using the Illumina Infinium Human MethylationEPIC BeadChip (850K array). This platform quantifies DNA methylation at 866,895 cytosine–phosphate–guanine (CpG) sites across the whole genome (Moran et al., 2016). To avoid potential confounding with biological and technical variables, we adopted a stratified randomized design and distributed samples approximately equally across chips and plates based on tissue type, sex, Medicaid status, hospital/birthing center and race. Additionally, we incorporated tissue-specific

[22] The placental collection methods will be updated with additional information in the final manuscript

technical replicates to monitor different stages of preprocessing and to determine the background technical noise per tissue.

DNA methylation preprocessing

We performed multiple sample quality control checks to determine if the samples met quality standards for inclusion in the downstream analyses. Specifically, we utilized the R ewastools package (R v 4.2) to confirm if samples passed the typical control metrics such as array staining, extension, restoration, hybridization, specificity, target removal and bisulfite conversion. Subsequently, functions in the minfi package were used to: i) check whether samples clustered together based on median intensities in both the methylated (M) and unmethylated (U) channels, ii) identify samples that exhibited bad detection p values in > 1% of their probes, iii) detect samples that had <3 beads contributing to the DNA methylation signal in >1% of their probes, and iv) predict sex of samples based on median intensities of the X chromosome probes and Y chromosome probes and confirm inferred sex with reported sex.[23] Sex mismatches were further investigated using the Conumee package which computes genomic copy number estimates. After sample quality control, background correction with dye bias equalization was performed using the minfi package followed by beta mixture quantile dilation normalization in the wateRmelon package to correct for probe-type differences on the 850K array. Next, based on the Pidsley et al. (Pidsley et al., 2016) annotation, cross-hybridizing probes that nonspecifically bind to different regions of the genome and cross-reactive probes that had a SNP at the measured CpG site and overlapping the single base extension locus were eliminated. Further, SNP probes, XY probes, and poorly performing probes with bad detection p-value >0.01 and with missing bead count of more than 3

[23] I will add the number of sex mismatches and samples with poorly performing probes that were removed in the final manuscript.

beads in 5% of the samples were removed. Finally, batch effects, specifically associated with Chip Id, Chip position, and Run were corrected using the sva package.

Sample cell type composition was assessed using the planet package, a reference-based deconvolution algorithm developed for placental DNA methylation with the EPIC array (Yuan et al., 2021). Principal components (PC) were then calculated to account for estimated cell type variability within future model creation. Robust isometric log ratio (ILR) PCA in the robCompositions package, appropriate for composite data ((Filzmoser et al., 2009)), was used to create principal components of the proportions estimated from the reference set with a small offset of 0.0001. For placental samples, the top three PCs represented >90% of the estimated cell type proportion variability for the six inferred cell type proportions (syncytiotrophoblasts, trophoblasts, Hofbauer cells, endothelial cells, stromal cells, nucleated red blood cells (nRBCs)). All models were run using the three PCs as covariates as well as only covarying for syncytiotrophoblast proportion. Results did not differ meaningfully with PCs vs. syncytiotrophoblast-only covariates, and so the sycytiotrophoblast-only covariate models are presented here.

Measures

Outcome

Placental accelerated aging

Placental epigenetic age was estimated using the Control Placental Clock (CPC) developed by Lee et al (2019). Briefly, investigators selected multiple publicly available training datasets with known prenatal history and delivery outcomes and genome-wide DNA methylation (Illumina EPIC array or the prior 450k array) and regressed gestational age on DNAm using penalized regression (elastic net regression). The result is a subset of CpG sites whose weighted average is the predicted

gestational age. The training set for the CPC included 963 normal pregnancies without diagnosis of preeclampsia, hypertension, diabetes, chromosomal abnormalities, or chorioamnionitis. Estimated gestational ages were calculated using EstiMaternal age, a websever-based hub for DNA methylation-based age computation across multiple tissues, including placenta (Di Lena et al., 2021). Two samples were dropped due to inability to predict CPC ages. The difference between predicted and actual gestational age was used as the outcome in all analyses.

Predictors

Racialized Inequality: Index of Concentration at the Extremes (ICE)

Two indices of structural racism, segregation and racialized income inequality, were measured with the Index of Concentration at the Extremes (ICE)(Chambers et al., 2019). ICE_{race} and ICE_{race_income} are standardized measures of racial segregation and income inequality at the census block level that take on values from − 1 to 1. Values less than zero correspond to living in a community that is highly minoritized and/or low income and positive values correspond to census blocks that are highly white and/or high income. ICE was calculated using the following equations:

$$ICE_{race} = (\text{white population - Black population})/ \text{Total population}$$

$$ICE_{race*income} = (\text{white population with annual income > \$100k -Black population with annual income < \$25k})/ \text{Total population}$$

ICE_{race} and $ICE_{race*income}$ measures were calculated for Shelby County census blocks using 5-year estimates from the American Community Survey 2010 – 2015 data, which correspond to the period in which CANDLE mothers were enrolled. Participant addresses at enrollment were geocoded and linked to the corresponding ICE indices for their respective census blocks.

Adverse Childhood Events (ACE)

Exposure to childhood adversity was measured by an adapted version of the Adverse Childhood Events (ACE) inventory from the Centers for Disease Control and Prevention–Kaiser ACE Study (Felitti et al., 1998), collected in a follow up wave 8 years postnatally. This 9-item measure inquires whether the participant experienced childhood maltreatment (i.e., physical, emotional, sexual) and/or household challenges (witnessing family violence, living with a family member with substance use disorders, mental illness, caregiver separation or divorce, family member incarceration, and economic hardship) before age 18. This measure collapsed the emotional maltreatment and emotional neglect items from the traditional ACEs questionnaire into one item for brevity. Adult retrospective vs. prospective recall of ACEs have been shown to be moderately good in a large longitudinal study, with both retrospective and prospectively collected ACEs predictive of mental and physical health outcomes(Reuben et al., 2016). The total score was used in analysis.

Prenatal Stressful Life Events (PSLE)

Pregnancy-specific stressors were assessed retrospectively at year 8. Maternal report of the number of stressful life events (SLE) that occurred during pregnancy was assessed. SLE were assessed with a list of 14 events adapted from the Centers for Disease Control and Prevention Pregnancy Risk Assessment Monitoring System (PRAMS) survey (Shulman et al., 2018). Participants were asked for yes/no responses to statements about experiences with illness, death, relationship problems, housing difficulties, legal issues, and financial problems during pregnancy. While we are unaware of any work that has specifically tested prospective vs. retrospective recall of PSLE, research has documented that prenatal experiences related to pregnancy and birth are recalled at high levels (Ramos et al., 2020). As in the research on early life adversity, retrospectively recalled prenatal adversity may not match prospective reports, yet still be informative as to the

kinds of experiences participants codify into memory as influential and impactful. The total score was used in analysis.

Stressful Life Events (SLE)

Stressful life events were assessed using the Traumatic Events Life Events Questionnaire, an index of 21 potentially traumatic life events ranging from impersonal (disaster, accidents) to highly personal (physical abuse, sexual assault) (Kubany et al., 2000). Participants indicated if they had ever experienced any of the events. Three of these items assess child maltreatment and poverty. Because of their similarity with ACEs items covering these experiences, these items were subtracted from the total score, and the remainder was used in analysis.

Data Analysis

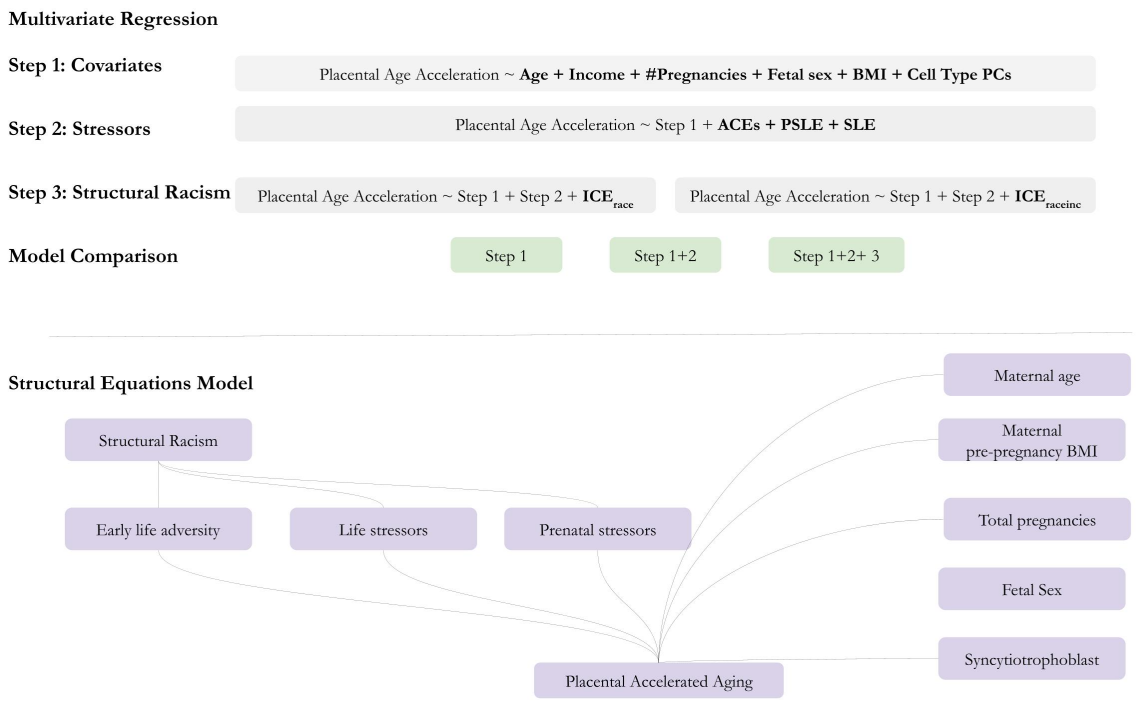
Multivariate regression and SEM of ICE, ACEs, PSLE, and SLE

We calculated descriptive statistics for demographic, stressor, and structural racism measures between Black and white women. Power analyses were conducted using the pwr package, and revealed that to detect a small effect size (d = .2) at 80% power with 12 degrees of freedom, 108 subjects would be needed. Given our stratified sample sizes of n_{Black} =242 and n_{white} = 175, we felt we were appropriately powered to continue. We conducted four sets of hierarchical regressions to examine the joint impact of ACEs, PSLE, SLE, and either ICE_{race} or $ICE_{race*income}$, on accelerated placental age. Models are stratified by maternal self-identified Black or white race. In the first step, we included maternal age, pre-pregnancy body mass index, income adjusted for family size, education, total number of pregnancies, fetal sex, and proportion of syncytiotrophoblasts. In the second step, we included ACEs, PSLE, and SLE total scores. In the final step, we added the ICE_{race} or $ICE_{race*income}$.. Multi-collinearity was assessed through variance inflation factor and visual inspection

of diagnostic plots (see Supplemental Figures 2-6). We then conducted model comparisons between the covariate-only, covariate + stressors, and covariate + stressors + structural racism models using Akaike Information Criterion. Predictors were standardized before analysis to facilitate interpretation across models. The subsample selection process and data analysis strategy are summarized in Figure 1.

In a final exploratory analysis, we computed a structural equation model of the full models for ICE_{race} and ICE_{racein} using the lavaan package.(Rosseel, 2012)) to estimate the associations between structural racism, life-course stressors, and accelerated placental aging. As in the multivariate regression, we included maternal age, pre-pregnancy BMI, adjusted income, total pregnancies, fetal sex, and syncytiotrophoblast proportion as covariates. Due to significant right-skew, the variance inflation transformation for count data (square-root transformation) was applied to ACES, PSLE, and SLE. We fit models using the resulting covariance matrix and estimated model parameters using the maximum likelihood estimator. We assessed model fit using the comparative fit index (CFI) and root mean square error of approximation (RMSEA), using a CFI cutoff of >0.9 as acceptable and >.95 as excellent fit and RMSEA values <.08 as acceptable and <.06 as excellent model fit. (Hu & Bentler, 1999).

Figure 1. Analysis plan



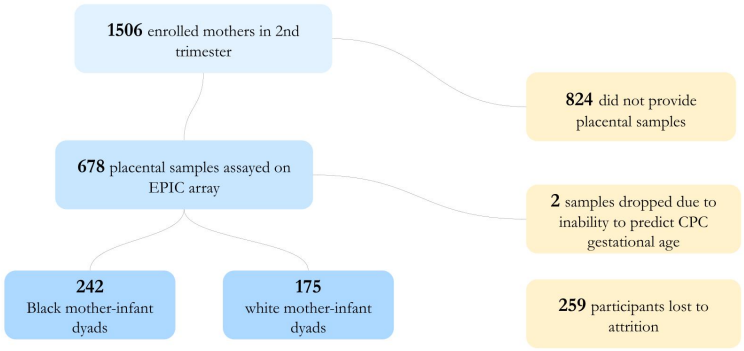
Assessment of missing data and subsample selection

All statistical procedures were conducted in R 3.5 statistical programming software (R-Core-Team, 2020). 677 participants had complete placental epigenetic age acceleration, but missing data for demographic, structural racism, and trauma measures ranged between 0 - 33.3% missing. Most missing data was due to study attrition in mothers who did not participate in the Wave 8 interview collecting ACEs and PSLE (33.3% missing each, see Supplemental Figure 1). T-tests and chi-squared tests revealed no significant differences between attrition and completion in income, race, stressful life events, infant sex, or term delivery. Maternal age was significantly lower in cases lost to follow-up ($M_{attrition}$=26.2, $M_{completion}$=27.4, p=.004). The mice package was used to apply Jamishidian and Jalal's test for missing completely at random/missing at random (MCAR/MAR), and both

Hawkin's and Anderson-Darling's test were significant ($p < .001$), indicating rejection of the null hypothesis that data was MCAR (Jamshidian & Jalal, 2010). Due to this we pursued complete case analysis.

The ICE measures examine Black/white inequality in income and residential segregation. For this reason, we chose to subset only those mothers who identified as either white (38.9%) or Black (58.2%), resulting in the exclusion of 6 participants (0.8%). While this sample largely identified as Black or white, this approach masks important intersections for other racial and ethnic identities, such as multi-racial, bi-racial, Indigenous, and Latinx identities. There were, however, few participants who identified as Latinx (2.1%), of whom half (n=5) identified as white and half (n=5) identified as Black. Race-stratified analyses were conducted in two datasets, one containing complete cases of Black self-identified mothers (n=242) and one containing complete cases of white self-identified mothers (n=175). See Figure 2.

Figure 2. Subsample selection



Results

We hypothesized that ACES, PSLE, and SLE would be associated with placental age acceleration, and that these associations would be attenuated by including the ICE segregation and racialized income inequality measures. We also hypothesized that the effect size of the ICE measures would be greater for Black participants than for white participants. We found no evidence to support these hypotheses, and some evidence that the observed effects were reversed (e.g., trauma was most associated with structural racism in white, not Black women). Descriptive statistics for white and Black mothers are provided below in Table 1. These reveal the enduring racialized socioeconomic inequality that characterizes Shelby County; Black mothers had lower levels of education, income, and higher pre-pregnancy BMI. By contrast, rates of life course trauma in early childhood, pregnancy, and across the lifespan were generally low and relatively similar among Black and white women enrolled in the study, although SLE and PSLE totals were slightly higher among Black women.

Table 1. Descriptive Statistics

	Identifies as Black n = 242	**Identifies as white** n = 175
Maternal Age	25.7 (5.3)	29.5 (4.9)
Total Pregnancies	2.8 (1.7)	2.3 (1.4)
Maternal education		
<HS	21 (8.7%)	7 (4%)
HS/GED	140 (57.9%)	38 (21.7%)
Technical School	32 (13.2%)	12 (6.9%)
College	40 (16.5%)	66 (37.7%)
Graduate/Professional	9 (3.7%)	52 (29.7%)
Annual income	14,830.4 (14,108.3)	33,530.3 (16,960.5)
Pre-pregnancy BMI	29.7 (8.2)	26.0 (6.1)
Fetal sex		
Female	124 (51.2%)	89 (50.9%)
Male	118 (48.8%)	86 (49.1%)
Adverse Childhood Experiences	2.5 (2.5)	2.2 (2.6)
Stressful Life Events	3.6 (2.6)	3.1 (2.3)
Prenatal Stressful Life Events	1.7 (1.9)	1.4 (1.7)
Segregation ICE_{race}	-0.5 (0.5)	0.4 (0.4)
Racialized Income Inequality $ICE_{race*income}$	-0.1 (0.1)	0.0 (0.1)
Predicted – Actual Gestational age (weeks)	-.0.9 (1.2)	-1.3(1.1

Note: Standard errors in parentheses.

The structural racism measures revealed distinct racialized residential segregation but relatively limited variability in racialized income inequality (see Figure 3). Black women and white women lived in distinctly racialized census tracts. Black women live in census tracts with a mean ICE_{race} score of -0.5, indicating that they live in highly racially segregated communities. White women also live in heavily segregated neighborhoods with mean ICE_{race} measures of 0.4. By contrast, $_{ICErace*incomeome}$ exhibited less variability across census tracts. This indicates that there were few census tracts where Black/white income differences were very high; given the racial socioeconomic differences and residential segregation, it may be that few low-earning Black and high earning white participants resided in the same census blocks. Similarly, those that were more racially integrated may have had greater income equity. In terms of placental age acceleration, white mothers had significantly higher predicted placental age acceleration compared with Black women ($p<.05$), but distributions were largely overlapping.

Descriptively, bivariate correlation plots revealed potential differences in the relationship between structural racism, income, and life-course trauma between Black and white participants. In white participants, there were negative associations between ICE_{racinc} and ACEs ($r = -.27$, $p <.05$) and SLE ($r = -.23$, $p < .05$) such that participants living in neighborhoods with greater racialized income inequality reported less early life adversity and stressful life events. The same pattern appeared for ICE_{race}, where white participants living in more segregated white neighborhoods reported less early life adversity ($r = -.24$, $p < .05$) and stressful life events ($r = -.25$, $p < .05$). Income also served as a traditional risk factor for white participants, with greater income associated with less trauma exposure across early life ($r =-.29$, $p < .05$), the prenatal period r-.37, $p < .05$), and cumulatively ($r= -.20$, $p < .05$). Put differently, White participants currently living in wealthier, homogeneously white neighborhoods are less likely to have experienced trauma early in life.

Strikingly, these associations were not present for Black participants. Although trauma measures were all intercorrelated as among white participants, no significant correlations between segregation, racialized income inequality, or income emerged among Black respondents. In addition, for both Black and white respondents, neither the trauma, inequality, nor demographic measures were correlated at the bivariate level with the outcome, placental age acceleration (see Supplemental Table 1 and Supplemental Table 2).

Figure 3. Distribution of key study variables and ICE across Shelby County census tracts

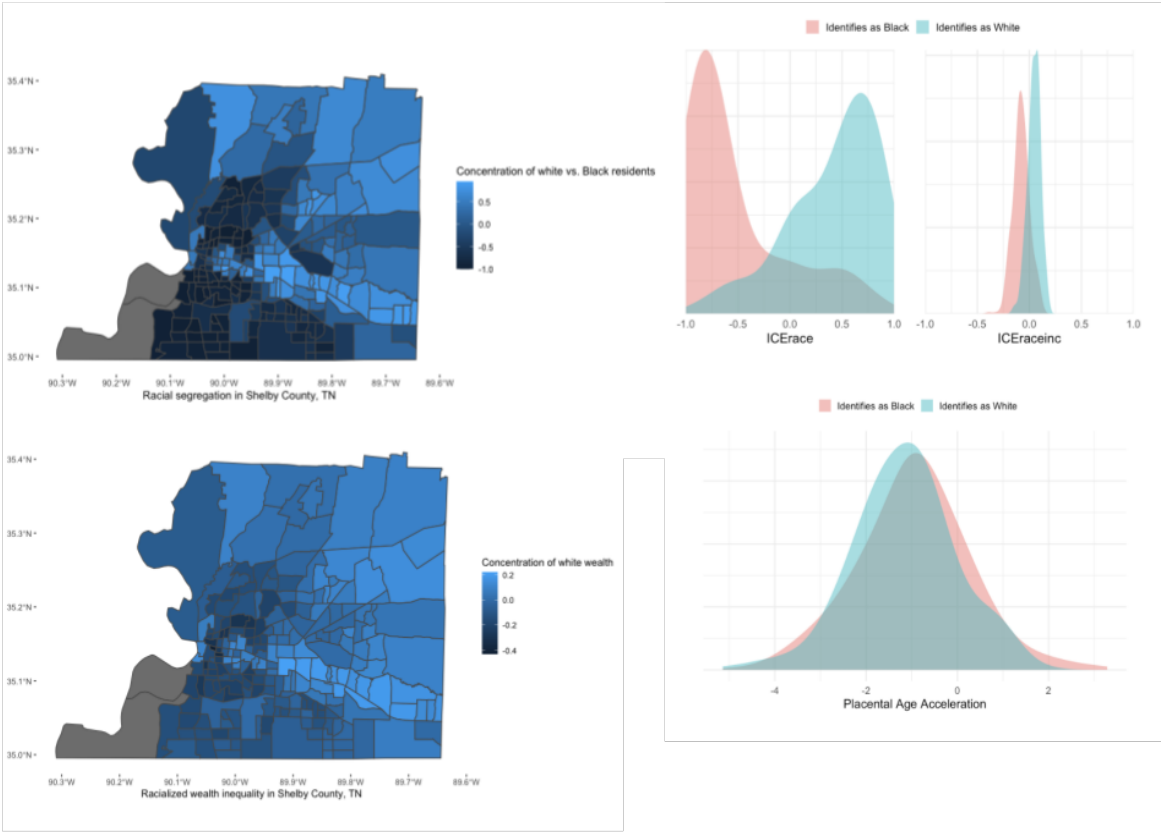
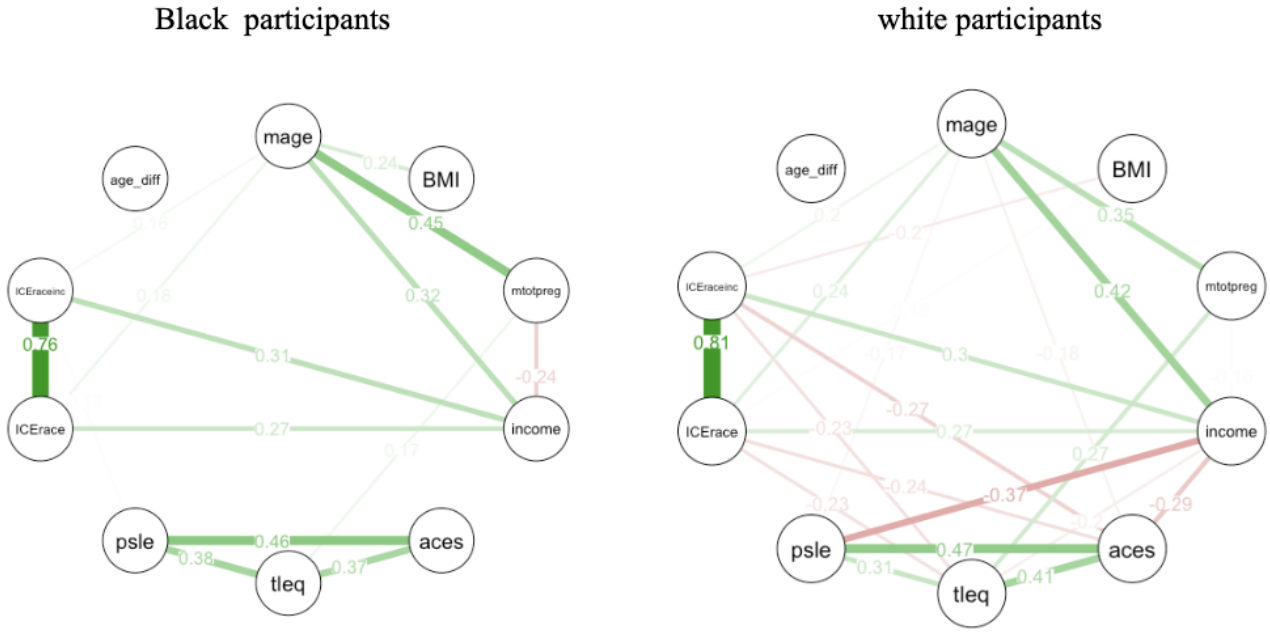


Figure 4. Pearson correlations between study variables



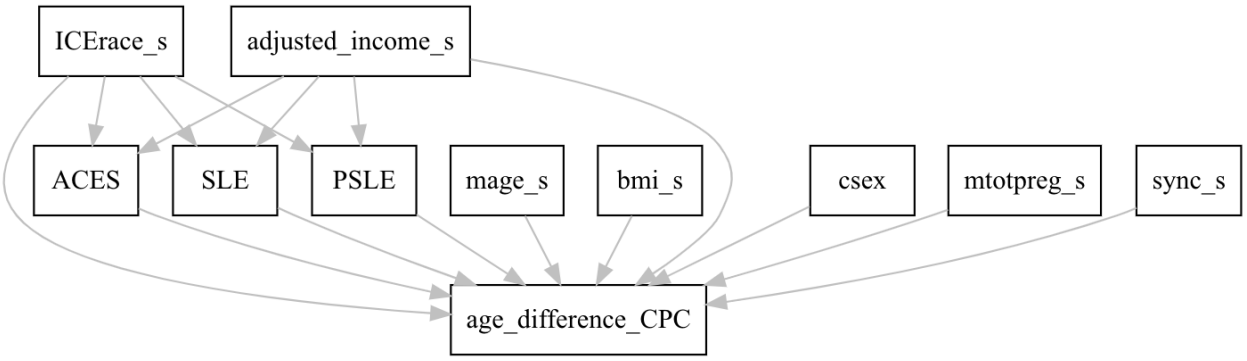
Multivariate regression and SEM of ICE, ACEs, PSLE, and SLE

Multivariate analyses confirmed the lack of association between hypothesized predictors and placental age acceleration. No demographic, trauma, or structural racism (e.g., ICE_{race} and $ICE_{race*income}$) predicted epigenetic placental age acceleration (See Supplemental Tables 3-10). None of the overall models was significant (all $R^2 < .06$, all $p > .05$). Model comparison using AIC for each set of analyses preferred the covariate-only model in each set of analyses (see Supplemental Table 11X).

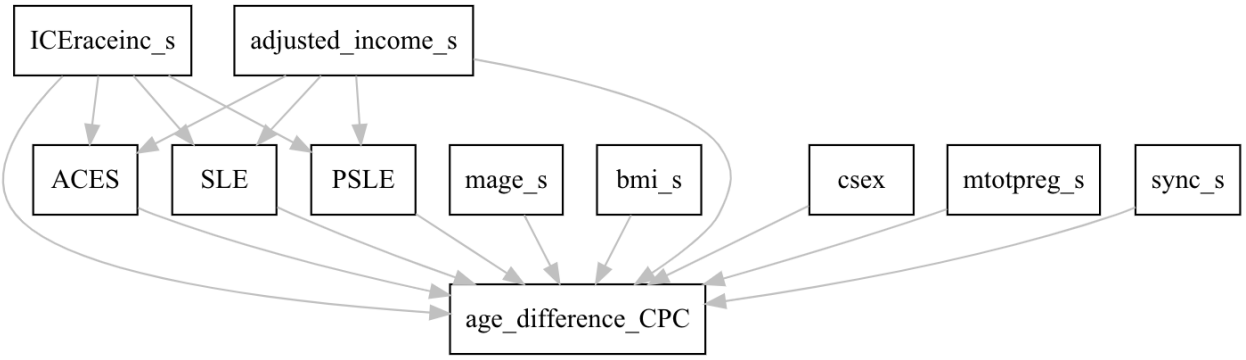
Patterns observed in the bivariate correlations as well as multivariate regression were supported by the structural equation models. Indices of fit for all models were poor, with none of the models achieving acceptable RMSEA or CFI. This is expected given the lack of any associations with placental age acceleration and study variables noted earlier. Of note, as in the bivariate analysis, both ICE_{race}, $ICE_{race*income}$, and income were associated with life-course stressors in white participants but not in Black participants. In white participants, ICErace (living in a whiter census tract) was associated with fewer ACEs (B = -.38, p <.05) and life stressors (B = -.45, p <.01). In the segregation models, higher income was also associated with lower prenatal life stressors (B = -.34, p <.01) and lower ACEs (B = -.28 p <.01), again only in white participants. This pattern was repeated and exaggerated in the racialized income inequality models. Again, for white women only, living in a neighborhood with a higher proportion of high-income white residents vs. low-income Black residents was associated with fewer ACES (B = -1.89, p <.05) and cumulative life stressors (B = -1.6, p <.05). In the racialized segregation model, in white women only, greater income again was associated with fewer ACES (B = -.34, p <.05) and prenatal stressful life events (B = -.39, p <.05).

Figure 5. Structural equation model results

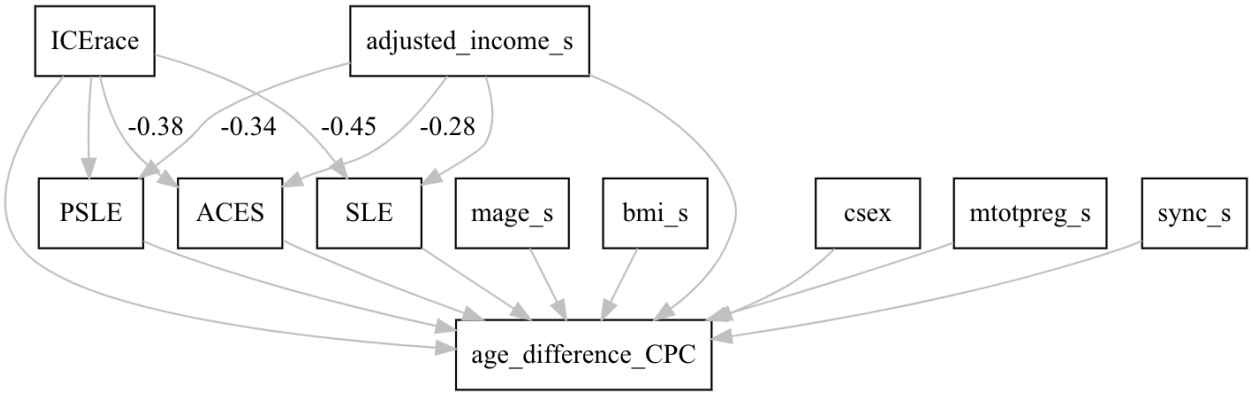
Identify as Black: Segregation



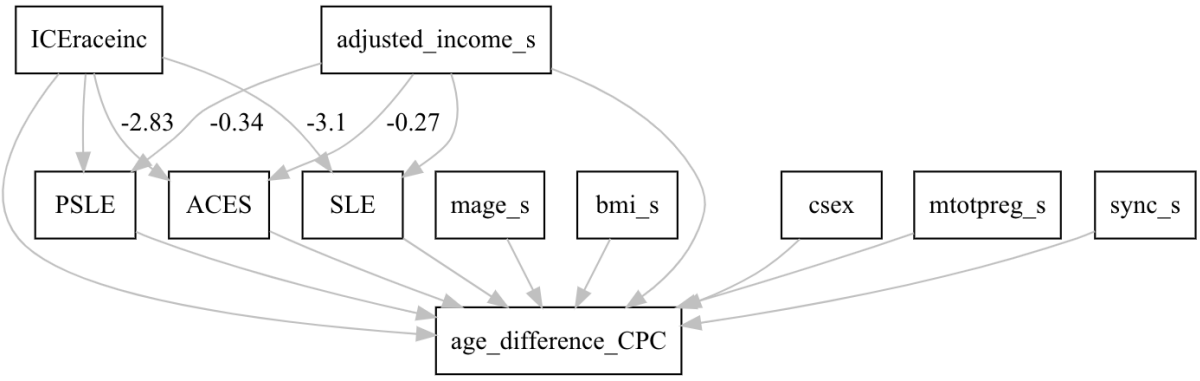
Identify as Black: Racialized income inequality



Identify as white: Segregation



Identify as white: Racialized income inequality



Discussion

Based on the Control Placental Clock (Lee et al., 2019), we found no evidence that differences between actual and predicted gestational age using were related to structural racism, life course stressors, or demographic variables in CANDLE mothers and children. We hypothesized that structural racism and life course stressors would be linked to accelerated placental aging in the full sample, and that this would be especially true among Black women. We find no evidence in support of this hypothesis. Along with this main finding, we also report key differences in the

conditions that pregnant Black and white women experience living in Shelby County. The mean annual income adjusted for family size for Black participants ($14,830.4) was nearly $19,000 less than white participants ($33,530.5), and hovers just over the 2015 federal poverty level of $11, 700. Although there were small differences in the total number of traumatic events Black and white participants experienced, these paled in comparison with the stark inequities in income and education lived out in highly racially segregated environments and neighborhoods.

Spatial models of health consider that the environments that individuals reside in shape their health by shaping their everyday experiences. Along with such processes, environments also indicate the economic and structural precarity of the people who live in them. In this paper, we instantiated the first view— structural racism (indicated by racialized income inequality and segregation) fundamentally shapes stress exposure in ways that are embodied in placental epigenetic age. Along with the null finding for placental age acceleration, we also found that this view of socially mediated stress was only partially valid for white, but not Black, participants. At the bivariate level and in our structural equations modelling, segregation and racialized income inequality were predictive of adverse childhood events and stressful life events only in white participants. Similarly, expected relationships between income and stressful life events emerged only for white women.

To further interpret such racial disparities, we turn to the second view of environments as indicators of precarity. The racialized pattern of incomplete buffering of risk by socioeconomic gains (and in this case, residential segregation) that we observed has been understood as 'status incongruity' by anthropologists and health psychologists (McDade 2018). Status incongruity refers to acquisition of prestigious status by a historically marginalized individual or group, who is then unable to reap the benefits of higher status due to social pressure to conform to extant social roles and expectations (William W. Dressler, 1995). For example, first-generation college graduates may experience some of the benefits of increased education—e.g., greater opportunities for

employment—but due to diminished intergenerational wealth and/or ongoing obligations to low-income family may not be able to afford similar patterns of consumption, homeownership, or other markers of social status that set them apart from peers. Status incongruity may be realized as lifestyle incongruity, when a desired and enacted lifestyle (patterns of consumption, leisure, and wealth displays) is incongruent with education, socioeconomic status, and social resources needed to adequately maintain and perform said lifestyle. McDade (2001) examined the embodied effects of lifestyle incongruity in a rapidly acculturating group of Samoan adolescents, finding that Epstein-Barr antibodies (a measure of stress-related immune suppression) was highest in those families where material lifestyle outpaced socioeconomic resources. Similar findings—for example, that status incongruity rather than socioeconomic status itself drives stress related outcomes— have also been reported from Puerto Rican and Brazilian samples (W. W. Dressler, 1999; William W. Dressler et al., 2017; Gravlee & Dressler, 2005).

Research on status incongruity and stress tends to emphasize that challenges of not being able perform expected social roles as an internal experience of the individual of incongruent status. The present study shines a light on how neighborhood status incongruity also reflects systematic privilege of those who are not 'incongruent'. Black participants experienced more traumatic events than their white counterparts living in mostly white neighborhoods. Such racial, rather than lifestyle, dimensions of status incongruity may be attributable to the ways structural racism influences the likelihood to experience loss, family conflict, economic insufficiency, failures of protection, parental and partner incarceration, and substance use from which Black women in white neighborhoods were unequally buffered (Duarte et al., 2020; R. M. Johnson et al., 2021). While high socioeconomic status white families may access credit or home equity (Freeman, 2016; Perry, 2019) to cover economic shortfalls or avoid justice system involvement due to racial bias, Black families who were economically mobile and moved to white neighborhoods may suffer similar or worse surveillance,

discrimination and economic precarity than those who remained in Black enclaves (Hannon et al., 2021; Petrocelli et al., 2003). In addition, the resources white families may use to mitigate trauma exposure, such as social support, kin networks, and religious communities may also be less available to Black families in white majority neighborhoods (Hope et al., 2017; Kramer & Hogue, 2009).

Intersectional approaches to social risk, racialization, and family wellbeing shed light on the ways in which traditional forms of "risk" may operate differently depending on racial identity. In his study of the Fragile Families Study, Williams (2021) found that white married mothers rated their relationship quality as better than dating or cohabiting mothers, but that these differences were attributable to differences in maternal cumulative risk (health problems, economic risks, partner incarceration, and social support). In Black mothers (but not white mothers), relationship quality and marital status were not consistently associated. Moreover, cumulative risk and marital status interacted to predict relationship quality, such that Black married mothers (vs. unmarried) experienced steeper decreases in relationship quality with increasing cumulative risk, a reversal of the supposed 'buffering' effects of marriage. Williams argues that intersectional analyses reveal that structural racism operates through racialized ability or inability to benefit from institutions and policies, including marriage. In related ways, our analysis reveals that traditional measures of social risk—living in a neighborhood that is whiter, wealthier and whiter, or having a greater income—is associated with reductions in life stressors for white, but not Black women. This finding emphasizes that rather than Black vulnerability, structural racism in this paper seems to operate via white participants' privilege in benefitting for social buffers.

Although accelerated placental aging was not associated with any of the predictors in either white or Black women, other recent work has shown that Black women living in white neighborhoods may not benefit in terms of perinatal health. A recent study found that the highest risk of low birthweight newborns—a stunning 14.5%—occurred among high income Black women

residing in predominantly white neighborhoods (Kothari et al 2016). Thus, findings from this study do not indicate that childhood trauma and neighborhood deprivation have no impact on the placenta and its function. Rather our results suggest that placental aging algorithms may not distinguish degrees of pathological processes of weathering or placental dysfunction in a sample with low levels of known perinatal pathology.

This insight may seem trivial but reveals gaps in our and other social epigenetic researchers' expectations for what aging algorithms truly reflect and what they can help us understand. That is, among nearly all aging algorithms, the difference between a predicted age and an actual chronological age are thought to contain biological meaning or information, rather than error. Epigenetic age is a prediction using an algorithm that was trained on a particular subset of data and then ported to a new data source. We might also term it a "polyepigenetic risk score for age" to draw attention to its conceptual sibling, the polygenetic risk score. The portability of polygenetic risk scores for various disease states across populations has been demonstrated to be problematic: training machine learning models on the genes and outcomes in one population does not necessarily provide insight when that prediction algorithm is deployed in another with different population structure, ancestry, or dynastic effects (Martin et al., 2019). None of the DNA methylation sites whose weighted average makes up the placental clock used in this paper (and many other currently published epigenetic clocks) exist in any genes within gene pathways known to relate to cellular senescence (Lee et al 2019).

While race, ethnicity, and socioeconomic status are not consistently reported in documentation of training datasets, the information that is available indicates participants in the training datasets differed from CANDLE mothers. For example, the New Hampshire Birth Cohort Study and Rhode Island Child Health Study, and 3D cohort are all predominantly middle-class and white, with the next most common race/ethnicity being Latinx or Asian/Pacific Islander (Appleton

et al., 2015). Although demographics in the other datasets used in training are not reported, most took place in medical centers in Vancouver or Toronto, Canada, where the estimated Black population is 1-9% (Statistics-Canada, 2019). Researchers who have deployed epigenetic aging clocks in populations very different from the biobank-based training data have found they fail to replicate; recent work examining 15 different maternal epigenetic clocks in a cohort of women and infants from Cebu, Philippines found none correlated with infant gestational age as they had in the U.S. based training sample (Ryan et al., 2022). Data such as this suggest that, like polygenetic risk scores for chronic disease or behavioral outcomes, epigenetic aging algorithms (as polyepigenetic risk scores for age) may not index universal causal processes in aging but may reflect population structure, its enmeshed social and genetic confounds, or other forms of statistical bias (Pingault et al 2022). A lack of portability of epigenetic aging algorithms across populations poses important questions for researchers who, as we did in this paper, hypothesize that differences between predicted and actual age are biologically meaningful. If such algorithms perform poorly in new datasets, it may be the data they were trained do not provide sufficient generalizability to novel datasets under investigation.

The existence of racial disparities detected by clinical algorithms also may naturalize them, entrenching the belief that Black Americans inherently have worse kidney or pulmonary function, delaying needed treatment and exacerbating health inequalities (Ledford, 2019). Sitting at the contact point between genome and exposome, epigenetics has been heralded as the proof that environmental inequality, not deficient racialized genetic variation, is the fundamental cause of health inequality (Mulligan, 2021). The research presented here provides a cautionary counterpoint to what can and should be inferred from epigenetic data in the context of the substantially different life experiences and embodiments of Black and white families in Shelby County.

Limitations

This study has several important limitations. Address data for mothers was available only during pregnancy; hence, their exposure to structural racism and the impacts of residential mobility before and after that time are unknown. We restricted our outcome to placental epigenetic age acceleration, although future work might examine other important determinates of placental function such as maternal hypertension and immune function. The ACES, PLSE, and SLE measures were not gathered prospectively and may be subject to recall bias. Finally, relative to other epigenetic clocks, placental clocks are in their own infancy, and subsequent refinements may help clarify what we can and cannot learn from them relative to placental function, physiology, and maternal-fetal health.

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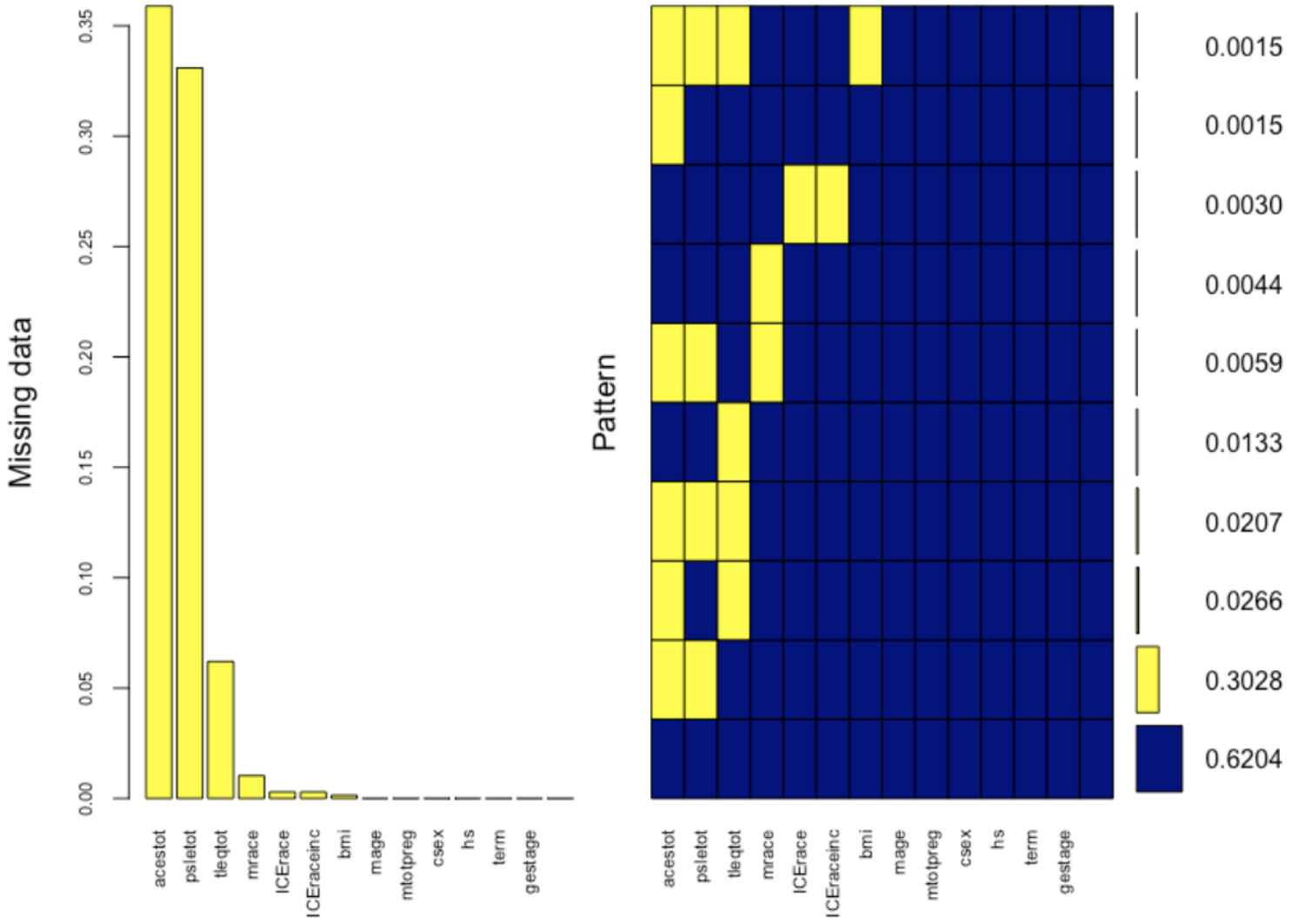
Chapter 3 Appendix

Supplemental Figure 1.

Missing Data Pattern



Supplemental Table 1.

Means, standard deviations, and correlations with confidence intervals for white participants

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. Maternal age	29.55	4.95								
2.BMI	26.05	6.10	-.03 [-.17, .12]							
3. Total Pregnancies	2.27	1.41	.35** [.21, .48]	-.04 [-.19, .11]						
4. Adjusted income	33530.32	16960.54	.42** [.29, .54]	-.14 [-.29, .01]	-.16* [-.30, -.01]					
5. ACEs	4.37	5.12	-.18* [-.32, -.04]	-.01 [-.16, .14]	.10 [-.05, .25]	-.29** [-.42, -.15]				
6. TLEQ	6.13	4.65	.03 [-.12, .18]	-.06 [-.21, .09]	.27** [.13, .41]	-.20** [-.33, -.05]	.41** [.28, .52]			
7. PSLE	2.74	3.43	-.17* [-.31, -.02]	-.02 [-.17, .13]	.01 [-.13, .16]	-.37** [-.50, -.24]	.47** [.35, .58]	.31** [.17, .44]		
8. ICErace	0.42	0.44	.24** [.09, .37]	-.16* [-.30, -.01]	.05 [-.10, .20]	.27** [.12, .40]	-.24** [-.37, -.09]	-.23** [-.36, -.08]	-.13 [-.27, .02]	
9. ICEraceinc	0.05	0.06	.20** [.05, .34]	-.20** [-.34, -.06]	.07 [-.08, .22]	.30** [.16, .43]	-.27** [-.40, -.12]	-.23** [-.36, -.08]	-.15 [-.29, .00]	.81** [.75, .85]
10. Placental age	-1.25	1.12	-.06 [-.21, .09]	-.02 [-.17, .13]	-.10 [-.24, .05]	.00 [-.15, .15]	-.06 [-.21, .09]	.02 [-.13, .17]	-.09 [-.23, .06]	-.02 [-.17, .13]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 2.

Means, standard deviations, and correlations with confidence intervals for Black participants

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. Maternal age	25.68	5.33								
2. BMI	29.74	8.23	.24** [.11, .35]							
3. Total pregnancies	2.82	1.75	.45** [.34, .54]	.10 [-.03, .22]						
4. Adjusted income	14830.37	14108.30	.32** [.20, .43]	.01 [-.12, .13]	-.24** [-.36, -.12]					
5. ACES	5.04	5.02	.01 [-.12, .14]	.05 [-.07, .18]	.06 [-.07, .19]	-.05 [-.17, .08]				
6. TLEQ	7.25	5.18	.10 [-.02, .23]	-.02 [-.15, .11]	.17** [.05, .29]	-.01 [-.14, .11]	.37** [.26, .47]			
7. PSLE	3.47	3.76	-.04 [-.17, .08]	-.02 [-.14, .11]	.12 [-.01, .24]	-.09 [-.22, .03]	.46** [.35, .55]	.38** [.27, .49]		
8. ICErace	-0.51	0.50	.18** [.05, .30]	-.01 [-.14, .12]	-.04 [-.17, .08]	.27** [.15, .39]	-.02 [-.14, .11]	-.04 [-.17, .09]	-.00 [-.13, .12]	
9. ICEraceinc	-0.07	0.08	.16* [.03, .28]	-.05 [-.17, .08]	-.09 [-.22, .03]	.31** [.20, .42]	-.07 [-.20, .05]	-.09 [-.21, .04]	-.13* [-.26, -.01]	.76** [.70, .81]
10. Placental age	-0.95	1.19	.02 [-.11, .14]	-.06 [-.19, .07]	.01 [-.11, .14]	.02 [-.11, .15]	-.00 [-.13, .12]	-.01 [-.14, .11]	.08 [-.04, .21]	.07 [-.06, .19]

Note. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 3.

Identify as Black: Covariate only Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.91**	[-1.14, -0.67]			
Maternal age	0.01	[-0.19, 0.22]	.00	[-.00, .00]	
Male fetal sex	-0.04	[-0.34, 0.27]	.00	[-.00, .00]	
Income	0.03	[-0.20, 0.25]	.00	[-.00, .00]	
Total pregnancies	0.02	[-0.16, 0.20]	.00	[-.00, .00]	
Pre-pregnancy BMI	-0.07	[-0.21, 0.07]	.00	[-.01, .02]	
Syncytiotrophoblast	0.15	[-0.04, 0.34]	.01	[-.01, .03]	
					R^2 = .015 95% CI[.00,.03]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 4.

Identify as Black: Covariates + Stressors Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.90**	[-1.14, -0.67]			
Maternal age	0.03	[-0.17, 0.24]	.00	[-.00, .01]	
Male fetal sex	-0.03	[-0.34, 0.27]	.00	[-.00, .00]	
Income	0.03	[-0.20, 0.26]	.00	[-.00, .00]	
Total pregnancies	0.00	[-0.18, 0.19]	.00	[-.00, .00]	
Pre-pregnancy BMI	-0.07	[-0.21, 0.07]	.00	[-.01, .02]	
Syncytiotrophoblast	0.14	[-0.06, 0.33]	.01	[-.01, .03]	
PSLE	0.13	[-0.04, 0.31]	.01	[-.01, .03]	
ACES	-0.04	[-0.22, 0.15]	.00	[-.01, .01]	
SLE	-0.04	[-0.20, 0.11]	.00	[-.01, .01]	
					R^2 = .025
					95% CI[.00,.03]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 5.

Identify as Black: Covariates + Stressors + Segregation Regression Resulta

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.84**	[-1.11, -0.57]			
Maternal age	0.02	[-0.19, 0.23]	.00	[-.00, .00]	
Male fetal sex	-0.03	[-0.33, 0.28]	.00	[-.00, .00]	
Income	0.01	[-0.23, 0.24]	.00	[-.00, .00]	
Total pregnancies	0.01	[-0.18, 0.19]	.00	[-.00, .00]	
Pre-pregnancy BMI	-0.07	[-0.21, 0.07]	.00	[-.01, .02]	
Syncytiotrophoblast	0.15	[-0.05, 0.34]	.01	[-.01, .03]	
PSLE	0.13	[-0.05, 0.30]	.01	[-.01, .03]	
ACES	-0.04	[-0.22, 0.15]	.00	[-.01, .01]	
SLE	-0.04	[-0.20, 0.12]	.00	[-.01, .01]	
ICErace	0.16	[-0.16, 0.48]	.00	[-.01, .02]	
					R^2 = .029
					95% CI[.00,.03]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 6.
Identify as Black: Covariates + Stressors + Racialized Income Inequality Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-0.88**	[-1.15, -0.62]			
Maternal age	0.03	[-0.18, 0.24]	.00	[-.00, .00]	
Male fetal sex	-0.03	[-0.34, 0.27]	.00	[-.00, .00]	
Income	0.02	[-0.21, 0.25]	.00	[-.00, .00]	
Total pregnancies	0.01	[-0.18, 0.19]	.00	[-.00, .00]	
Pre-pregnancy BMI	-0.07	[-0.21, 0.07]	.00	[-.01, .02]	
Syncytiotrophoblast	0.14	[-0.06, 0.34]	.01	[-.01, .03]	
PSLE	0.13	[-0.04, 0.31]	.01	[-.01, .03]	
ACES	-0.04	[-0.22, 0.15]	.00	[-.01, .01]	
SLE	-0.04	[-0.20, 0.12]	.00	[-.01, .01]	
ICEraceinc	0.35	[-1.72, 2.43]	.00	[-.00, .01]	
					R^2 = .025
					95% CI[.00,.03]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 7.

Identify as white: Covariates only Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-1.24**	[-1.51, -0.97]			
Maternal age	-0.02	[-0.25, 0.22]	.00	[-.00, .00]	
Male fetal sex	-0.06	[-0.40, 0.28]	.00	[-.01, .01]	
Income	-0.01	[-0.23, 0.20]	.00	[-.00, .00]	
Total pregnancies	-0.13	[-0.36, 0.10]	.01	[-.02, .03]	
Pre-pregnancy BMI	-0.04	[-0.25, 0.17]	.00	[-.01, .01]	
Syncytiotrophoblast	0.14	[-0.10, 0.38]	.01	[-.02, .03]	
					R^2 = .019
					95% CI[.00,.04]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 8.

Identify as white: Covariates + Stressors Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-1.22**	[-1.49, -0.95]			
Maternal age	-0.03	[-0.26, 0.21]	.00	[-.00, .00]	
Male fetal sex	-0.08	[-0.42, 0.26]	.00	[-.01, .01]	
Income	-0.06	[-0.29, 0.17]	.00	[-.01, .01]	
Total pregnancies	-0.15	[-0.39, 0.08]	.01	[-.02, .04]	
Pre-pregnancy BMI	-0.05	[-0.26, 0.16]	.00	[-.01, .01]	
Syncytiotrophoblast	0.13	[-0.12, 0.37]	.01	[-.02, .03]	
PSLE	-0.14	[-0.36, 0.08]	.01	[-.02, .04]	
ACES	-0.06	[-0.27, 0.15]	.00	[-.01, .01]	
SLE	0.09	[-0.11, 0.30]	.00	[-.02, .03]	
					R^2 = .036
					95% CI[.00,.05]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 9.

Identify as white: Covariates + Stressors + Segregation Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-1.21**	[-1.52, -0.91]			
Maternal age	-0.02	[-0.26, 0.22]	.00	[-.00, .00]	
Male fetal sex	-0.08	[-0.42, 0.27]	.00	[-.01, .01]	
Income	-0.06	[-0.29, 0.17]	.00	[-.01, .01]	
Total pregnancies	-0.15	[-0.39, 0.09]	.01	[-.02, .04]	
Pre-pregnancy BMI	-0.05	[-0.26, 0.17]	.00	[-.01, .01]	
Syncytiotrophoblast	0.13	[-0.12, 0.37]	.01	[-.02, .03]	
PSLE	-0.14	[-0.36, 0.08]	.01	[-.02, .04]	
ACES	-0.06	[-0.27, 0.15]	.00	[-.01, .01]	
SLE	0.09	[-0.11, 0.30]	.00	[-.02, .02]	
ICErace	-0.01	[-0.44, 0.41]	.00	[-.00, .00]	
					R^2 = .036
					95% CI[.00,.04]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.* indicates $p < .05$. ** indicates $p < .01$

Supplemental Table 10.
Identify as white: Covariates + Stressors + Racialized Income Inequality Regression Results

Predictor	b	b 95% CI [LL, UL]	sr^2	sr^2 95% CI [LL, UL]	Fit
(Intercept)	-1.27**	[-1.57, -0.98]			
Maternal age	-0.03	[-0.27, 0.21]	.00	[-.00, .01]	
Male fetal sex	-0.07	[-0.41, 0.27]	.00	[-.01, .01]	
Income	-0.08	[-0.32, 0.15]	.00	[-.01, .02]	
Total pregnancies	-0.17	[-0.41, 0.07]	.01	[-.02, .04]	
Pre-pregnancy BMI	-0.03	[-0.24, 0.19]	.00	[-.01, .01]	
Syncytiotrophoblast	0.12	[-0.12, 0.37]	.01	[-.02, .03]	
PSLE	-0.15	[-0.37, 0.07]	.01	[-.02, .04]	
ACES	-0.04	[-0.25, 0.17]	.00	[-.01, .01]	
SLE	0.11	[-0.09, 0.32]	.01	[-.02, .03]	
ICEraceinc	1.40	[-1.68, 4.48]	.00	[-.02, .02]	
					R^2 = .041
					95% CI[.00,.05]

Note. A significant b-weight indicates the semi-partial correlation is also significant. b represents unstandardized regression weights. sr^2 represents the semi-partial correlation squared. LL and UL indicate the lower and upper limits of a confidence interval, respectively.
* indicates $p < .05$. ** indicates $p < .01$.

Supplemental Table 11.

Model comparison results for Black participants

	df	AIC
Covariates only	8	781.3285
Covariates + Stressors	11	784.9743
Covariates + Stressors + Segregation	12	785.9858

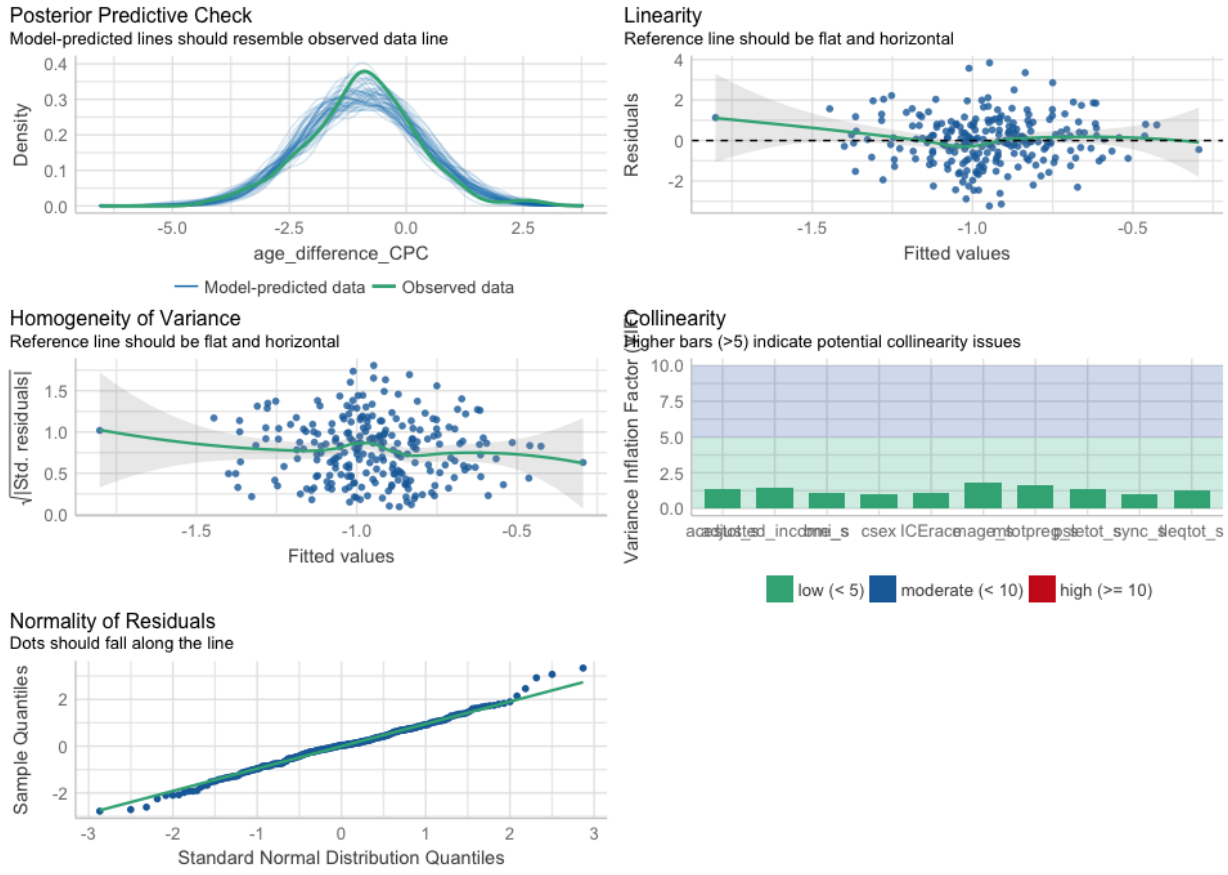
	df	AIC
Covariates only	8	781.3285
Covariates + Stressors	11	784.9743
Covariates + Stressors + Racialized income inequality	12	786.8558

Model comparison results for white participants

	df	AIC
Covariates only	8	548.9419
Covariates + Stressors	11	551.9242
Covariates + Stressors + Segregation	12	553.9196

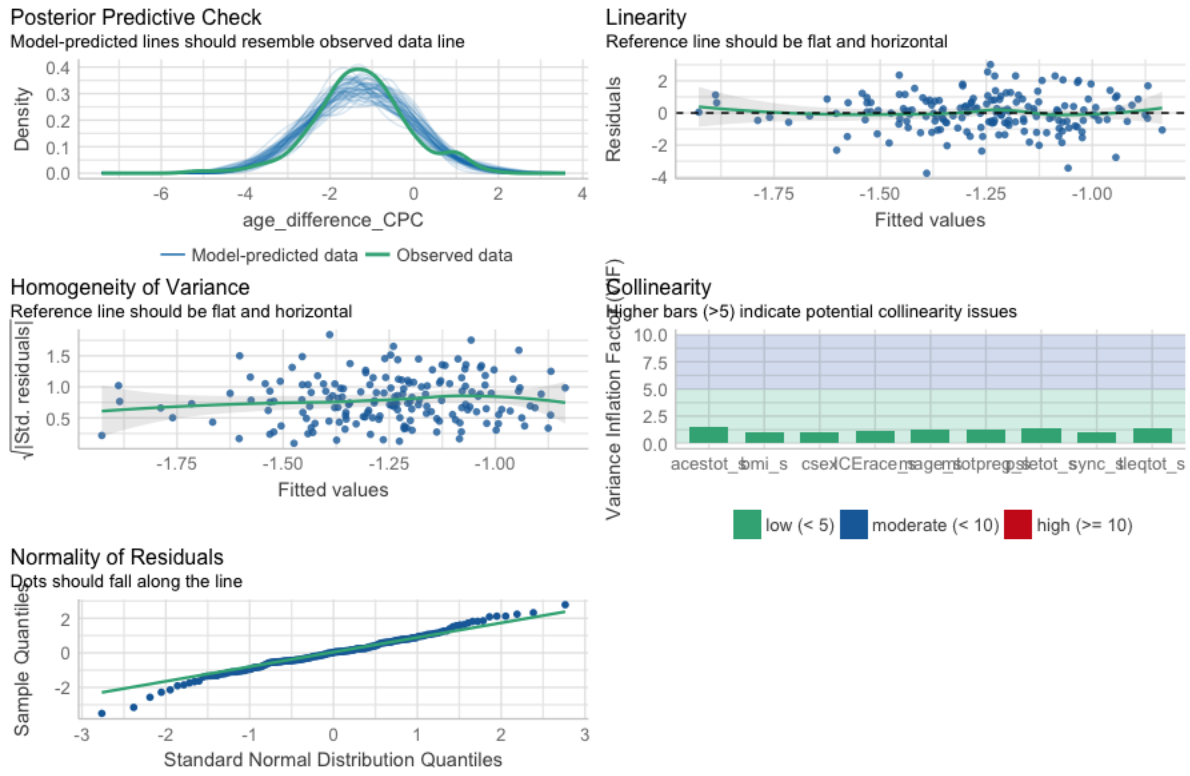
	df	AIC
Covariates only	8	548.9419
Covariates + Stressors	11	551.9242
Covariates + Stressors + Racialized income inequality	12	553.0692

Supplemental Figure 2. Model Fit Diagnostic Plots and VIF for Full Segregation model in Black participants



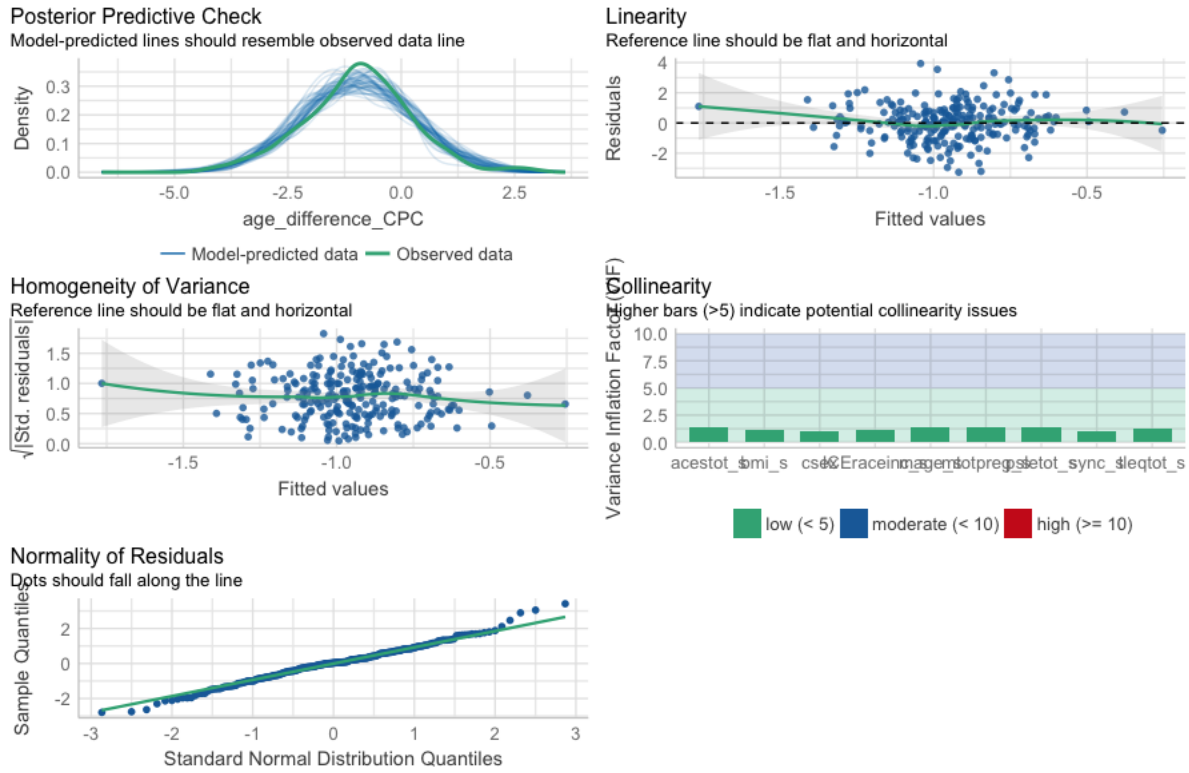
Variable	VIF
Maternal age	1.79766076
Male fetal sex	1.01389609
Income	1.47689975
Total preg	1.6312491
BMI	1.08104217
Syncytiotroph	1.03406799
PSLE	1.39978187
ACES	1.36318487
SLE	1.29352958
ICErace	1.10967114

Supplemental Figure 3: Model Fit Diagnostic Plots and VIF for Full Segregation model in white participants



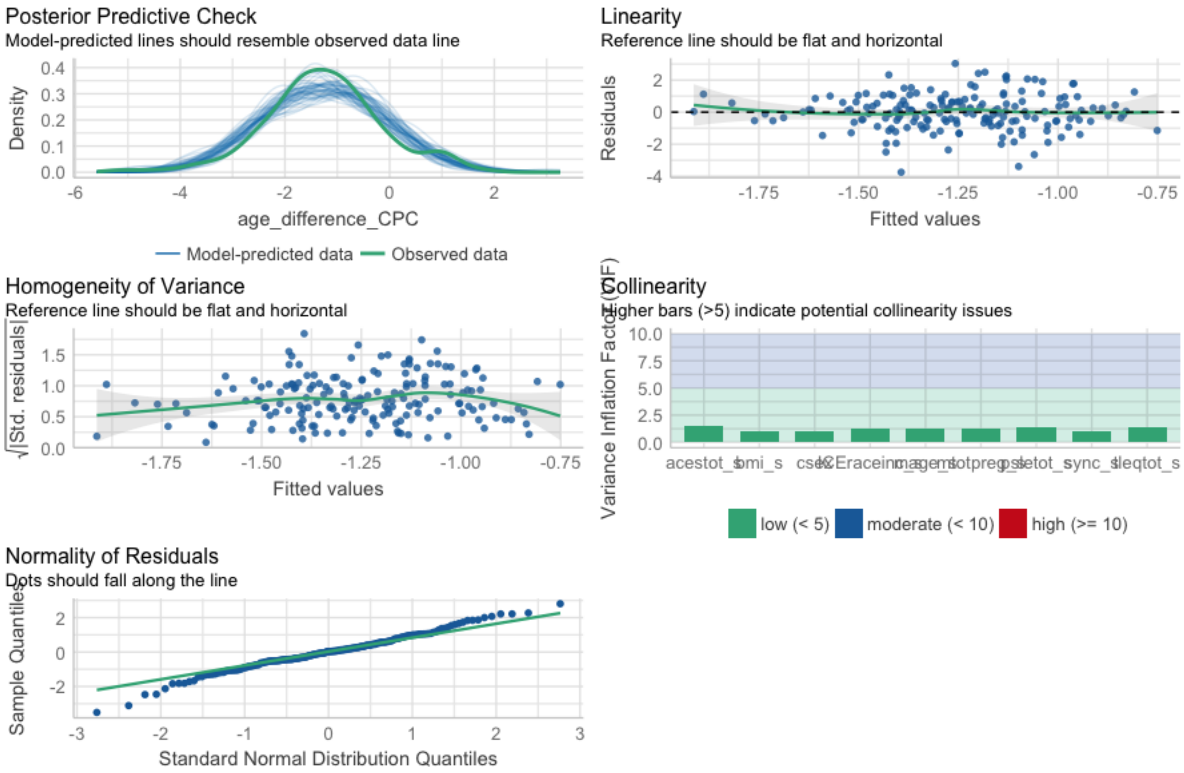
Variable	VIF
Maternal age	1.64540801
Male fetal sex	1.01655467
Income	1.67965836
Total preg	1.42690633
BMI	1.06638609
Syncytiotroph	1.04574274
PSLE	1.46393705
ACES	1.50471241
SLE	1.41499682
ICErace	1.20159275

Supplemental Figure 4: Model Fit Diagnostic Plots and VIF for Full Racialized Income Inequality model in Black participants



Variable	VIF
Maternal age	1.79310042
Male fetal sex	1.01213727
Income	1.49643438
Total preg	1.63309275
BMI	1.08539949
Syncytiotroph	1.04075071
PSLE	1.40115266
ACES	1.36317741
SLE	1.29362148
ICEraceinc	1.15672339

Supplemental Figure 5: Model Fit Diagnostic Plots and VIF for Full Racialized Income Inequality model in white participants



Variable	VIF
Maternal age	1.62304162
Male fetal sex	1.01916781
Income	1.72688626
Total preg	1.45185747
BMI	1.08098233
Syncytiotroph	1.04685558
PSLE	1.46642513
ACES	1.52196112
SLE	1.41263209
ICEraceinc	1.26109538

Conclusion

This dissertation explores intergenerational trauma, epigenetics, and embodiment from a critical biocultural lens. Each of those three terms—intergenerational trauma, epigenetics, embodiment—are terms with profoundly different meanings between and within academic disciplines and anthropology. Throughout this dissertation, I have both used them in traditional biosocial terms while also interrogating the assumptions they contain. While intergenerational trauma can be understood diversely (e.g., spiritual wounding or collective/cultural trauma and loss), for biosocial scientists it typically refers to psychological trauma symptoms and/or traumatic stress exposure in one generation that result in deleterious changes to physical or mental health subsequent generations (Cerdeña et al 2021). While embodiment can refer to how bodies are politically constructed and understood, biosocial scientists often engage its material dimensions. While such approaches may re-stigmatize or naturalize a model of intergenerational biological deficiency, I rationalized my work by using a narrative choreography of embodiment as social forensics. Through the process of building a progressive narrative for re-centering the social in social epigenetics, I have come to understand that this means reflecting on the cultural models and subjectivities of biosocial science itself. While reflexivity and attention to the metaphoric aspects of language are typically the praxis of the sociocultural wing of our biocultural department, I have found it to be a relevant and productive practice in my own science.

Indeed, even the term 'epigenetics' is hotly contested—'purists might demand that it refer only to covalent molecular modifications of DNA while others expand it to mean chromatin structure, developmental processes, or even the environment itself (Greally 2018). Many biosocial studies of intergenerational trauma and embodiment seek to 'control' for subsequent child trauma exposure; like Dias and Ressler's intergenerationally fear-conditioned rat pups, there is an aspect to

biosocial understandings of intergenerational trauma that want to understand impairments to offspring health independent of re-exposure to adversity (Dias & Ressler 2014). In this sense, intergenerational transmission carries metaphoric aspects of infection or contagion. The infectious metaphoric aspect is echoed in figures of stress hormones, like viruses, crossing the placenta. The idea that trauma is transmitted across generations through invisible epigenetic mechanisms also carries an aspect of mystery, investigation, and forensic recovery.[24] Thus, that trauma is transmitted regardless of current conditions or experiences seems to be woven into the scripts of the biosocial science of intergenerational transmission. Recentering the 'social', this dissertation unsettles how the field avoids the fact that the risk of present trauma exposure is not independent of past exposure. As I point out across each chapter in this dissertation, people inherit genes, non-coding RNAs, maternal soma—but also caregiving behaviors, poverty, state and gender violence, and neocolonial economies structured on the systematic disenfranchisement of racialized peoples and the Global South. While the science of epigenetic transmission remains unclear and contested, the intersectional inheritance of structural violence is apparent across each of the chapters I present here.

Similarly, while biosocial notions of resilience tend to locate it as a feature of individuals 'resistant' to the 'toxic stress' of their environments (again drawing on infectious or toxicology related metaphors), the papers in this dissertation suggest rethinking resilience as a social process. In Chapter 2, I explore how anthropological theories of subjectivity help shift our understanding of trauma and shared notions of the self across generations. Rather than accumulating trauma across generations, intergenerational Chaculense narratives revealed that while the present and past are linked by gender and state violence (and impunity), grandmothers and mothers harness internal and external resources to 'retether' themselves to their sense of self and their social fabric (Lester 2013).

[24] There is also some aspect of recovered/lost/unconscious memories of childhood in this discourse, or finding the cause to a mysterious suffering experienced despite living a 'good' life.

Throughout their narratives, mothers and grandmothers described negotiating for better familial and romantic relationships and manifesting an ethic of mutual understanding in children. In this way, they navigate—and transform—their internal models of loving care, agency, and hope for the future.

Chapter 3 also unsettles certain ideas we have about the universality of risk and resilience processes, locating resilience not just in everyday social relations and subjectivities, but macro-social institutional privilege indexed by race. Although the epigenetic outcome in this paper gave null results, an unexpected result was the differential risk of trauma in early life, adulthood, and the prenatal period across white and Black participants. In white participants, resilience 'looked' the way one might expect; income, the density of white households, and the density of white wealthy households were associated with lower levels of trauma across the life course. In Black women, none of the economic or residential segregation measures were associated with any of the trauma indexes. Black women with greater incomes or who lived in more white neighborhoods did not report experiencing less trauma. As Williams (2020) and others suggest, this finding reveals the ways in which institutions and social structures preferentially benefit white people in the United States, reframing 'resilience' as another aspect of social privilege. Critically, the epigenetic outcome was not related to any risk factors and demonstrates the current limitations of epigenetic placental age acceleration as a potential pediatric screening tool and/or therapeutic target for reducing the outsize burden of perinatal mortality born by Black mothers and children in the U.S.

The story of the story

In the introduction, I outlined my original research plans and goals, and how they changed after the disruption of the COVID-19 pandemic. I had originally planned to do a mixed-method study of intergenerational trauma, cultural models of resilience, and child socioemotional

development, HPA function, and DNA methylation of HPA-relevant genetic pathways. This work was solidly within the biocultural framework I had been trained in, and I negotiated its potential to stigmatize and biologically essentialize trauma by employing a narrative of 'embodiment as social forensics'. However, the process of conducting research—the praxis, to use another word from across the hall—drew me away from my original goals. As I describe in the chapter interludes in this dissertation, I found myself disturbed by the power inequalities, forms of erasure, and failed communication of my goals lived out in the everyday practice of my research. The necessary reductionism of social complexity in the face of academic expediency, challenges of representation and interpretation of othered experiences, and the reception of my work by lay and scientific audiences made me feel that the ethical ground I was standing on was shifting and unstable.

After leaving the field in March 2020, I did not act for several months, using the time to reflect and work on other projects and teaching. I developed a self-collection protocol and attempted to conduct interviews over Zoom, but cell service and language barriers made them feel intrusive, unpleasant, and uninformative. Many of my interlocutors told me that they had found doing the in-person interviews meaningful and therapeutic; they were a space for desahogo. I also found them deeply meaningful, but experienced intense distress at the need to continually keep pushing, asking, moving the conversation along, and generally subjecting an interpersonal experience to the constraints of a biosocial study. I found it difficult to put the stories behind me and eventually sought psychological assistance for vicarious trauma. I knew that without more stories, the hormone and epigenetic assays would be useless. Between my own feelings that I may be committing harm by interviewing people about traumas I could not help them psychologically or practically resolve and my growing doubt that DNA methylation in such a small sample could tell us anything physiologically meaningful, by December of 2021, I decided I needed to find a different way of completing the dissertation.

I chose to write up the ethnographic data that I had collected and add the systematic review and structural racism and placental epigenomics papers at this time. It seemed to me to be a valuable way to take a step back and use the dissertation as an opportunity to critically examine the field of social epigenetics and intergenerational transmission. This also involved negotiating power gradients, but this time within my own community of biosocial scientists. Ultimately engaging the field of social epigenetics as my area of inquiry—rather than just postwar Guatemala—felt more ethically tenable and less extractive to me. This is why the dissertation became less about intergenerational trauma and more about epigenetics.

That choice brings us to the last way one might integrate the social into the social epigenetics of intergenerational trauma—by exploring its social functions and implications. I grant my contributions here have been partial and navel-gazing, not as expansive as other scholars who have explored how epigenetic intergenerational trauma discourse has been up taken by trauma-affected communities. While I lack space for a full review of this work, what I wish most to do is draw attention to the fact that epigenetic discourse has a vibrant social life outside of the confines of cohort studies and psychobiology labs. Muller and Kenney (2020) for example, explore how neuroscience-based trauma training programs in primary schools teach instructors that young children with epigenetically embedded trauma are more likely to 'blow their top'[25]—referring literally to reduced cortical inhibition of the limbic system. While some of the schools they surveyed (especially those that served Black children) found the approach stigmatizing and rejected research participation, others found biological de-stigmatization (or deblaming, as alluded to in the introduction) to be a meaningful rhetoric that promoted restorative justice.

[25] In the trainings, this phrase is evocatively accompanied with a hand gesture, a fist that opens into an open palm, an embodied sign of the cortex 'blowing up' to give way to fear-based responses.

With its attention to the social life of epigenetics as practiced, this dissertation also functions as contributes to the anthropology of scientific reflexivity. It tells a progressive story about the value of biosocial epigenetic approaches to understanding the impact of intergenerational trauma which focus on the body. In chapter one, I diagnose the problem, noting methodological and conceptual challenges in epigenetic causal inference and poor consideration of how ecological inheritance of adversity shapes children's experiences and their embodiments in somatic function and health. In chapter two, I explore the embeddedness of trauma in social conditions by exploring the subjective experiences of grandmothers, mothers, and children growing up in a community affected by war trauma in the grandmaternal generation and by systemic inequities thereafter. Rather than locating trauma and resilience in bodies or individuals, I suggest research should attend to the fundamental causes of political-economic of trauma that shape intersectional experiences of violence. In my last chapter, I try to solve several of the problems I articulate in the first two chapters. Responding to concerns about the rigor of candidate-gene or epigenome-wide approaches in surrogate tissues, I chose an epigenetic phenotype trained on clinically meaningful data in a target tissue— accelerated epigenetic aging of placenta. Instead of locating risk only in individuals, I used intersectional methods to nest participant experiences of life-course stress in a measure of structural racism.

While this chapter structure implies a progressive, forward-moving science of epigenetic embodiment capable of doing 'social forensics', its critique unfolds in an echoing counterpoint in my chapter interludes and is woven throughout the discussion sections of each chapter. These interludes draw attention to the everyday acts of erasure, reduction, and misrepresentation that I experienced as I practiced my work. In a way it is a story about a growing awareness of epistemic violence. Epistemic violence refers to oppression, harm, domination, and subjugation done through the creation of legitimated forms of knowledge and the obliteration of other ways of knowing and creating knowledge. The term 'violence' in this context may feel hyperbolic to non-social scientists;

what could be violent about understanding gene regulation? But anthropologists and sociologists have long theorized power relations as articulated across a continuum of violence that includes microaggressions in everyday speech acts, neoliberal political economy that locates ill-health in the behaviors of the poor, to violence's most gross endpoints of war and genocide (Holmes 2013).

Many trace the term epistemic violence from Gayatri Spivak's reading of the implications of the Foucauldian episteme for the legibility of subaltern subjectivity (Spivak, 2015). In later turns Karen Dotson (2011) and Patricia Hill Collins (2017) have elaborated on the ways epistemic violence serves to maintain orderly structures of "progressive" knowledge production by silencing, squeezing out, and punishing academics who fail to conform to academia's antipolitics machine. The epistemic violence of my work was mirrored in ways it was interpreted and mirrored back to me, the subjugation of experiences of violence and injustice to the frantic productivity demands of academia, and the exclusion of bodies and narratives that could not conform to the limits of my biosocial models and instrumentation. I recognize that this perspective may feel provocative, and I do not mean to judge or claim moral righteousness over those who continue biosocial science of disenfranchised communities. The process I describe in this dissertation is my own, and while I hope it is of interest to those I work with, I understand that our logics and pathways to the work are diverse and valid.

Indeed, rather than a morally upright, progressive narrative with a satisfying conclusion, the narrative structure of this dissertation is also one of failure. In Chapter 1, I reveal that there is very little evidence that preconception trauma leaves assayable epigenetic signatures, and that the biological and conceptual leaps we'd have to make for it to be so should fill us with skepticism. In Chapter 2, I reflect on the ethical challenges of theorizing intergenerational trauma across the power inequality gradient between myself and the community. In Chapter 3, the epigenetic aging algorithm that I had hope would redress methodological critiques of epigenetic embodiment as a means of

making claims for the reparation of structural racism resulted in "null" findings. Such algorithmic approaches come loaded with their own sets of problematic assumptions of biological normativity, especially salient with the growth of machine learning methods and computational approaches in epigenetics. While powerful, algorithms reflect the epigenetic characteristics of training sets that may unintentionally reinforce difference from privileged biologies as pathological.

In my chapter interludes, I trace the developmental milestones that shape my growing moral injury, my growing awareness that the story I had told myself about material embodiment as forensics of harm was indeed, as Tuck says, a flawed theory of social change. Although there are many researchers who study the practices and logics that epigenetic scientists express, most of this work analyzes those expressions as discourse in scholarship that practicing biosocial scientists may be unlikely to read. Along with its contributions to theories of intergenerational trauma and epigenetic embodiment, I hope that this dissertation may also contribute to interdisciplinary, engaged biosocial science. Rather than study-up, the embedded critique and reflexive auto-ethnography within the dissertation functions as studying-in (Nader 1972).

I trained at Emory University's department of anthropology, one of the last remaining biocultural training programs in the United States. In an early seminar where sociocultural and biological anthropology students struggled to communicate across theoretical divides, we asked a professor what kind of biocultural training we would receive; he responded that the faculty hoped that by us being together, we would find it ourselves. Although the work presented here is not truly a 'synthesis' of the methods and ways of knowing I learned in sociocultural and biological courses, I also hope it serves as record of the value of cross-disciplinary training in shaping the practice of science.

Through a mirror

Before I started my year of fieldwork, a student in my program sent me a paper to read. Published in Development and Psychopathology, it was a widely cited paper exploring the combination of genetic and environmental factors that predicted risk of child maltreatment (Jaffee et al., 2005). I had to read the abstract a few times to really understand that the paper was about the genetic risk of being maltreated. While some part of me could comprehend that there were behavior genetics researchers out there studying the genetic contributions to aggression, including the maltreatment of children, I wasn't wholly prepared to consider research that seriously engages with the idea that some children are inherently more likely to be abused because of intrinsic genetic factors. This study and several others used widely employed twin and family designs to partial out genetic vs. environmental variation, but a new set of genetic cohort studies now use the same logics (that certain children have temperaments and other intrinsic qualities that make people, on average, more likely to maltreat them) with large scale genomic datasets (Dalvie et al., 2020; Pezzoli & Saudino, 2021). Maybe this is something you could look at in your study, my friend said.

Research paradigms that suggest that some people are more likely to experience trauma because their genetic makeup makes them more likely to attract violence evince critical blind spots in the biosocial research on intergenerational trauma. But while I am quick to acknowledge the epistemic violence of this work, I have come to recognize that it is also a mirror to my own. As described in my introduction original plan for this study was to assay the DNA methylation of children to see if grandmaternal and maternal trauma exposure contributed above and beyond children's own stressful experiences to differences in the genetic regulation of the stress response. In the broader impacts section of my funded NSF-Biological Anthropology Doctoral Dissertation Research Grant, I allude to many of the same arguments as the researchers examining genetic vulnerability to child maltreatment. I write that the research has the potential for "significant public health impacts", saying I will help discover "culturally salient sources of psychobiological resilience"

and reveal the mechanisms that confer "vulnerability and resilience to intergenerational trauma." What did I mean by this exactly?

Primarily, I meant to show that I too could enact the "narrative choreography" of the people whose lifeworlds I wanted to emulate. I wanted to feel I was doing rigorous, cutting-edge work for the good of Latinx and other communities of color, because I had worked with and been inspired by other scholars who used this rhetoric. My own positionality as a Latina who had experienced intergenerational violence also motivated me in ways I ultimately have had to work very hard to disentangle from my research aims. Along with these personal dynamics, I found that much of what we do as academics is constrained by our own political economy, precarity, and need to justify our continued existence. An important aspect of the social life of epigenetics is the labor environment in which biosocial scientists find themselves. We are compelled analyze all available data (a scarce resource), produce more publications, and suggest that more research is needed. This cycle of productivity and speed oftentimes means that the people writing papers very rarely spend extensive time with the people they study. We do not have the time to contemplate what it will be like to sit and explain to the survivors of violence that some of them were always going to be easier to hit (Gibbon & Lamoreaux 2021). In Chapter 1 of this dissertation, I discuss the need to address genetic confounding in epigenetic studies as a great deal of epigenetic variation is under tight genetic control. That thought still animates a moral reaction in me: I have been enculturated to think it would be wrong not to address a potential confound and make an incorrect causal claim. That moral still exists within me, but the process of my dissertation research has transformed what I think of as an incorrect claim. I think about the triad in which all three members— grandmother, daughter, and 9-year-old granddaughter— have all been raped. How much variance with that familial association pull in my model? How would I explain it to them?

These kinds of thoughts have led me to the belief that not-doing a study can also be an act of justice. During early 2021, I thought I might be able to ethically continue my work by re-homing the project within Guatemalan genomic science. This seemed like a way at least to address the extractives and unequal benefit that had troubled me. My committee member Rachel Hall-Clifford was good enough to connect me to Fredy Peccerrelli, the head of the Guatemalan Forensic Anthropology Foundation (FAFG). In the long tradition of forensic anthropology that documented and helped prosecute the atrocities of the genocide, FAFG uses genomic methods to identify the remains of loved ones and reconnect them with their families for proper burial and closure. If anyone might find my story about embodiment and intergenerational trauma compelling, I thought it would be him. Dr. Peccerelli listened kindly. After my presentations he paused, and then looking at me through the Zoom window asked, where are you from anyway? We talked about what it meant for Puerto Rican anthropologist to study the Guatemalan genocide and discussed many of the practical and epistemic concerns involved in a potential collaboration. How would we explain null results? How much time would it take to interview and compensate everyone? Who would pay for that? In the end, the samples would need to go to a high-throughput lab somewhere in the United States or Canada anyway, because FAFG did not have the laboratory equipment needed to do the DNA methylation microarray. Would that still be Guatemalan genomics? The more we talked, the less it seemed so.

It's an important story to tell, but it isn't your story, he said, as we closed the call.

I agree with Dr. Peccerelli, although I know not all my colleagues may. I don't think that that story can't be told, I just think I am not the one to tell it. My story, the one I have epistemic ownership of, is the one I am telling my community of biosocial researchers. I still claim them as my community, and they still claim me. Many people have asked me why I choose to stay in biological anthropology. Several of my other friends who underwent similar changes in self-concept and moral

orientation became cultural anthropologists and science and technology studies scholars. Others left academia entirely and committed themselves to applied work. In the next section, I explore what kinds of transformative approaches I see in biocultural anthropology and biosocial science, and lay out a research trajectory as a critical biosocial anthropologist moving forward.

Critical Biosocial Futures

I want to close this dissertation with an expression of hope, not just for what might be, but for what is already happening in the field of critical biosocial studies.

Decolonial and intersectional methods have been rooted historically in qualitative research methods. These methods center the expertise of lived experience, rather than objective distance, and ontological stances that permit multiple, overlapping truths to be 'braided' into knowledge made for and by the communities concerned (Kimmerer, 2013; Smith, 2021). While qualitative methods' emphasis on narrative, subjectivity, and self-determination are resonant with the call to decolonize knowledge production, I find myself excited to be a part of a community of scholars committed to critical biosocial and decolonial quantitative methods. Rather than claiming the right to 'truth', critical biosocial methods explore how the same data might mean different things in different contexts and explore how power and science work together to produce or occlude different forms of knowing. Using quantitative methods that are reflexive and accountable, critical biosocial methods also "braid knowledge" by demonstrating the ways in which bionormativity is constructed—and how it can be reconstructed more equitably.

Decolonial theorists identify and disrupt the ways in which scholarship from the Global North claims ontological and epistemic supremacy; "The First World has knowledge, the Third World has culture; Native Americans have wisdom, Anglo Americans have science." (Mignolo 2009, p.160). Decolonial approaches and the ontological turn within anthropology share resonances, but

whereas ontological anthropology has emphasized authorial reflexivity and positionality, decolonial thought stresses that without returning epistemic power and resources to colonized peoples, such reflexive projects may only perpetuate extant knowledge hierarchies (Fúnez-Flores 2022). Similarly, decolonial approaches within biosocial genomics and biological anthropology have emphasized returning data sovereignty to Indigenous communities (Rodriguez-Lonebear 2016). Much like calls to end performative land acknowledgements given the lack of any real intention to return land to Indigenous communities, genomic data sovereignty challenges Western assumptions about the 'universal benefit' of genetic research and demands return of control over genetic data and profits derived therefrom to Indigenous communities (Lambert et al 2021).

In 2020, Native Hawaiian geneticist Keolu Fox outlined the stakes of genomic commodification and Indigenous data sovereignty in a piece in the New England Journal of Medicine, aimed at a general audience (Fox 2020). In it, Fox outlines how datasets that link genotypes to phenotypes have become valued in the hundreds of millions of dollars to pharmaceutical companies; given the unique genetic diversity of Indigenous populations, he argues Indigenous genotype-phenotype datasets produced by researchers may result in the same kind of commodification as occurred with the 1000 Genomes Project, International Hap-Map Project, and Human Genome Diversity Project. While the explicit aims of these projects (and their attendant open-access data sharing policies) were meant to benefit 'humankind', he notes that they have ultimately gone on to concentrate wealth in private ancestry testing companies and pharmaceutical companies, as well as benefit the careers of researchers in the Global North. Without attention to how Indigenous communities may guide the use of their genetic data, the recent NIH 'All of Us' initiative (which aims to increase the representation of Indigenous populations in federally funded genomic research), he argues, is likely to repeat the same pattern.

Fox describes two proposed approaches to returning control over and vesting the benefits of research participation in Indigenous communities: individual share-holder models, in which individuals receive fractional stock ownership in companies that use their data, and collective ownership models in which Indigenous community trusts return profits to communities that share genetic data in the form of subsidized treatments, drugs, and other benefits. While Fox emphasizes Indigenous sovereignty over knowledge vis a vis the profits created from Indigenous genomics, other approaches emphasize Indigenous rights to review research procedures and deny research access through the creation of local and/or tribal Institutional Review Boards (IRB). Academic IRBs have been critiqued as serving primarily to protect the legal liability of the institutions they serve, and for failing to consider how research might harm communities in addition to risks of harm to individuals (Friesen et al. 2017). Tribal and Indigenous community IRBs frequently draw harm to the community and to cultural values into decision making. Denying research access may bring the different ethical frameworks employed by researchers and Indigenous communities into contrast. For example, in 2006, tribal IRBs in Oklahoma rejected a genomics modification to an NIH funded diabetes trial due to the risk to cultural values and limited benefits. Reasons for rejecting the modification included broad consent for genomic research, indefinite storage of biological samples, and open access data-sharing— features that many researchers might argue increase how much we can learn from the data and compel us to share it with other scientists to increase its impact.

The values that guide these beliefs— that all knowledge is valuable, that science is a collaborative, progressive effort that benefits all of society— are held in contrast with those of tribal IRBs. The vaguely defined broad consent might include genetic testing for mental illness or tribal affiliation. Individuals from relatively small communities with rare genetic variants might be identifiable in public datasets, and tribal leaders were concerned that biological samples would not be stored or disposed of with the care their belief systems required.

Given their close work with Indigenous communities in the United States and abroad, biological anthropologists have been scrutinized (and have scrutinized themselves) for perpetuating extractive relationships. Some have responded by participating actively in supporting Indigenous genomic and data sovereignty, diversely imagined. An example of this is the Summer Internship for Indigenous Peoples in Genomics (SING) program, a global consortium that trains Indigenous students in New Zealand, Australia, Canada, and the United States in genomic science (Bardill et al. 2018). Although increasing the representation of Indigenous scientists does not de facto change scientific practice, such training programs hope that increasing the number of trained Indigenous geneticists may center Indigenous ethics in contemporary and paleogenetic research: balancing harms to community, caring for ancestral remains and biological samples, and considering the implications of their work for Indigenous political sovereignty. Similar calls for increased participation in knowledge production processes and re-thinking of the collective harms of settler colonial research have been made by anthropologists Krystal Tsosie, David Lawson, Alyssa Crittenden and many others (Tsosie et al. 2019; Urassa et al. 2021; Mangola et al. 2022).

Another powerful example of this approach I'd like to describe is the GenderSci Lab at Harvard directed by Sarah Richardson. GenderSci centers feminist politics of care and accountability in their everyday scientific practices as well as research foci. Richardson directs a multidisciplinary group of scholars in using feminist science to actively contest heteronormoative scripts in biosocial research. An example of this approach, GenderSci recently published a re-analysis of Levine and colleagues (Levine et al., 2017) influential meta-analysis on declining sperm counts. They trace how the original paper framed "global" declines as emergent threats to masculinity and virility and re-analyze the data under a new normative biovariability framework that finds global sperm counts have remained in a species-typical range over the past 20 years and that declines in Western populations may be attributable to demographic trends as opposed to fundamental threats to male

fertility (Boulicault et al., 2021). This approach is very similar to many I have seen in my career as a biocultural anthropologist, but its interdisciplinarity and active interrogation of white supremacy and antifeminist movements stands out as a new form of activist biosocial science.

The future work I hope to do is grounded in these principles, as well the other ones I retain about what constitutes good and bad science. In my planned postdoc with Zaneta Thayer, I begin that work by studying menstrual epigenomics using decolonial and feminist science principles like those at GenderSci Lab. For me, living those principles is about how I conduct my work and the ethic of care, humility, and equity I bring towards scientific practices. The first part of that involves identifying communities who wish to participate in research and with whom the power hierarchies between us are more equitable than those I enacted in my fieldwork in Guatemala. As many know, studying U.S. populations is less prestigious in biological anthropology. Our field emphasizes the value of studying non-WEIRD populations as a means of contesting bionormativity[26]. This project is important, but my experiences in the doctorate have pushed me to reconsider how and where I want to work. My postdoctoral project involves exploring the menstrual epigenomics of women like the ones we learned about in Chapter 3: middle-class Black and Brown women, nonbinary people, and trans people who despite achieving socioeconomic parity still experience the stress associated with status incongruity and structural inequality. This is a community I am part of and accountable to, and one that may access, contest, and make claims over the work. This is the kind of power hierarchy I would like to be in as a researcher going forward. From a methodological standpoint, current algorithmic methods can make the best of what we know DNA methylation microarrays are very good at: identifying cell-types[27]. Rather than pushing the biological reductivism of methylation

[26] I am being somewhat generous here. We also study the Other because of beliefs about the evolutionary hypotheses that can be tested in "non-industrial" societies.

[27] They are so good at this that we use DNA methylation to infer cell type proportions to remove potential confounding in social epigenetic studies, as discussed in Chapter 1

as a stable gene repressor and durable mark of developmental adversity, I want to reroot my use of the biomarker in the reliable physiological signals we already know epigenetics can give us.

I close with a final reflection about why staying in community with biosocial researchers is important to me going forward. While I often joke about the emptiness of a refrain that a better science is possible, I have no doubts that a worse science is possible. A worse science is probable. In this dissertation, I have said very little about COVID-19 pandemic that has claimed over six million lives globally. I have said nothing about the rising polarization and political violence in the United States. I have said nothing about the erosion of reproductive and LGBTQ rights or the immense challenges that face the U.S. electorate.

I am staying in the community because I still believe social relations— friendships, intimacy, care— are a means of manifesting different politics. The culture of science is no different, and I truly believe that the friendships and care I live out as a scientist suffuse through our lifeworld and transform it, just as they have transformed me. I don't know if I will become an academic, but I do know I will always remain in community with the biosocial scientists and researchers who have made up my family of choice for the last decade of my life. If a worse science comes, I want us to face it together.

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