Mapping Dissimilarity in Adolescence: Interpersonal Variability in Experience, Brain Structure, and Mental Health

by

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DISSERTATION ABSTRACT

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Doctor of Philosophy in Psychology

Title: Mapping Dissimilarity in Adolescence: Interpersonal Variability in Experience, Brain Structure, and Mental Health

Adversity is a well-established risk factor for poor health outcomes, in part due to its influence on physiological and neurodevelopmental pathways. While considerable research has focused on when, how, and to what extent adversity shapes development, fewer studies have investigated how positive experiences—alone and in combination with adversity—contribute to developmental trajectories. This dissertation argues for a developmental neuroscience approach that integrates both adverse and positive experiences to explain the heterogeneous outcomes observed across the lifespan. Adolescence is emphasized in this work as a critical period for brain development and the emergence of mental health challenges. Throughout, I used a novel methodological framework, inter-subject representational similarity analysis (IS-RSA), to examine these processes from an individual differences perspective.

Chapter II showed that dissimilarity in adversity was more strongly linked to externalizing than internalizing symptoms in adolescence. Positive experience dissimilarity had broader associations across both symptom domains, and they interacted with adversity to predict externalizing symptoms. Chapter III, developed through a registered report process, found that differences in threat and deprivation—and their interaction—were associated with differences in structural brain development from early- to mid-adolescence. While some hypothesized regions showed expected stronger associations with a particular adversity dimension, in general adversity-related brain differences were distributed more broadly than predicted. Chapter IV

extended these findings by showing that differences in cumulative positive experiences, when controlling for adversity, were also linked to differences in structural brain development. Results also underscored the importance of analytic decisions, such as how experiences are quantified and when they are measured. Chapter V integrates these findings and contextualizes them within broader theory and research on theoretical models of adversity, multivariate modeling of the brain, and developmentally informed intervention.

Together, these findings support a dimensional and distributed model of neurodevelopmental risk and resilience. They highlight the value of considering both adverse and positive experiences, and their interplay, to better understand individual differences in adolescent mental health and brain development.

This dissertation includes published co-authored material.

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CHAPTER 1: INTRODUCTION

You are the sum total of everything you've ever seen, heard, eaten, smelled, been told, forgot—it's all there. Everything influences each of us, and because of that I try to make sure that my experiences are positive.

—Maya Angelou, *O, The Oprah Magazine*

1. Beyond Adversity: Integrating Positive and Negative Childhood Experiences to Understand Brain Development

The architecture of the human brain is shaped through experience. Life events, whether nurturing or harmful, leave imprints on neural pathways and influence development in ways that can echo throughout one's lifespan. Although this concept is widely accepted today, the idea that humans can be so vulnerable to our environments can be a challenging one to accept, and the fields of psychology and psychiatry have slowly and circuitously recognized how external influences can shape the mind (Lasiuk & Hegadoren, 2006). With this growing awareness, research in the 21st century has made remarkable strides in characterizing the effects of adverse experiences across many domains of development, transforming clinical practice and guiding public policy (Murphey & Bartlett, 2019).

Many scientific disciplines, such as developmental psychology, prevention science, resilience science, and public health, have seen a growing interest in how positive experiences can also shape well-being (Han et al., 2023; Lich et al., 2013; Merrick & Narayan, 2020; Sege & Harper Browne, 2017). A critical next step for developmental neuroscience, I argue, is to examine the role of not only adverse, but also positive experiences in equal measure. This perspective review highlights current scientific knowledge on how both adverse and positive

experiences relate to mental and behavioral health, and it advocates for an integrated approach to studying brain development. By acknowledging the other half of the developmental equation, the field can move toward a more comprehensive framework for understanding how the complexity of experience shapes the body and mind.

1.1. Conceptualizing Adverse Experiences

This dissertation draws from the extensive body of research on toxic stress, adversity, and psychosocial risk, and uses the conceptualization of adverse childhood experiences (ACEs) as violations of the expectable environment that may (though not necessarily) trigger chronic or acute stress responses (Nelson & Gabard-Durnam, 2020a). Though not an exhaustive list, examples of adverse experiences include maltreatment, exposure to violence, extreme poverty, serious illness, discrimination, and parental discord. Most children and adolescents in the United States experience at least one form of adversity, and recent data suggest that exposure rates could escalate in younger birth cohorts (Hartwell et al., 2023). Adversities are also known to cluster together, and exposure to one increases the likelihood of exposure to further adversity (McLaughlin et al., 2012). Adverse exposures increase risk for a range of both internalizing and externalizing problems (Haahr-Pedersen et al., 2021), and are estimated to contribute to nearly half of psychiatric disorders in childhood and a third in adulthood (Green et al., 2010). As Zarse (Zarse et al., 2019) wrote, "Adverse childhood experiences and rearing may generate a public health burden that could rival or exceed all root causes."

Despite the clear associations between adversity and psychiatric risk, understanding how, why, and when these associations emerge remains a central challenge. The cumulative risk model, which highlights the dose-response nature of adversity by attending to the accumulation effect of adverse experiences on long-term outcomes (Evans et al., 2013a), is one conceptual

model that offers a valuable heuristic. Through this operationalization, researchers have identified that exposure to four or more adversities in childhood, typically measured out of ten, is associated with heightened risk for myriad health outcomes (Hughes et al., 2017). However, the model's simplicity comes at the cost of specificity (Lacey & Minnis, 2020a). Most individuals with a history of adversity do not experience any given negative outcome, and sum scores that represent the number of types of adversity experienced are poor predictors of an individual's later health problems (Baldwin et al., 2021). Retrospective self-reports by adolescents or adults dominate the literature on childhood adversity and cumulative risk, yet they often identify different individuals as highly exposed than prospective reports (Baldwin et al., 2019). To address this gap, researchers have begun examining prospective reports of ACEs (Baldwin et al., 2021; Naicker et al., 2021; Patten et al., 2014; Reuben et al., 2016), and the new tools have been developed to facilitate the assessment of adversity in early childhood, when self-report is not possible (e.g., PEARLS; (Thakur et al., 2020). Such efforts are critical for advancing understanding of cumulative risk across specific developmental periods.

Additional contextual factors that can influence health outcomes, such as intensity, type, timing, or interaction of adversities (Briggs et al., 2021; Nelson et al., 2020), however, are not captured in the cumulative risk framework. Dimensional models offer an alternative by distinguishing between different dimensions of adversity, such as *threat* to one's physical well-being, *deprivation* of species-expected cognitive, social, emotional, or material inputs, and *unpredictability* in one's environment. These dimensions are hypothesized to uniquely influence physiological development and consequently, behavior (Ellis et al., 2009a; McLaughlin, Sheridan, & Lambert, 2014). This approach recognizes both the cumulative effect and how specific elements of adverse exposures can interact or operate along separate mechanistic

pathways. Although promising, this approach currently lacks a consensus operationalization, as no measure has yet been designed to fully capture these dimensions (Berman et al., 2022; Machlin et al., 2025).

1.2. Conceptualizing Positive Experiences in the Context of Adversity

Research on resilience in the face of adversity has traditionally sought to identify specific protective factors and mechanistic components that result in positive outcomes, or in the lack of negative outcomes. Early resilience models often focused on either stable traits or protective environmental contexts, though most contemporary models assume a dynamic interplay between both (Rutter, 2012). Similar to how early investigations into childhood adversity largely focused on single-exposure models, much of the investigations into resilience have focused on single resilience factors in isolation. A systematic review that identified many individual- and family-level resilience factors that reduce psychopathology following various types of childhood adversity found that only eight out of 22 studies corrected for another resilience factor, despite the likelihood that such factors often co-occur and may exert mutual effects (Fritz et al., 2018a). Another review of 28 studies on mental health resilience in the context of cumulative adversity identified two studies that accounted for multiple protective factors (Buchanan et al., 2023). The field continues to struggle with clear and consistent operational definitions of resilience ((Kaufman et al., 1994; W. A. Walsh et al., 2010), leading researchers to call for cross-disciplinary integration to better operationalize childhood experiences and their neurodevelopmental impacts (Ioannidis et al., 2020).

Related to resilience is the growing interest in positive childhood experiences (PCEs)—including supportive relationships, enjoyable school environments, and routine family practices. Many of these overlap with protective factors in resilience models, though researchers have only

recently begun to systematically measure the co-occurrence of such experiences (Narayan et al., 2018; Schmitz et al., 2024). These are referred to throughout the literature as PCEs, protective and compensatory experiences (PACES), advantageous childhood experiences (counter-ACEs), and benevolent childhood experiences (BCEs). Importantly, such experiences appear to not just reflect the absence of adversity, but represent a somewhat-independent presence of enriching experiences.

An explosion of research has investigated how multiple positive experiences influence outcomes in the face of multiple adversities in recent years. A systematic review by Han et al. (Han et al., 2023) found that most studies identified direct, promotive effects of positive experiences on positive outcomes, including mental health outcomes. Although some studies identified an interactive effect wherein more positive experiences lowered negative outcomes for those exposed to adversity (seven of 23 studies), many did not. Such mixed evidence may indicate that positive experiences could simply enhance well-being regardless of adversity levels, rather than buffer its impact. However, most of these studies were cross-sectional, and whether interactive effects might depend on timing of exposure to either positive or adverse experiences is still unclear. A recent review of eight published longitudinal studies did find more evidence for a moderating or interactive effect, which may be observable through prospective rather than retrospective measurement (Kallapiran et al., 2025).

Integrating the literatures of adversity, resilience, and positive experiences reveals notable measurement discrepancies. Adversity is often assessed with relatively broad and validated tools, while positive experiences have traditionally been more narrowly and inconsistently measured. The BCEs revised scale, which includes ten new items (e.g., good sleep quality, exposure to the outdoors, fair treatment in the community), represents progress in

capturing further variability and breadth in enriching experiences (Narayan, Merrick, et al., 2023). Still, most scales rely on a summed score of yes/no exposure items that do not capture intensity, duration, or timing of any individual positive experience (Raghunathan et al., 2024). Moreover the scale of measurement in public policy initiatives is vastly different, and researchers have called for the increased screening and assessment of positive childhood experiences, which would have its own unique benefits, particularly in circumstances where screening for adversity has drawbacks (Merrick & Narayan, 2020). Additionally, it remains unclear whether dimensions akin to threat and deprivation exist within the domain of positive experiences, though some work suggests a relatively high impact of some domains (La Charite et al., 2023; Narayan, Frederick, et al., 2023), and what specific mechanisms such dimensions might involve. Given the vast body of research demonstrating the notable role both positive and adverse experiences play in health development, a research focus on mechanisms through which these influences occur is imperative for informing risk identification, treatment approaches, and treatment timing.

1.3. Brain Development as a Mechanism

Understanding how experiences shape the brain is a central aim of developmental neuroscience. Neural development has been proposed to be one of several core mechanisms, in coordination with immune, endocrine, metabolic, and cardiovascular development, through which adversity "gets under the skin" in a biological embedding process wherein experiences alter long term biological and developmental trajectories (Berens et al., 2017; McEwen, 2012). Indeed, childhood maltreatment and poverty, in particular, are known to affect both the structure, function, and connectivity of neural systems (Hamza et al., 2024; Kim et al., 2017; Luby, 2015; Teicher et al., 2016). Broad theoretical models of development, such as life course developmental theory and the transactional model of development, highlight the compounding

and reinforcing interplay between experiences and biology—including brain development—that occurs over the lifespan (Elder, 1998; Sameroff, 2009), wherein components of adaptation, risk, and resilience interact and evolve over time.

While adversity has the potential to exert effects on the brain at any point in life (Hilberdink et al., 2023), there are reasons to expect it to have increased influence at specific developmental stages, particularly when considering the relevance of the type of adversity at that stage. Important studies on severe levels of psychosocial adversity in the first few years of childhood, like the Bucharest Early Intervention Project, have revealed both the importance of receiving species-expected care within sensitive periods and the incredible capacity for resiliency when timely and appropriate intervention occurs within those sensitive periods (Zeanah et al., 2017).

Positive experiences are also known to shape neural development, though this literature remains comparatively underdeveloped. Much has focused on the enriching experiences of caregiving factors in younger childhood, such as caregiver warmth, responsiveness, and support, which are notably influential in brain development (Luby et al., 2013; National Academies of Sciences, Engineering, and Medicine, 2019). Little is known how multiple positive experiences together shape brain development. In a scoping review of the measurement of positive experiences, no identified study examined neurobiology as a potential mechanism or outcome (Raghunathan et al., 2024).

A useful theoretical framework that integrates many of these ideas is the Intergenerational and Cumulative Adverse and Resilience Experiences (ICARE) model proposed by Hays-Grudo and colleagues (Hays-Grudo et al., 2021). The ICARE model emphasizes that both adverse and resilience factors in early childhood result in adaptations across neurobiological

systems that, in turn, influence systems involved in cognitive, emotional, and social development, which ultimately affect health outcomes. The model acknowledges that these processes are not static but shaped by the continued presence of environmental stressors and can be modified by protective factors, suggesting trajectories of these related systems can dynamically adjust over time.

To date, it remains unclear how multiple positive experiences, in combination with adversity, shape the developing brain. Future work is needed to investigate fundamental questions: Do positive experiences exert independent effects that accumulate alongside adversity to influence development? Do they buffer or alter adversity-related changes in brain development? Does the type or amount of positive experiences matter in relation to specific dimensions of adversity? Clarifying these questions may help uncover mechanisms that contribute to the marked heterogeneity in outcomes following adversity and improve individual prediction.

1.4. A Focus on Adolescence

Adolescence may provide a key window to investigate these open questions. It is a neurobiologically-sensitive period, particularly in response to the stress response system (Eiland & Romeo, 2013; Sisk & Gee, 2022) wherein potential for both vulnerability and opportunity exist (Dahl, 2004; Sisk & Gee, 2022). Neurodevelopment in adolescence is especially dynamic with considerable inter-individual variability in trajectories (K. L. Mills et al., 2021a). As complex neural systems continue to refine, they may be susceptible to a variety of environmental inputs (Cheng et al., 2024; Ferschmann et al., 2022).

Adolescence is also notable for its increased rates of onset for many forms of psychopathology (Paus et al., 2008), and thus an important period to characterize how concurrent

experiences shape mental health trajectories. Longitudinal research tracking adverse and positive experiences in adolescents from ages 10-13 over five years found that positive experiences both reduced the effect of adversity and exerted independent effects on several aspects of mental and behavioral health in early adulthood (Crandall et al., 2020a).

Emerging evidence suggests that different positive experiences may also influence the impact of adversity on adolescent brain development. For instance, one study suggests that caregiving support could interact with early adversity in hippocampal and amygdala development (Luby et al., 2019). In a large sample from the Adolescent Brain Cognitive Development (ABCD) study, a data-driven approach identified distinct subtypes of environmental exposure based on caregiver support and neighborhood safety. These subtypes were differentially associated with cortical thickness and myelination, suggesting that different combinations of experiences contribute to different neural outcomes (Hong et al., 2021). Such early findings underscore the importance of not just characterizing the relationships between adversity and the developing brain but also assessing how positive experiences affect those mechanisms.

1.5. Recommendations

Ultimately, integrating the full spectrum of childhood experiences into a unified framework of neurodevelopment has the potential to offer a more complete understanding of how experience becomes biologically embedded and manifests in mental and behavioral health across the lifespan. As Sapolsky (Sapolsky, 2017) has compellingly noted, recognizing the value of safe, stable, nurturing, and enriching childhood should not depend on neurobiological evidence. Yet, when such insights help reduce stigma, foster self-understanding and compassion,

motivate policy change, or refine our approaches to intervention and prevention, they serve a critical purpose.

Testing integrative models of childhood experience and neurodevelopment will require attention to several key directions, many which have been noted by other prominent researchers. First, a focus on individual differences in both experience and neurodevelopmental outcomes will be essential. Given the complexity of these processes, person-centered and multivariate methods may be particularly useful for identifying meaningful patterns of co-occurring adverse and positive experiences in individual trajectories.

Second, longitudinal work remains indispensable to examine a core tenet of developmental science, which is that change unfolds over time (Fuligni et al., 2018). While some neurobiological or behavioral consequences of early experience may manifest with little temporal distance, others may remain latent until triggered later in development.

Third, the cultural and sociopolitical context in which experiences occur should be recognized and represented in study design. Experiential components, interpretation, and reporting can change across contexts, and systemic inequities shape both exposures type and amount and moderate outcomes. Researchers must attend to measurement invariance and conceptual relevance across diverse racial, ethnic, and cultural groups (T. M. Jensen et al., 2024; Kysar-Moon, 2019).

Finally, while research and interventions have often focused on reducing the deleterious consequences of adversity, there is less conceptual clarity around how to actively promote well-being and positive development (Pluess, 2024). Pluess emphasizes the importance of defining what positive outcomes look like in childhood and adolescence itself, rather than solely focusing on adult functioning. As models increasingly incorporate both adversity and positive

experiences, so too can outcomes reflect a balance in considering not only the presence of difficulties but also indicators of flourishing. This dual focus can inform both practical intervention goals as well as characterize a more holistic understanding of development.

2. Goals of this Dissertation

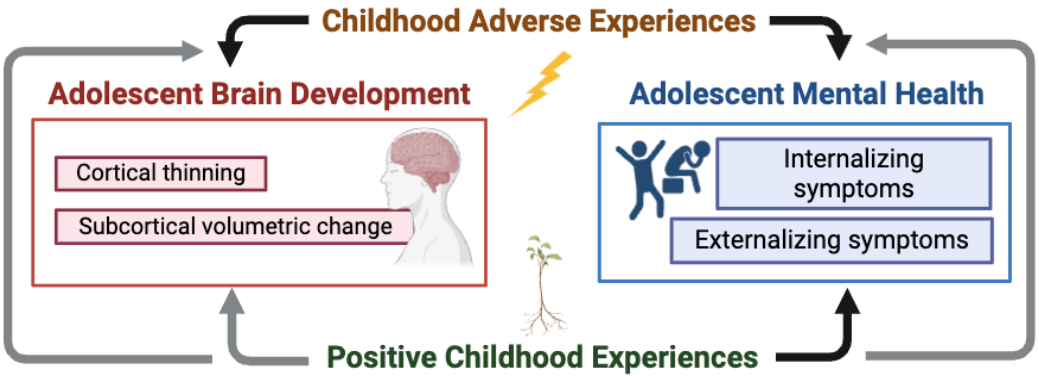
This dissertation aims to build upon the current research by integrating the rapidly developing fields of adversity science, clinical science, developmental neuroscience, and resilience science to advance scientific discourse on conceptual models, sharpen mechanistic understanding, and advance an integrative framework of risk and resiliency.

2.1. An Integrative Conceptual Model in Adolescence

Aligned with elements of the ICARE model, a focused model for this dissertation will be on how adverse and positive experiences independently influence adolescent brain development and mental health symptoms throughout adolescence and whether positive experiences interact with the adversity. Figure 1 illustrates both the hypothesized and exploratory pathways among these constructs of interest to this work. Although there is likely a bidirectional relationship between mental health and brain development, this dissertation does not examine that pathway, and it is therefore not included in this selectively representative model.

Figure 1

A Conceptual Model of Dissertation Aims



Note. Role of brain development in the relationship between adverse and benevolent childhood experiences and adolescent mental health. The black arrows signify hypothesized relationships, and the gray errors signify exploratory relationships.

2.2. Overview of Approach, Longitudinal Study, and Aims

Through the use of intersubject representation similarity analysis (IS-RSA), a multivariate method that represents and assesses relationships among inter-individual differences (Chen et al., 2020; Finn et al., 2020; van Baar et al., 2019), this dissertation aims to leverage novel methodological tools to provide a more granular understanding of how individuals diverge and converge in these constructs. IS-RSA offers distinct advantages for examining the complex interrelationships between experiential profiles and mental health trajectories. Traditional variable-centered approaches typically assess relationships between individual variables or composite scores, potentially obscuring the multidimensional patterns that characterize real-world experiences. IS-RSA, by contrast, preserves the multivariate structure of experiences by representing each individual's profile as a relative position in a high-dimensional space.

The fundamental premise of IS-RSA in this dissertation is that similarity in experiential profiles should predict similarity in outcome trajectories if these experiences meaningfully shape development. Rather than reducing complex experiential patterns to single variables, IS-RSA

quantifies the dissimilarity between individuals across multiple dimensions simultaneously, capturing the holistic configuration of experiences. This approach is particularly well-suited for developmental research, as it acknowledges that experiences do not occur in isolation but rather in complex, interconnected patterns that may have synergistic, or possibly antagonistic, effects on developmental outcomes. Though this technique emerged in cognitive neuroscience, very recent work has begun to integrate IS-RSA into developmental science (Y. Li et al., 2025; Saragosa-Harris et al., 2025). However, no one to my knowledge has modeled longitudinal data with IS-RSA.

By applying IS-RSA to longitudinal data, this dissertation can examine whether adolescents who have similar patterns of adverse and positive experiences also show similar trajectories of mental health symptoms or brain development over time. To conduct these analyses, I draw upon the Transitions in Adolescent Girls Study (though not all participants identify as girls), which provides repeated measurements in these constructs across multiple years (Barendse et al., 2020). As the empirical chapters draw from the same sample and have overlapping methods, descriptions of those elements will be correspondingly identical across chapters.

Each chapter is organized to further the overall research question. Chapter II is an empirical study into the relationships of both adverse and positive experiences with internalizing and externalizing problems across adolescence. Chapter III was published and co-authored with T. Cheng, J. Flannery, K. Mills, P. Fisher, C. McCann, and J. Pfeifer. It investigates conceptual models of adversity and their relationship to adolescent brain development. Chapter IV is an empirical study into the relationship between positive experiences and brain development, and

assesses whether positive experiences moderate adversity's effect on development. Finally, Chapter V integrates and discusses these findings.

CHAPTER II: CHILDHOOD EXPERIENCES & MENTAL HEALTH

1. Introduction

1.1. Current State of Adolescent Mental Health

Adolescence is a critical developmental period characterized by rapid psychological, biological, and social transformations. Within this key life transition, the increasing prevalence and onset rates of mental illness have emerged as a significant public health concern. Early- to mid-adolescence, in particular, constitutes a period of heightened risk for numerous mental health problems, with mental health-related distress typically worsening throughout the adolescent years (Hankin et al., 1998; Hawton et al., 2012; Kessler et al., 2005; Yoon et al., 2023). The rising rates of mental health difficulties are predominantly driven by increasing internalizing symptoms—symptoms reflecting internal experiences of psychological distress—rather than externalizing symptoms, which manifest as externally directed behaviors indicative of psychological distress (Askari et al., 2022; Sappenfield et al., 2018). Despite differences in prevalence rates, research indicates that internalizing and externalizing symptoms frequently co-occur (Bista et al., 2025).

Adolescent girls demonstrate particular vulnerability to internalizing symptoms and the development of related disorders, including anxiety and depression (Askari et al., 2022; Keyes & Platt, 2024; Sappenfield et al., 2018; Yoon et al., 2023). Recent epidemiological data from 2023 revealed anxiety and depression rates among adolescent girls in the United States (U.S.) at approximately 20% and 10%, respectively (Sappenfield et al., 2018). Elucidating the contextual factors, risk mechanisms, and protective elements relevant to adolescent girls remains critically important for the development and refinement of effective prevention strategies.

Psychopathology is profoundly influenced by contextual environments, and contemporary generations of adolescents are developing within unique and influential contexts. Although concerns regarding adolescent mental health were already escalating prior to the COVID-19 pandemic, this global health crisis substantially exacerbated psychological distress among youth (Wolf & Schmitz, 2024). Pandemic-related increases in depression and anxiety symptoms were most pronounced among older adolescents and adolescent girls (Benton et al., 2021), with youth possessing a history of mental health challenges demonstrating particular vulnerability (Panchal et al., 2023). Beyond this relatively acute contextual factor, adolescents also must navigate both novel and persistent societal challenges. In presenting a new ecology of early childhood, Fisher and Lombardi ((Fisher & Lombardi, 2025) acknowledge the challenges presented by modern contexts to development outcomes in early childhood. Certain challenges, such as intensifying economic inequality and a dangerously changing climate, also likely impose unique stresses and present an uncertain future for adolescents.

The influence of pervasive digital technology on adolescent mental health remains the subject of considerable scientific inquiry and public debate. An umbrella review encompassing 25 systematic reviews (Valkenburg et al., 2022) determined that associations between social media use or social networking site engagement and mental health outcomes are generally weak or inconsistent. The directional effect on psychological well-being, either beneficial or detrimental, largely depends on individual users, with equal proportions of adolescents experiencing either benefits or harm to their well-being. For most adolescents, psychological functioning appears relatively unaffected. While the effects of digital contexts are not uniform, they nonetheless represent important environments that can amplify both the positive (Zhou et al., 2015) and negative experiences during adolescence.

1.2. Adversity and Adolescent Mental Health

Exposure to adversity stands out as one of the most robust predictors for mental health difficulties during adolescence. Childhood maltreatment, encompassing forms of abuse (i.e., physical, sexual, and emotional) and neglect (i.e., emotional and physical), has been consistently associated with both internalizing and externalizing problems during adolescence (Badr et al., 2018; Khadr et al., 2018; R. Mills et al., 2013). Bullying victimization, another salient form of psychosocial distress during adolescence, is also associated with deteriorated mental health outcomes across both in-person and online contexts (Bottino et al., 2015; Man et al., 2022; Ringdal et al., 2021). Peer victimization, isolation, and rejection have been shown in some studies to have greater impacts on adolescent mental health compared to parental maltreatment (Finkelhor, 2018; Price et al., 2020).

Socioeconomic status (SES) constitutes another well-established risk factor for adolescent mental health problems (Peverill et al., 2021; Reiss, 2013). It is important to acknowledge that while lower SES may not be an adversity per se, it represents a contextual condition that substantially increases the likelihood of exposure to various adversities and constrains access to resources that might buffer against negative outcomes. While most investigations examine cross-sectional associations, several longitudinal studies have explored how SES relates to trajectories of internalizing and externalizing over time. Though relatively limited in number, these investigations have yielded somewhat mixed findings. One multinational study encompassing low-, middle-, and high-income countries identified that higher household income was associated with greater decreases in children's internalizing symptoms across a three-year period spanning late childhood to early adolescence (Lansford et al., 2019). Conversely, data from the Adolescent Brain Cognitive Development (ABCD) study

indicated that lower income-to-needs ratio was associated with *less* increase in internalizing symptoms compared to high income-to-needs ratio (Rakesh et al., 2025). These researchers did find, however, that lower income-to-needs ratio was associated with higher internalizing and externalizing problems for girls in cross-sectional analyses.

With many adversities exerting individual risk effects, researchers have investigated whether the accumulation of adversities influence mental health outcomes. Accumulation is often measured through the Adverse Childhood Experience (ACEs) questionnaire, which quantifies exposure across ten adverse experiences related to childhood maltreatment and household dysfunction (e.g., parental mental illness). Large, cross-sectional studies indicate that cumulative ACEs scores increase risk of mental health problems (Bomysoad & Francis, 2020; Gajos et al., 2022). Adolescents with lower socioeconomic backgrounds are disproportionately exposed to higher rates of ACEs (D. Walsh et al., 2019), and ACEs appear to indirectly mediate a portion of the relationship between SES and mental health outcomes (Barnhart et al., 2022; Straatmann et al., 2020). Notably, the ACEs questionnaire is not fully inclusive of many adverse exposures and does not encompass peer-induced adversity.

1.3. The Influence of Cumulative Positive Experiences

While adversity and its cumulative effects have received substantial scientific attention as risk factors, researchers have increasingly begun to examine how positive experiences and their accumulation shape adolescent outcomes. Positive experiences, including elements of predictability, safety, support, connection, and enjoyment, are not merely the absence of adverse experiences but occur with a degree of independence (Crandall et al., 2020b; Doom et al., 2021; Merrick et al., 2019; Narayan et al., 2018). Positive experiences can coexist with adversity, and studies have identified populations characterized by both high adverse and positive exposures

(Hou et al., 2022; Narayan et al., 2018). A proportion of individuals also experience both low positive experiences and low adversity (Hou et al., 2022). Converging evidence suggests that positive experiences are associated with improved mental health outcomes even within the contexts of adversity (Han et al., 2023). A 2025 systematic review identified eight published longitudinal studies, most encompassing adolescence, that examined relationships between positive experiences and various physical and mental health outcomes (Kallapiran et al., 2024). In contrast to an earlier review that included predominantly cross-sectional and adult-focused studies (Han et al., 2023), this longitudinal review found stronger evidence for a moderating role of positive experiences.

To my knowledge, no research has investigated the relationship between positive experiences and trajectories of mental health symptoms within any developmental period. As adolescence may also represent a period of enhanced opportunity for resilience (Sisk & Gee, 2022), it is imperative to integrate investigations of positive experiences with adverse experiences to understand heterogeneity in mental health trajectories.

1.4. The Present Studies

While the effects of adversity on developmental outcomes are well-established, less is known about how these experiences influence change in mental health symptoms throughout adolescence. Additionally, it remains unclear whether analyses focused on direct estimates of these measures align with approaches that investigate the dissimilarity patterns of these constructs. Few studies on adolescence have incorporated multidimensional variables of both adverse and positive experiences to test their parallel and interactive effects.

In the current studies, I employ a multivariate approach to investigate how structures of participant experiences relate to developmental trajectories of internalizing and externalizing

symptoms. Given that adverse and positive experiences are multifaceted, and often co-occurring, this method allows the continuous landscape of interpersonal dissimilarity across these different components to be reflected. In modeling adversity, I examine both the effect of all measured adversity data, and I also separate the adversity data into two representations that aim to capture threat and deprivation dimensions. Although the dimensional model of adversity and psychopathology does not theorize that threat and deprivation would differ in their relationship to internalizing or externalizing symptoms, some evidence does suggest that there is reason to believe these specific dimensions of adversity have differential or interactive effects on internalizing and externalizing symptoms (Miller et al., 2018a; Schäfer et al., 2023; Stein et al., 2022a). Because of this evidence and the novelty of this study's analytic approach, I have chosen to additionally examine the constructions of threat and deprivation.

The primary research question driving this work is to determine whether differences in individuals' experiential profiles—encompassing both negative and positive dimensions—relate to differences in their mental health trajectories across adolescence. To address this question, I examine both internalizing and externalizing symptoms from multiple reporters across early- and mid- adolescence. Study 1 examines how inter-individual differences in adverse childhood experiences, measured through early adolescence, relate to inter-individual differences in mental health symptoms. Study 2 investigates whether inter-individual differences in positive experiences exert additive and/or interactive effects with inter-individual differences in adversity. By leveraging longitudinal data and a representational similarity framework, these studies aim to advance understanding of how experiential contexts shape adolescent mental health in nuanced and potentially synergistic ways.

2. Method

2.1 Sample Characteristics

This project uses up to six waves of data gathered through the Transitions in Adolescent Girls* (TAG; *not all study participants identify as girls) study (Barendse et al., 2019). Study recruitment occurred in 2015 and was aimed towards students registered as female in Lane Country, Oregon, USA who were aged 10 to 13 years of age. The study was approved by the University of Oregon Institutional Review Board, and assent and informed consent were obtained from the adolescents and their caregivers, respectively, before inclusion in the study. Exclusionary criteria included parent-reported diagnosis of select developmental disabilities (i.e., an autism spectrum disorder, cerebral palsy, or intellectual disability), diagnosis with a psychotic disorder, use of psychotropic medication other than stimulants, reported or suspected pregnancy, claustrophobia, and presence of ferromagnetic material in the body that would prohibit magnetic resonance scanning. 189 adolescents and their parents were initially recruited, and 174 passed exclusionary criteria and participated in the first wave of the study.

The sample includes a more diverse distribution of racial and ethnic identities than the surrounding community; a detailed breakdown is presented in Table 1. Table 2 also includes information on participants' gender identities, which reflect changes in identification across study waves. These social identities are fluid and may evolve over time. The study did not disproportionately lose participants with racial or ethnic minority identities due to attrition.

Table 1

Race and Ethnicity

Race	Non-Hispanic/Latina/Chicana	Hispanic/Latina/Chicana	Percent Non-Hispanic/Latina/Chicana	Percent Hispanic/Latina/Chicana
Black	0	1	0.00%	0.57%
Asian	2	0	1.15%	0.00%
NAAN	1	0	0.57%	0.00%
White	118	20	67.82%	11.49%
White and NAAN	9	2	5.17%	1.15%
White and NHPI	0	1	0.00%	0.57%
White and Middle Eastern	1	0	0.57%	0.00%
White and Asian	8	0	4.60%	0.00%
White and Asian and NHPI	0	1	0.00%	0.57%
White and Asian and NAAN	1	0	0.57%	0.00%
Black and White	8	0	4.60%	0.00%
Not Specified	0	1	0.00%	0.57%

Note. 32.2% of participants identified as a racial or ethnic minority. Comparatively, 18.2% of the local county identifies as a racial or ethnic minority. NAAN = Native American and Alaskan Native; NHPI = Native Hawaiian and Pacific Islander.

Table 2

Gender Identification across Five Waves

	Wave 1	**Wave 2**	**Wave 3**	**Wave 4**	**Wave 5**
Clinician-Administered Measure	% (N)	% (N)	% (N)	% (N)	% (N)
Female	98.35% (179)	96.93 (158)	93.57% (131)	88.39% (99)	85.71% (84)
Male	0.00% (0)	0.00% (0)	0.71% (1)	1.79% (2)	2.04% (2)
Other	1.65% (3)	2.45% (4)	5.71% (8)	9.82 (11)	12.24% (12)
Total N	182	162	140	112	98

Note. Gender identity as self-reported by the adolescent to a clinical interviewer. Sample size at Wave 1 exceeds 174 due to the collection of gender data from several adolescents prior to study exclusion.

Each wave of data collection took place approximately 18 months apart (range of 0.72-

36.65 months due to pandemic-related disruptions). Out of 174 participants in the first wave, 163 (93.68%) participants participated in the second wave, 150 (86.21%) participated in the third wave, 109 (62.64%) completed the fourth wave, 100 completed the fifth wave (57.47%), and as of the time of preparing this manuscript, 59 completed the sixth wave (33.91%), which is still ongoing. Questionnaires were completed at session visits and at home; the clinical interview was completed during the session visit either in person or via Zoom.

Prior analyses with these data examined relationships from one of the proposed study variables (Wave 1 adversity measures; (Barrett et al., 2024)). Instead of examining brain development as an outcome, this study investigates how adversity relates to mental health trajectories.

2.2. Measures

2.2.1. Mental Health Measures

2.2.1.1. Child Behavioral Checklist (CBCL)

The CBCL is a 111-item parent-report measure designed to assess emotional and behavioral problems in children (Achenbach & Rescorla, 2001). Caregivers rated items on a three-point scale (0 = not true, 1 = somewhat/sometimes true, 2 = very true/often true). Items were summed to yield subscale scores for internalizing problems (i.e., anxious/depressed, withdrawn, somatic complaints) and externalizing problems (i.e., attention problems, rule-breaking behavior, and aggressive behavior).

2.2.1.2. Center for Epidemiological Studies-Depression Scale Child (CES-DC)

Adolescents completed the CES-DC, a 20-item self-report measure assessing depression-

related symptoms experienced over the previous week (Weissman et al., 1980). Items were rated on a four-point scale (0 = not at all to 3 = a lot) and summed to generate a total score, with higher scores indicating greater depressive symptomatology.

2.2.1.4. Screen for Anxiety Related Emotional Disorders (SCARED) Brief Assessment of Anxiety and PTSD Symptoms

Self-reported anxiety symptoms were measured using nine items; five from the brief SCARED (Muris et al., 2000) and four from the PTSD subscale items of the revised SCARED (Muris et al., 1999). Items were summed, with higher scores indicating great anxiety symptoms.

2.2.1.5. Revised Self-Consciousness Scale for Children (R-SCS)

The R-SCS consists of 29 self-reported items assessing components of self-consciousness (Takishima-Lacasa et al., 2014). Items were rated on a five-point Likert scale ranging from strongly disagree to strongly agree. For the present analyses, only the social anxiety subscale was used.

2.2.1.6. Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS)

Trained interviewers administered a semi-structured diagnostic interview, the K-SADS, to assess the presence or absence of common mental health diagnoses at the time of interview and since the last wave of participation (or across the lifetime at Wave 1). Internalizing disorders assessed included depressive disorders, anxiety disorders, obsessive-compulsive disorders, trauma-related disorders, and eating disorders. Participants were coded as 1 if they met criteria for an internalizing disorder during the

relevant time frame and as 0 if they did not. During the same interview, participants also self-reported whether they had received any school detentions in the past year; responses were similarly binary coded.

2.2.1. Adverse Childhood Experiences

2.2.1.1. Childhood Trauma Questionnaire (CTQ)

We administered the CTQ form to adolescent participants with 32 items, but only the standard 25 items from the validated short form were used for these analyses. It has five subscales: physical abuse, emotional abuse, sexual abuse, emotional neglect, and physical neglect. The CTQ has demonstrated high convergent validity (Bernstein et al., 1994, 1997) with therapist ratings and trauma interviews and exhibits high levels of internal consistency across age groups (Bernstein et al., 2003; Scher et al., 2001; Spinhoven et al., 2014). Initial test-retest reliability coefficients vary between .79 and .86 across 2-6 months (Bernstein et al., 2003) and .44 to .67 across five years (Cammack et al., 2016).

2.2.1.2. Socioeconomic Status (SES) Measures

We used a single-item caregiver report on whether their child received free lunch at school since both household income and family size influence eligibility. We also included caregiver education (highest report if a participant had more than one caregiver) which indicates if caregivers received no high school diploma (or equivalent); high school diploma (or equivalent); some college credit (no degree), trade, technical, or vocational training; associate degree, undergraduate degree, or postgraduate degree. Finally, we used participants' residential addresses to create an index of neighborhood deprivation based on the computation provided by Messer et al. (Messer et al., 2006) that

incorporates census data on income, education, employment, housing, and occupation.

2.2.1.3. Youth Risk Behavior Scale (YRBS)

Two items from the YRBS were used to assess if the participant had ever been bullied on school property or electronically (i.e., through email, chat rooms, instant messaging, websites, or texting). Data was also summed to provide one composite measurement.

2.2.1.4. Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS)

The following events evaluated in the K-SADS Post-Traumatic Stress Disorder module were used to assess further experiences of trauma: a significant car accident that resulted in injury and required medical intervention, other accident that resulted in injury and required medical intervention, witnessed a fire that caused significant property damage or moderate-severe physical injuries, witnessed a natural disaster that caused significant devastation, witness to threatening or violent crime, a victim of threatening or violent crime, terrorism-related traumatic event, war zone traumatic event, witness to domestic violence, physical abuse, and sexual abuse. These events were also summed to create a composite score.

2.2.3. Positive Childhood Experiences

2.2.3.1. Benevolent Childhood Experiences (BCEs) Scale

The BCEs scale (Narayan et al., 2018) includes ten binary items that assess if adolescent participants have experienced components of safety, support, predictability, and enjoyment during their childhood. Items were summed with the PCEs (see below) to create a total score of positive experiences, such that higher scores indicate more positive

experiences.

2.2.3.2. Positive Childhood Experiences (PCEs)

The PCEs were seven binary items adapted by Bethell et al. (Bethell et al., 2019) that assess if adolescent participants have had specific positive interpersonal experiences, such as being able to talk to family about one's feelings, enjoying participating in community traditions, and feeling safe and protected by an adult at home. Items were summed with the BCEs scale to create a total score of positive experiences.

2.3. Data Transformation

2.3.1. Adversity Data

Adversity data were transformed so that experiences that were measured with a greater number of items (e.g., physical abuse) do not have greater weight when calculating dissimilarity (see Analyses section for further details) than others. Items were divided by the number of items per experience (e.g., each of the five physical abuse items were divided by five). All items were transformed to a percent of the maximum score so that continuous variables were not weighted more heavily than categorical variables.

2.3.2. Mental Health Data

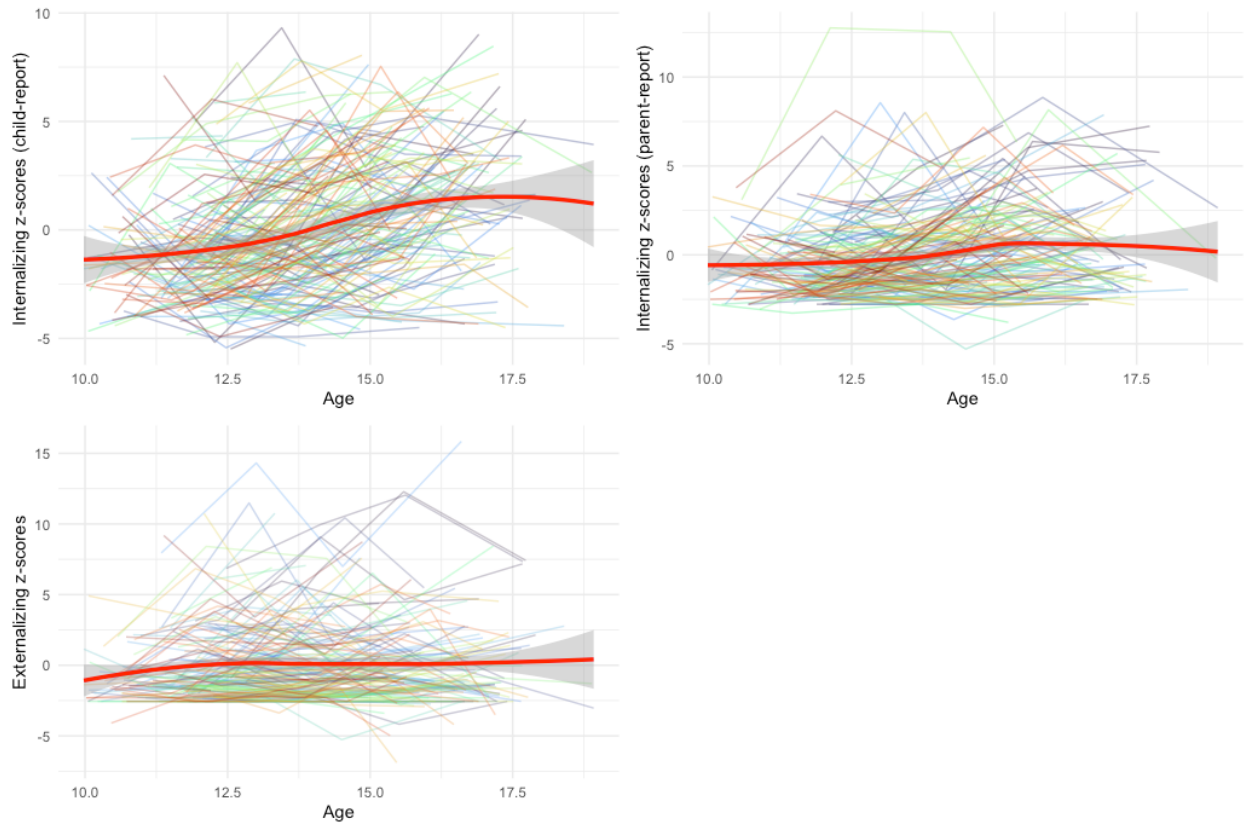
Three z-scored composites were created from the mental health symptom and diagnostic data: (1) parent-reported internalizing symptoms, (2) adolescent-reported internalizing symptoms, and (3) externalizing symptoms. The parent-reported internalizing composite included the three CBCL internalizing subscales: anxious/depressed, withdrawn, and somatic complaints. The adolescent-reported composite included the SCARED-R total score, the social

anxiety subscale of the R-SCS, and the presence of an internalizing diagnosis. The externalizing composite included the CBCL externalizing subscales for attention problems, rule-breaking behavior, and aggressive behavior, as well self-reported experience of school detention.

To examine trajectories in mental health symptoms over time, multilevel models were conducted to estimate random intercept and slopes by age for each participant. Only the adolescent-reported internalizing model converged with random slopes, suggesting sufficient variance in symptom changes over time. For the parent-reported internalizing and externalizing composites, only random intercepts were estimated, reflecting stable individual differences in the sample across adolescence. See Figure 2 for visualizations of individual trajectories.

Figure 2

Individual Mental Health Trajectories



Note: These trajectories illustrate the composite scores over time as estimated in one of five

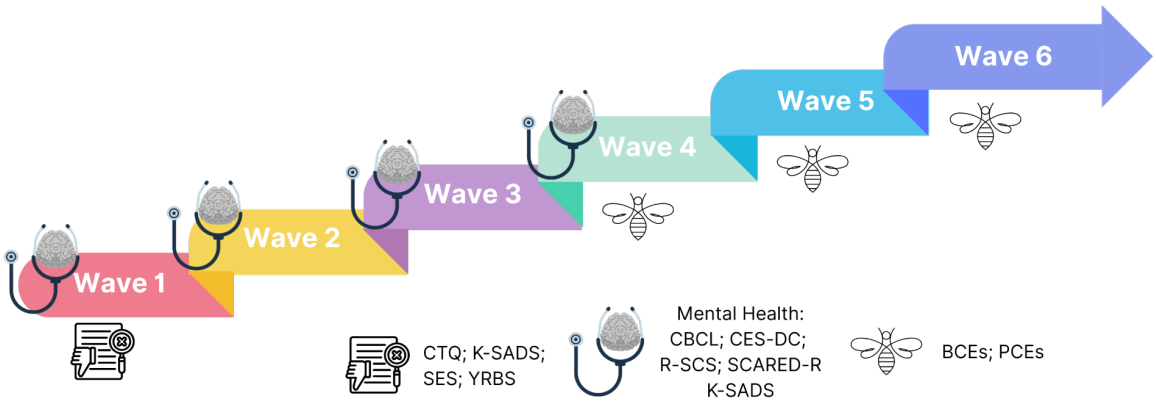
imputed data sets.

2.4. Procedure

Figure 3 displays the measures from the TAG study that were used for these analyses. For the primary analyses, adversity measurements–including CTQ data, K-SADS items, SES indicators, and the YRBS bullying items–were drawn from Wave 1. Measurements of positive experiences (i.e., BCEs and PCEs) were used from either Wave 4, 5, or 6, depending on availability. Mental health symptoms were drawn from Waves 1 through 4, as later wave data were not yet available for these analyses.

Figure 3

TAG Study Design: Key Timepoints and Measures Used in Analyses



Note. Though positive experience measures (BCEs and PCEs) were administered three times, only a small proportion of the sample completed multiples of those assessments, so only one time point was chosen of available data.

2.5. Missing Data

To address missingness in Wave 1 adversity measures, a stepwise imputation strategy was employed using the missForest algorithm (Stekhoven & Bühlmann, 2012), a non-parametric random forest–based method well-suited for high-dimensional data with mixed variable types and potential nonlinear relationships. Imputations were conducted in a sequential order based on theoretical relevance and presumed data quality. First, income was imputed using caregiver education, age, and race—variables that were largely complete and considered foundational demographic indicators. Next, K-SADS items, free lunch status, and neighborhood disadvantage index (NDI) variables were imputed using these demographic items along with the newly imputed income variable as predictors. Then, Childhood Trauma Questionnaire (CTQ) items were imputed using all demographic measures and the K-SADS items, to reflect the associative nature of adversity exposure. Finally, Youth Risk Behavior Survey (YRBS) items were imputed using CTQ items in addition to the prior variables. Across the Wave 1 adversity measures, 4 to 27 responses were missing among the full sample of 174 participants (2.3% to 15.2% missingness per item).

The percentage of missing data that were imputed for each mental health measure is reported in Table 3. Missing data on mental health variables were addressed using multiple imputation by chained equations (MICE; (Van Buuren, 2018). Given the longitudinal structure of the data, continuous variables were imputed using the 2l.pan method to account for within-subject clustering. Due to model convergence issues, the binary measurement of internalizing diagnosis was imputed using single-level logistic regression (logreg). Five imputed datasets were generated by using available mental health variables and the following adversity measures as predictors: history of being bullied, recipient of free lunch, and total CTQ score.

Table 3

Mental Health Measure Imputation

Measure	% Missing
CBCL Anxiety	13.83
CBCL Withdrawn	13.83
CBCL Somatic	14.67
CBCL Rule breaking	14.33
CBCL Aggression	14.67
CBCL Attention	14.00
CES-DC	11.17
SCARED-R Total	9.50
R-SCS Social Anxiety	4.33
Detentions	3.67
Internalizing Diagnosis	0.67

Note. Percentage of missing data out of 600 assessments.

Independent samples t tests comparing participants who completed the positive experience questionnaires and those who did not yielded no discernable differences in any of the mental health or adversity measurements. Therefore, I did not impute missing for those who had no positive experience questionnaire data for Study 2 analyses. For those who did complete those questionnaires (n = 110), there was one missing value for a BCEs item and one missing value for a PCEs item (0.9% of each item). These items were imputed with the mode value. After mode imputation of the two missing items, both the BCEs (α = .71) and PCEs (α = .75) were found to be reliable. Together, the 17 BCEs and PCEs items demonstrated high reliability (α = .86).

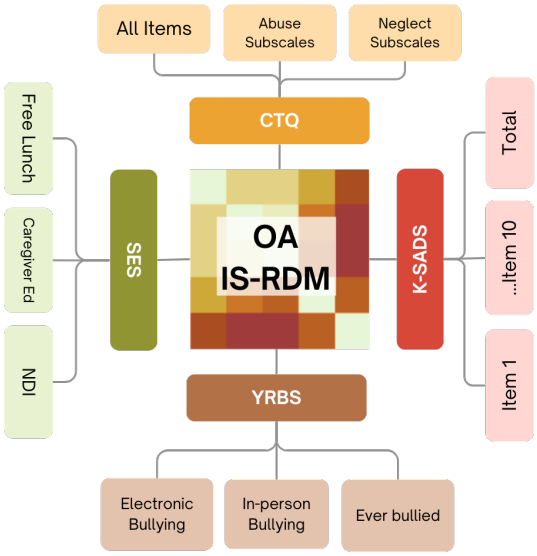
2.6. Analyses

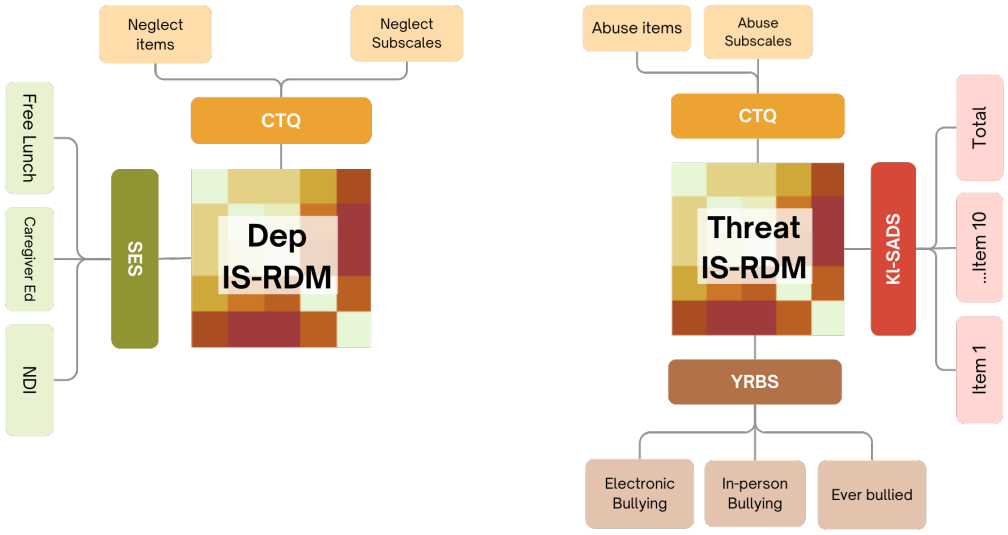
To assess whether inter-individual differences in childhood experiences relate to coordinated or divergent patterns of mental health symptoms, I used inter-subject representational similarity analysis (IS-RSA). IS-RSA is a multivariate framework that characterizes dissimilarity between participants by constructing inter-subject representational dissimilarity matrices (IS-RDMs), which contain pairwise Euclidean distances between participants based on constructs of interest. These square, symmetric matrices reflect all possible participant comparisons and preserve the full multivariate structure of the input data.

IS-RDMs were constructed for adversity-related constructs derived from relevant Wave 1 data: threat (Threat IS-RDM), deprivation (Deprivation IS-RDM), and overall adversity (OA IS-RDM), as illustrated in Figure 4.

Figure 4

Construction of Adversity Data into IS-RDMs



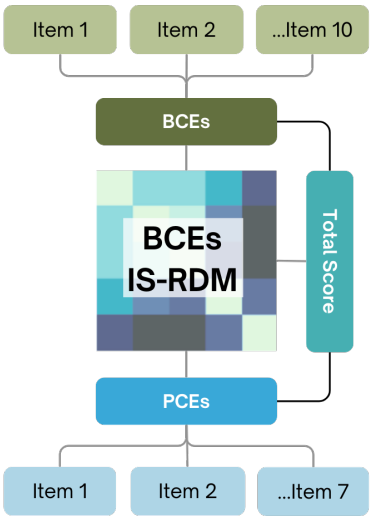


Note. The overall adversity (OA) inter-subject representational dissimilarity matrix (IS-RDM) represents all adversity measures. The Threat IS-RDM represents abuse items and subscales, trauma items and total, and bullying items. The Deprivation (Dep) IS-RDM represents neglect items and subscales, free lunch, caregiver education, and neighborhood deprivation index (NDI).

Additionally, an IS-RDM was constructed for benevolent childhood experiences (BCEs IS-RDM), calculated using later-wave BCE and PCE items and their total score (Figure 5).

Figure 5

Construction of Positive Experience Data into IS-RDM



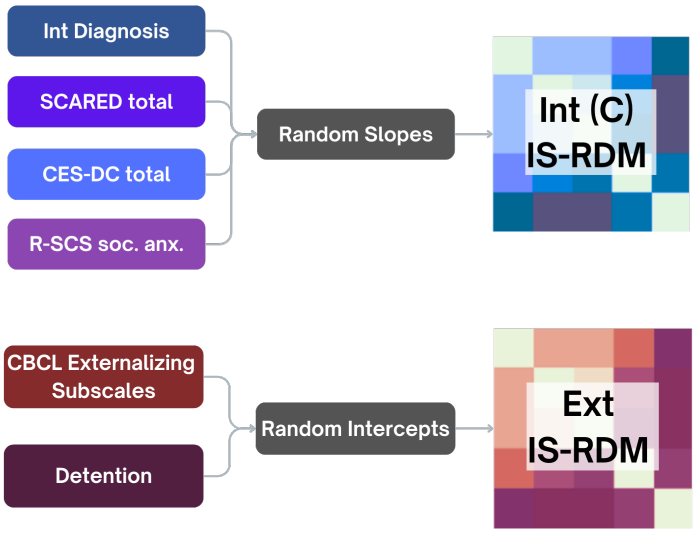
Note. The positive experiences inter-subject representational dissimilarity matrix (BCEs IS-RDM) represents all retrospectively collected BCEs and PCEs items as well as their joint total score.

IS-RDMs were also generated for mental health constructs. These matrices reflected pairwise differences between participants in their symptom experiences, based on either stable differences in reported symptoms (i.e., random intercepts) or change over time (i.e., random slopes, available only for adolescent-reported internalizing symptoms) as illustrated in Figure 6.

Figure 6

Construction of Mental Health Data into Outcome IS-RDMs





Note. This diagram shows how mental health data were grouped and transformed into the main outcomes for the parent-reported internalizing symptom intercepts (Int (P) IS-RDM), child-reported internalizing symptom slopes (Int (C) IS-RDM), and externalizing symptom intercepts (Ext IS-RDM).

All IS-RDMs were vectorized below the diagonal, standardized, and included in linear regression models. IS-RDMs were calculated separately for each of the five multiply imputed data sets; regression models were run within each set and results were pooled using Rubin's rules (Rubin, 1987). Multivariate Wald (D1) tests were used for nested model comparisons.

Because pairwise similarity data violate independence assumptions, additional non-parametric tests were conducted, similar to Mantel permutation tests. To test individual predictors for each imputed data set, one IS-RDM was randomly sampled and regression models were rerun 2,000 times, yielding 10,000 parameter estimates across imputations. The observed model parameter was compared to this null distribution to generate permuted *p*-values. To provide a conservative test of interaction effects, both IS-RDMs were randomly sampled and their interaction estimate was compared to the null distribution. Permuted p-values were

corrected for false discovery rate within each conceptually distinct set of model tests using the Benjamini-Hochberg (BH) procedure (Benjamini & Hochberg, 1995).

3. Results

3.1. Mental Health Descriptive Statistics

Table 4 displays the psychometric properties of the repeated measures of mental health variables before imputation.

Table 4

Summary Statistics of Mental Health Variables

vars	*n*	mean	sd	median	min	max	range	se
CBCL Anxiety	517	3.66	3.58	3.00	0.00	21.00	21.00	0.16
CBCL Withdrawn	517	2.20	2.43	1.00	0.00	14.00	14.00	0.11
CBCL Somatic	512	1.83	2.30	1.00	0.00	16.00	16.00	0.10
CBCL Rule-breaking	514	1.41	2.42	0.00	0.00	23.00	23.00	0.11
CBCL Aggression	512	3.29	3.89	2.00	0.00	24.00	24.00	0.17
CBCL Attention	516	3.23	3.36	2.00	0.00	17.00	17.00	0.15
CES-DC Total	533	16.70	11.96	13.00	0.00	57.00	57.00	0.52
SCARED Total	543	2.09	1.94	2.00	0.00	10.00	10.00	0.08
RSCS Social Anxiety	574	22.20	5.94	22.00	7.00	35.00	28.00	0.25
Detentions	578	0.34	2.68	0.00	0.00	50.00	50.00	0.11
Internalizing Diagnosis	596	0.37	0.48	0.00	0.00	1.00	1.00	0.02

Note. CBCL = Child Behavior Checklist; CES-DC = Center for Epidemiological Studies-

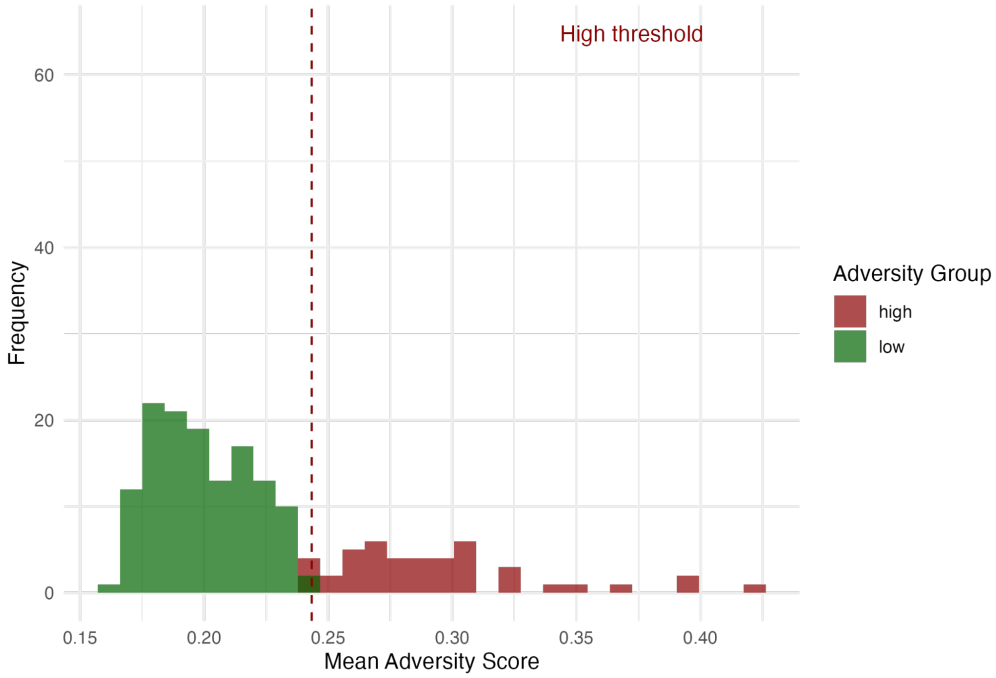
Depression Scale Child; SCARED = Screen for Anxiety Related Emotional Disorders; RSCS = Revised Self-Consciousness Scale for Children.

3.2. Drivers of Dissimilarity in Adverse Childhood Experiences

The adversity dissimilarity matrices were based on a relatively heterogeneous set of measures, making it unclear which specific variables contributed most to the highest distances. It was also ambiguous whether greater dissimilarity tended to occur between individuals with overall high versus low levels of adversity, or between individuals who both had high adversity scores but experienced qualitatively different types of adversity. To contextualize these dissimilarity patterns, I created a total adversity score by calculating the mean across all adversity-related variables and divided participants into high and low adversity groups. Those in the top 25% of adversity scores were classified as the high adversity group, while the remaining 75% comprised the low adversity group (see Figure 7 for the distribution of adversity scores). Each pairwise comparison was thus categorized as high-high, high-low, or low-low. Figure 8 displays the distribution of dissimilarity scores for each of these comparison types. I also examined the 5% of interpersonal pairs with the highest dissimilarity to identify which groupings contributed most to these extreme values. The majority were high-low comparisons (n = 599), followed by some high-high comparisons (n = 154); none were low-low.

Figure 7

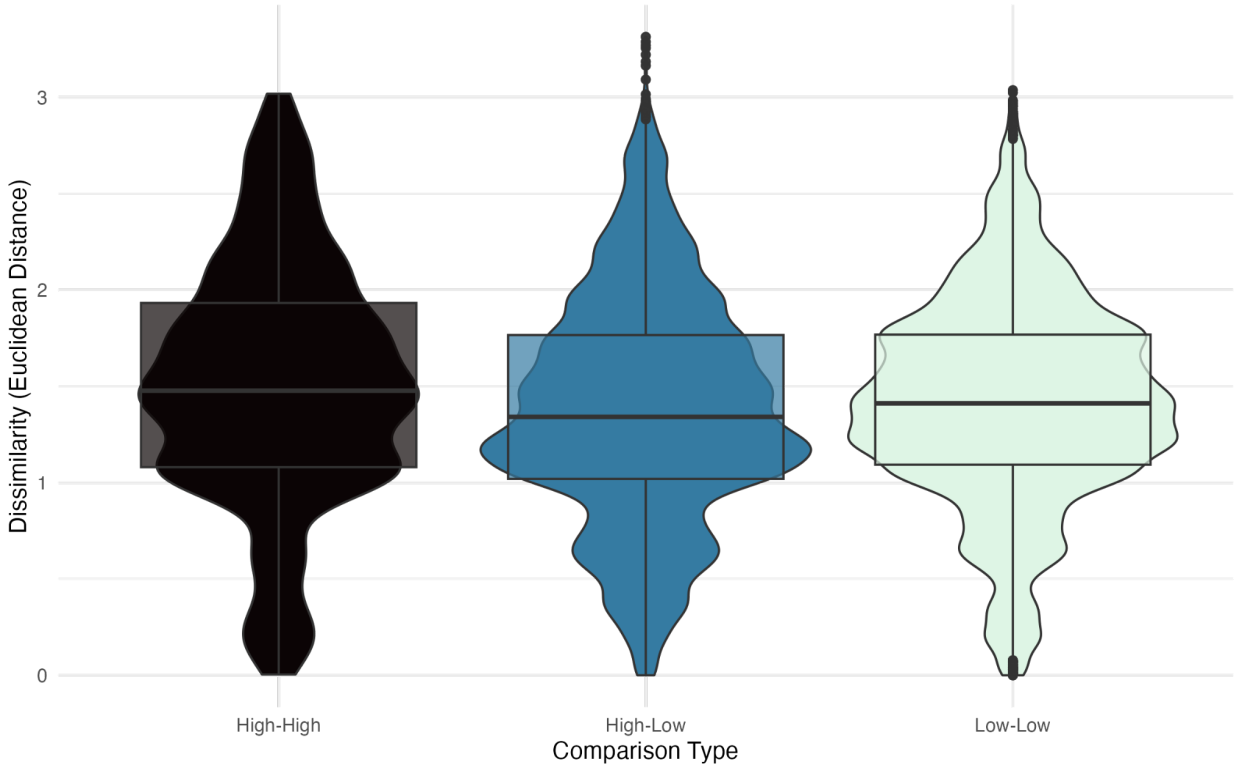
Distribution of Mean Adversity Scores



Note. The green group represents the lower 75% Wave 1 participant adversity scores.

Figure 8

Dissimilarity in Overall Adversity by High and Low Comparison Group Type



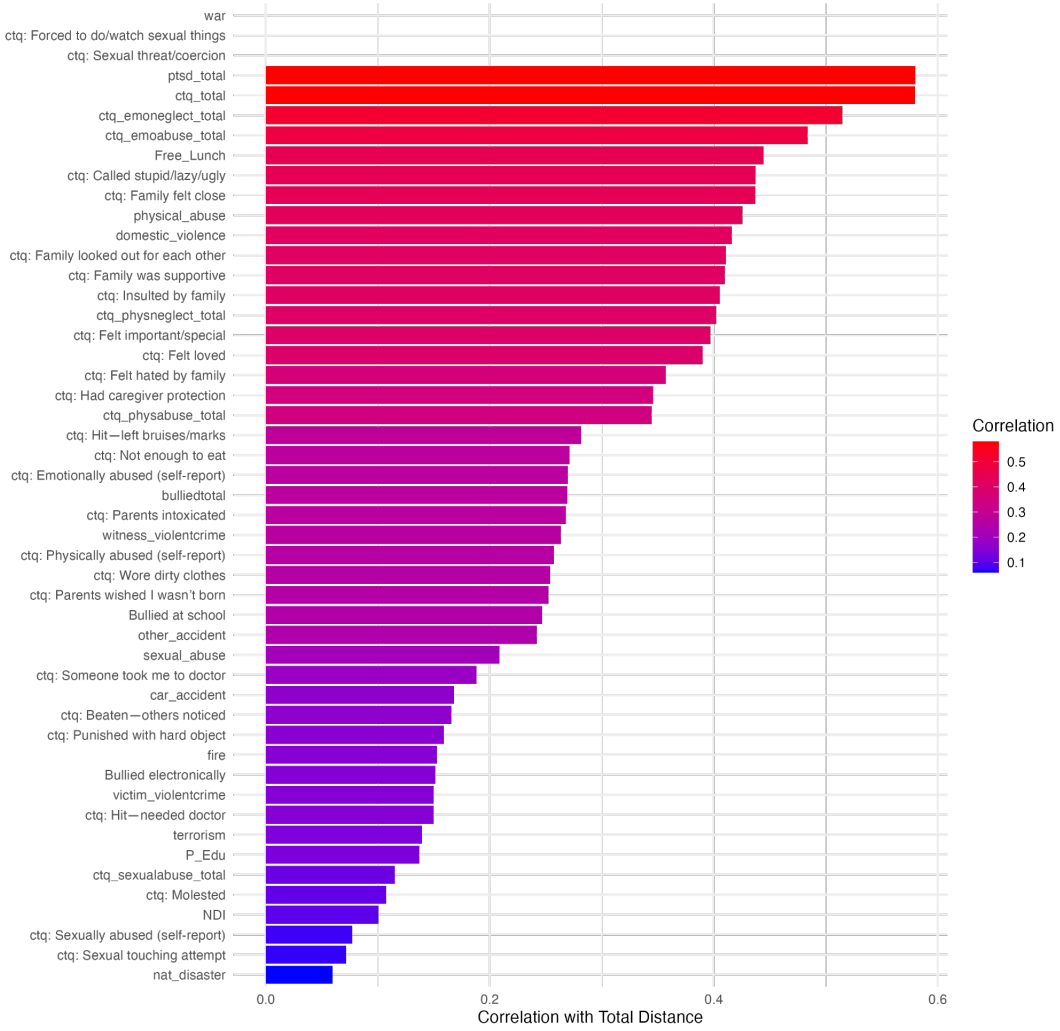
Note. Boxplots show that dissimilarity mean was largely similar across adversity comparison types, though the high-high comparisons contained most of the highest distance scores.

I also investigated how each variable individually contributed to the OA IS-RDM. To calculate those contributions, I computed an IS-RDM for each individual variable separately (i.e., one-dimensional Euclidean distances) and calculated the Pearson correlation between the vectorized lower triangle of the matrix of the individual IS-RDMs with the full OA IS-RDM. Higher correlations indicate greater alignment between variable-specific distances and the overall pattern of dissimilarity for all included items among all participants. This approach allows for the identification of which variables most strongly reflect the structure captured by the full IS-RDM, as shown in Figure 9. Top variable contributors to the OA IS-RDM include total exposure to trauma (as endorsed in the K-SADS interview PTSD module), CTQ total and the emotional neglect and emotional abuse subscale scores, and qualification for free school lunch.

Variables that were least represented by the OA IS-RDM included those less frequently endorsed in our sample, such as sexual abuse items and experiencing a natural disaster, as well as NDI and caregiver education. Three items (experiencing war, forced to do/watch sexual things, and experiencing sexual threat/coercion) had no variance, and consequently were not represented in this OA IS-RDM.

Figure 9

Contribution of Individual Variables to OA IS-RDM



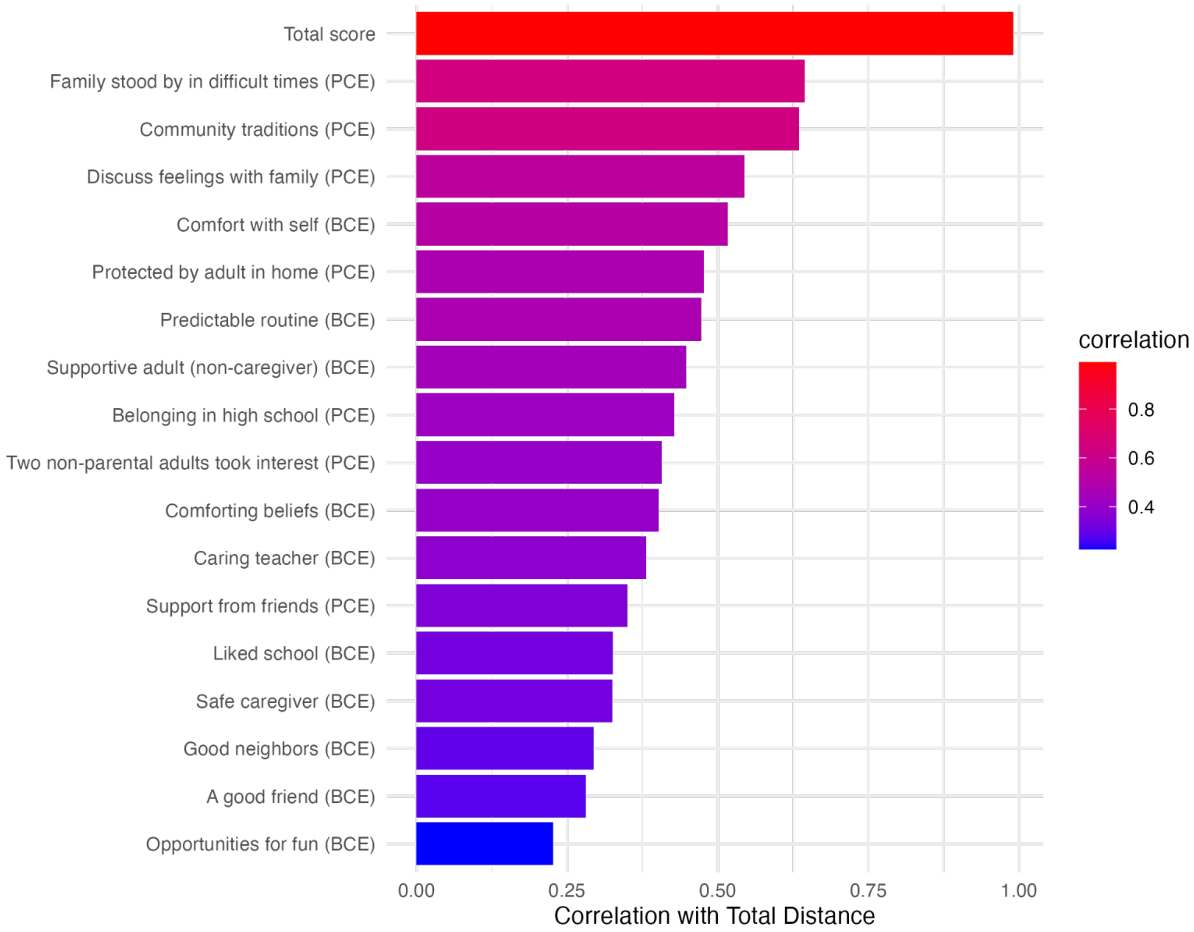
Note. Distances in each adversity variable correlating with the overall adversity (OA) IS-RDM.

3.3. Drivers of Dissimilarity in Benevolent Childhood Experiences

Figure 10 displays the contribution of each of the PCEs and BCEs items, as well as their total score, to the full BCEs IS-RDM. Distances according to the total score are almost completely correspondent with the full RDM. Individual items that contain the most distance among participants (i.e., are correlated over .5 with the full RDM) include feeling that one's family stood by oneself during difficult times, having enjoyed participating in community traditions, having been able to discuss feelings with family, and having liked or felt comfortable with oneself.

Figure 10

Contribution of Individual Variables to BCEs IS-RDM



Note. Distances in each positive experience variable correlating with the BCEs IS-RDM. PCE items are part of the positive experiences scale. BCE items are part of the BCEs scale.

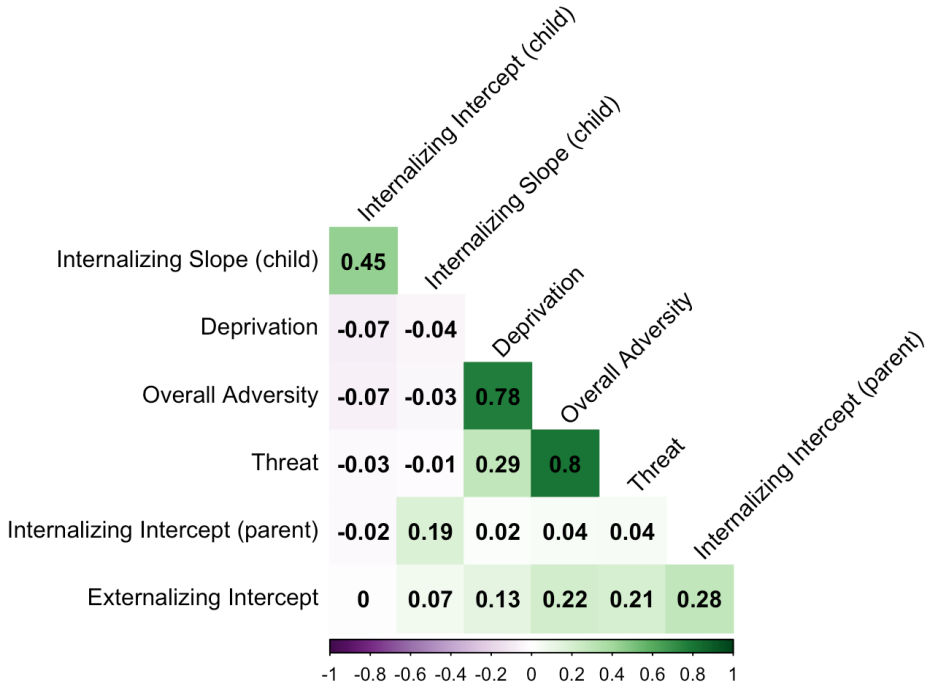
3.4. Study 1 Results: Adverse Experiences and Mental Health

3.4.1. Associations among Study 1 IS-RDMs

Figure 11 visualizes the correlations for the IS-RDMs for the subsample in Study 1 (n = 174). Correlations were pooled across imputed datasets using. For each pair, Pearson correlations were extracted from each imputed dataset, transformed with Fisher's z-transformation, averaged to compute a pooled estimate, and back-transformed to the correlation scale. Notably, parent-reported internalizing intercept is not strongly associated with the child-reported intercept, however it is more correlated with child-reported internalizing slope.

Figure 11

IS-RDM Pooled Correlations



Note. These correlations are between the mental health, and adverse experience IS-RDMs. Correlation coefficients were pooled across imputed data sets.

3.4.1. Adversity and Internalizing Intercepts (Parent-report)

Linear models indicate that both overall adversity (OA) and dimensional models of adversity dissimilarity constructions predict dissimilarity in inter-individual stable differences in internalizing symptoms as measured through parent reports (Internalizing (P); see Table 5 for model results). The OA IS-RDM was significantly associated with the Internalizing (P) IS-RDM. In the dimensional model, only the Threat IS-RDM showed a significant main effect on the Internalizing (P) IS-RDM, though this relationship was moderated by the Deprivation IS-RDM. This interaction suggests that the association between threat and internalizing symptom dissimilarity was weaker when participants were more similar in deprivation exposure. However, this interaction did not significantly improve model fit as determined by D1 comparison tests (F[1, 4, 15047] = 4.72, p = .096). Although both overall adversity and dimensional model effects were statistically significant, the variance of parent-reported internalizing intercepts explained by these models was quite small (adjusted R^2 = .001–.002).

Table 5

Internalizing Symptom (Parent-report) Dissimilarity: Pooled Adversity Model Results

	OA	Dimensional	Threat*Dep
Intercept	0.000 (0.008)	0.000 (0.008)	0.005 (0.009)
	p = 1.000	p = 1.000	p = 0.520
Overall Adversity (OA)	0.035** (0.012)		
	p = 0.008		
Threat		0.037* (0.015)	0.037* (0.015)
		p = 0.044	p = 0.042
Deprivation (Dep)		0.014 (0.012)	0.015 (0.011)
		p = 0.240	p = 0.208
Interaction			-0.019* (0.009)
			p = 0.027
R2	0.001	0.002	0.002
R2 Adj.	0.001	0.002	0.002
Num.Obs.	15051	15051	15051
Permuted Term	OA	Threat	Threat*Dep
Permuted p-value	<.001***	<.001***	0.018*
Adjusted p-value	<.001***	<.001***	0.018*

Note. Standardized estimates for regression models where outcome is parent-reported internalizing intercept dissimilarity are shown in each column. Estimates are standardized. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation.

3.4.2. Adversity and Internalizing Slopes (Child-report)

Linear models tested whether inter-individual differences in adversity were associated with differences in adolescents' internalizing symptom trajectories (i.e., random slopes;

Internalizing (C) IS-RDM) as reported by youth, controlling for stable differences in internalizing symptoms (random intercepts; see Table 6 for model results). Across all models, the random intercept IS-RDM was included as a covariate. The stable effect of the random intercept IS-RDM across models (estimates ranged from .441 to .443) indicates that individual differences in adolescent-reported symptom severity in early adolescence reliably predict differences in symptom change over time.

In the cumulative adversity model, the OA IS-RDM was not significantly associated with the Internalizing (C) slope IS-RDM when controlling for the self-reported intercept IS-RDM. Similarly, in the dimensional model including both Threat and Deprivation IS-RDMs, neither predictor showed a significant main effect. However, when modeling only the interaction between the Threat IS-RDM and the random intercept IS-RDM, a significant negative interaction emerged. This interaction, however, did not significantly improve model fit as determined by D1 model comparisons (F[1, 4, 15047] = 1.52, p = .271).

Table 6

Internalizing Symptom (Child-report) Dissimilarity: Pooled Adversity Model Results

	OA	OA*Int	Dimensional	Threat*Int	Dep*Int
Intercept	0.000 (0.007)	-0.002 (0.007)	0.000 (0.007)	-0.001 (0.007)	-0.001 (0.007)
	p = 1.000	p = 0.815	p = 1.000	p = 0.897	p = 0.945
Overall Adversity (OA)	-0.002 (0.022)	-0.002 (0.022)			
	p = 0.938	p = 0.938			
Threat			0.004 (0.026)	0.000 (0.025)	
			p = 0.899	p = 0.988	
Deprivation (Dep)			-0.010 (0.015)		-0.009 (0.015)
			p = 0.546		p = 0.566
Internalizing Random Intercept (Int)	0.443* (0.095)	0.441* (0.095)	0.443* (0.096)	0.442* (0.096)	0.441* (0.096)
	p = 0.010	p = 0.011	p = 0.011	p = 0.011	p = 0.011
Interaction		-0.026 (0.013)		-0.035* (0.013)	-0.006 (0.012)
		p = 0.076		p = 0.029	p = 0.613
R2	0.199	0.200	0.199	0.201	0.199
R2 Adj.	0.199	0.200	0.199	0.200	0.199
Num.Obs.	15051	15051	15051	15051	15051
Permuted Term	OA	OA*Int	Dep	Threat*Int	Dep*Int
Permuted p-value	0.801	0.001**	0.183	<.001***	0.468
Adjusted p-value	0.801	0.003**	0.305	<.001***	0.585

Note. Standardized estimates for regression models where outcome is child-reported internalizing slope dissimilarity are shown in each column. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation. Int = child-reported internalizing intercept.

3.4.3. Adversity and Externalizing Intercepts

All models predicting inter-individual differences in externalizing symptoms (Externalizing IS-RDM) showed significant effects of adversity constructions (see Table 7).

The OA IS-RDM significantly predicted the Externalizing IS-RDM indicating that participants with more dissimilar adversity histories also showed more dissimilar patterns of externalizing behaviors.

In the dimensional adversity model, both the Threat IS-RDM and the Deprivation IS-RDM showed significant main effects, suggesting that differences in both forms of adversity independently contribute to divergence in externalizing symptom profiles. The interaction model retained these main effects and also revealed a significant negative interaction between threat and deprivation dissimilarity. Figure 12 illustrates how greater similarity (i.e., less dissimilarity) in deprivation enhances the relationship between threat and externalizing intercept similarity; whereas greater dissimilarity (i.e., less similarity) in deprivation dampens that relationship. This interaction did significantly improve model fit compared to the dimensional model without the interaction ($F[1, 4, 15047] = 15.97$, $p = .016$).

The consistent significance across models and an adjusted R^2 of approximately .050–.051 suggest that adversity-related experiences are meaningfully linked to stable differences in externalizing symptoms during adolescence.

Table 7

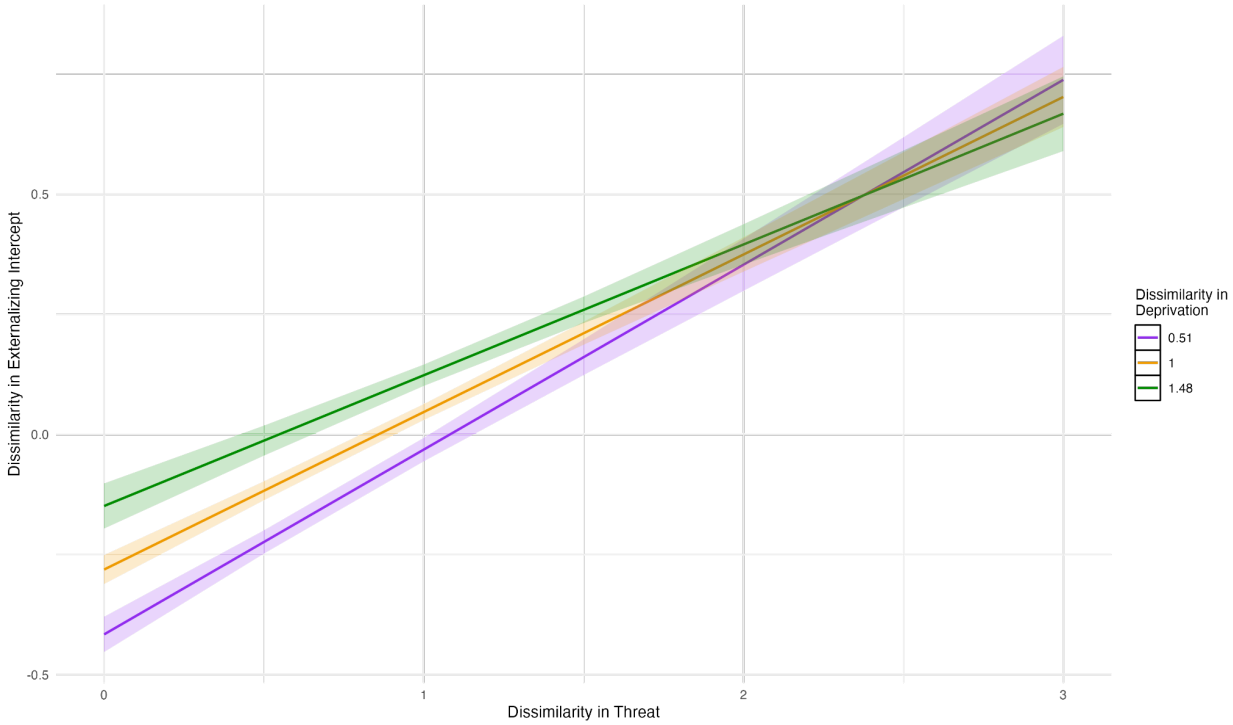
Externalizing Symptom Dissimilarity: Pooled Adversity Model Results

	OA	Dimensional	Threat*Dep
Intercept	0.000 (0.008)	0.000 (0.008)	0.007 (0.008)
	p = 1.000	p = 1.000	p = 0.384
Overall Adversity (OA)	0.225*** (0.012)		
	p = <0.001		
Threat		0.183*** (0.015)	0.184*** (0.015)
		p = <0.001	p = <0.001
Deprivation (Dep)		0.091*** (0.010)	0.092*** (0.010)
		p = <0.001	p = <0.001
Interaction			-0.025** (0.008)
			p = 0.002
R2	0.051	0.051	0.052
R2 Adj.	0.050	0.051	0.052
Num.Obs.	15051	15051	15051
Permuted Term	OA	Threat	Threat*Dep
Permuted p-value	<.001***	<.001***	0.002**
Adjusted p-value	<.001***	<.001***	0.002**

Note. Standardized estimates for regression models where outcome is externalizing intercept dissimilarity are shown in each column. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation.

Figure 12

Interaction between Threat and Deprivation IS-RDMs Relating to Externalizing IS-RDM



Note. With more similarity in deprivation (i.e., less dissimilarity), more similarity in threat predicts more similarity in externalizing intercepts.

3.4.4. Direct model relationships

I also conducted analyses with direct measurements to examine whether total childhood trauma exposure, as operationalized by CTQ total score, and its subdimensions (abuse and neglect subscale scores) predicted individual differences in mental health symptoms. In these models. In these models, participant slopes and intercepts from the internalizing and externalizing z-score composite variables were outcomes.

Total trauma exposure significantly predicted the random slope of the child-reported internalizing symptoms across time, β = 0.11, *SE* = 0.05, *t*(162.92) = 1.99, *p* = .04. However, D1 model tests found that the predictive value of trauma exposure was not significantly better than a model only accounting for child-reported internalizing intercepts. Neither abuse (β = 0.07, p =

.315) nor neglect (β = 0.06, p = .366) significantly predicted child-reported internalizing symptom trajectories.

Total trauma exposure was significantly associated with parent-reported internalizing symptom intercepts, β = 0.20, SE = 0.08, t(151.77) = 2.60, p = .010, and the model explained a small amount of variance (R^2 = .04). In the disaggregated model, abuse (β = 0.06, p = .537) and neglect (β = 0.16, p = .071) were not significant predictors.

Finally, total trauma exposure was a robust predictor of greater externalizing symptoms intercepts, β = 0.30, SE = 0.07, t(164.59) = 4.11, p < .001, with more explained variance (adj. R^2 = .09). In the disaggregated model, abuse significantly predicted externalizing symptom severity (β = 0.25, p = .005), whereas neglect was not a significant predictor (β = 0.11, p = .211).

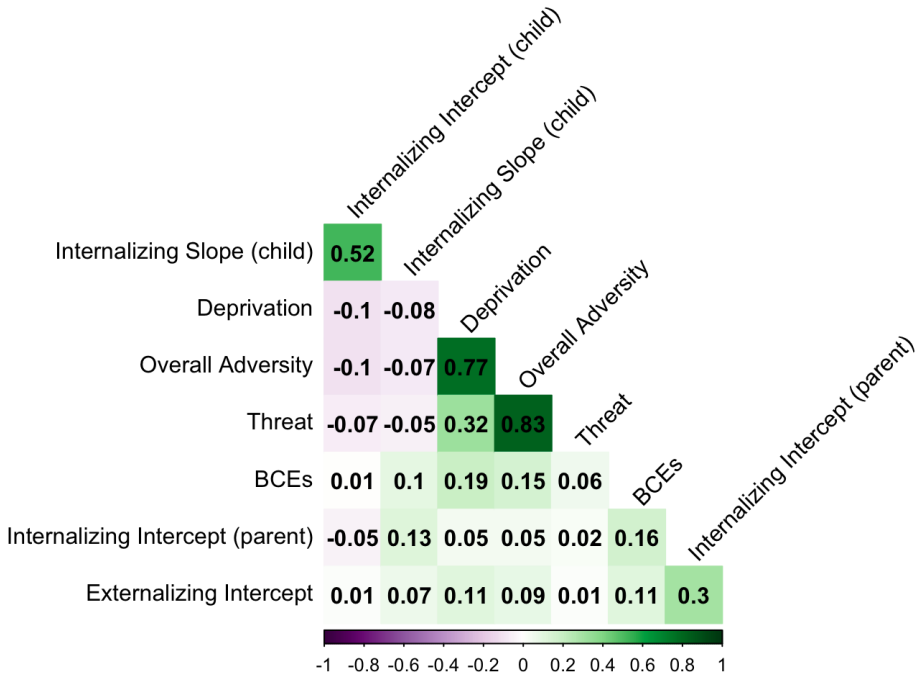
3.5. Study 2 Results: Positive Experiences and Mental Health

3.5.1. Associations among Study 2 IS-RDMs

Figure 13 visualizes the correlations for the IS-RDMs for the subsample in Study 2 (n = 110). Out of all IS-RDMs representing childhood experiences, the BCEs IS-RDM has the highest associations with every mental health IS-RDM of interest (i.e., the Internalizing (C) IS-RDM, Internalizing (P) IS-RDM, and Externalizing IS-RDM).

Figure 13

IS-RDM Pooled Correlations



Note. These correlations are between the mental health, adversity, and positive experience IS-RDMs. Correlation coefficients were pooled across imputed data sets.

3.5.2. Parent-reported Internalizing Models

Dissimilarity in positive experiences (BCEs IS-RDM) predicted dissimilarity in random intercepts of parental reports of internalizing symptoms (Internalizing Intercept (P) IS-RDM) across all models (estimates ranged from 0.161 to 0.162), controlling for childhood adversity dissimilarity. In contrast, neither the dimensional nor overall adversity models showed a significant main effect of childhood adversity dissimilarity on the Internalizing Intercept (P) IS-RDM when controlling for the BCEs IS-RDM. Full model results are presented in Table 8. No interactions were shown between adverse and positive experience IS-RDMs.

Table 8

Internalizing Symptom (Parent-report) Dissimilarity: Pooled BCEs Model Results

	OA	OA*BCEs	Dimensional	Threat*BCEs	Dep*BCEs
Intercept	0.000 (0.014)	0.001 (0.014)	0.000 (0.014)	-0.001 (0.014)	0.006 (0.014)
	p = 1.000	p = 0.966	p = 1.000	p = 0.941	p = 0.677
BCEs	0.161*** (0.019)	0.161*** (0.019)	0.160*** (0.018)	0.161*** (0.019)	0.162*** (0.018)
	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001
Overall Adversity (OA)	0.020 (0.016)	0.021 (0.016)			
	p = 0.202	p = 0.203			
Threat			0.005 (0.023)	0.003 (0.023)	0.009 (0.023)
			p = 0.818	p = 0.901	p = 0.716
Deprivation (Dep)			0.017 (0.019)	0.015 (0.019)	0.017 (0.019)
			p = 0.383	p = 0.424	p = 0.364
Interaction		-0.004 (0.015)		0.018 (0.014)	-0.031 (0.017)
		p = 0.794		p = 0.214	p = 0.076
R2	0.027	0.027	0.027	0.028	0.029
R2 Adj.	0.027	0.027	0.027	0.027	0.028
Num.Obs.	4950	4950	4950	4950	4950
Permuted Term	BCEs	OA*BCEs	BCEs	Threat*BCEs	Dep*BCEs
Permuted p-value	<.001***	0.589	<.001***	0.219	0.026*
Adjusted p-value	<.001***	0.589	<.001***	0.273	0.044*

Note. Standardized estimates for regression models where outcome is parent-reported internalizing intercept dissimilarity are shown in each column. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation.

3.5.3. Child-reported Internalizing Models

The BCEs IS-RDM also predicted dissimilarity in random slopes of child reports of

internalizing symptoms (Internalizing Slopes (C) IS-RDM) across all models (estimates ranged from 0.102 to 0.107), controlling for both childhood adversity dissimilarity and random intercept dissimilarity of child reports (Internalizing Intercepts (C) IS-RDM). Models indicated a very small significant main effect of childhood adversity dissimilarity on the Internalizing Intercept (P) IS-RDM when controlling for the Internalizing Intercepts (C) IS-RDM and the BCEs IS-RDM. In the dimensional model, the Deprivation IS-RDM similarly showed a small effect. There were no significant interactions between adversity dissimilarity and positive experiences dissimilarity in any model. Full model results are presented in Table 9.

Table 9

Internalizing Symptom (Child-report) Dissimilarity: Pooled BCEs Model Results

	OA	OA*BCEs	Dimensional	Threat*BCEs	Dep*BCEs
Intercept	0.000 (0.012)	0.000 (0.012)	0.000 (0.012)	0.000 (0.012)	0.000 (0.012)
	p = 1.000	p = 0.981	p = 1.000	p = 0.971	p = 0.976
BCEs	0.102*** (0.019)	0.102*** (0.019)	0.105*** (0.020)	0.105*** (0.020)	0.105*** (0.020)
	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001
Overall Adversity (OA)	-0.034* (0.016)	-0.034* (0.016)			
	p = 0.043	p = 0.043			
Threat			-0.010 (0.019)	-0.011 (0.020)	-0.010 (0.020)
			p = 0.613	p = 0.589	p = 0.622
Deprivation (Dep)			-0.041* (0.019)	-0.042* (0.018)	-0.041* (0.019)
			p = 0.043	p = 0.035	p = 0.043
Internalizing Intercept	0.516** (0.099)	0.516** (0.099)	0.514** (0.100)	0.514** (0.099)	0.514** (0.100)
	p = 0.007	p = 0.007	p = 0.007	p = 0.007	p = 0.007
Interaction		0.002 (0.012)		0.007 (0.012)	-0.002 (0.012)
		p = 0.873		p = 0.548	p = 0.875
R2	0.286	0.286	0.287	0.287	0.287
R2 Adj.	0.285	0.285	0.286	0.286	0.286
Num.Obs.	4950	4950	4950	4950	4950
Permuted Term	BCEs	OA*BCEs	BCEs	Threat*BCEs	Dep*BCEs
Permuted p-value	<.001***	0.873	<.001***	0.537	0.870
Adjusted p-value	<.001***	0.873	<.001***	0.873	0.873

Note. Standardized estimates for regression models where outcome is child-reported internalizing slope dissimilarity are shown in each column. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation. Int = child-reported internalizing intercept.

3.5.4. Externalizing Models

The full results of the externalizing models are presented in Table 10. In all models predicting dissimilarity in externalizing intercepts (Externalizing IS-RDM), the BCEs IS-RDM exerted a main effect (estimates ranged from 0.090 to 0.100) while controlling for adversity dissimilarity.

Table 10

Externalizing Symptom Dissimilarity: Pooled BCEs Model Results

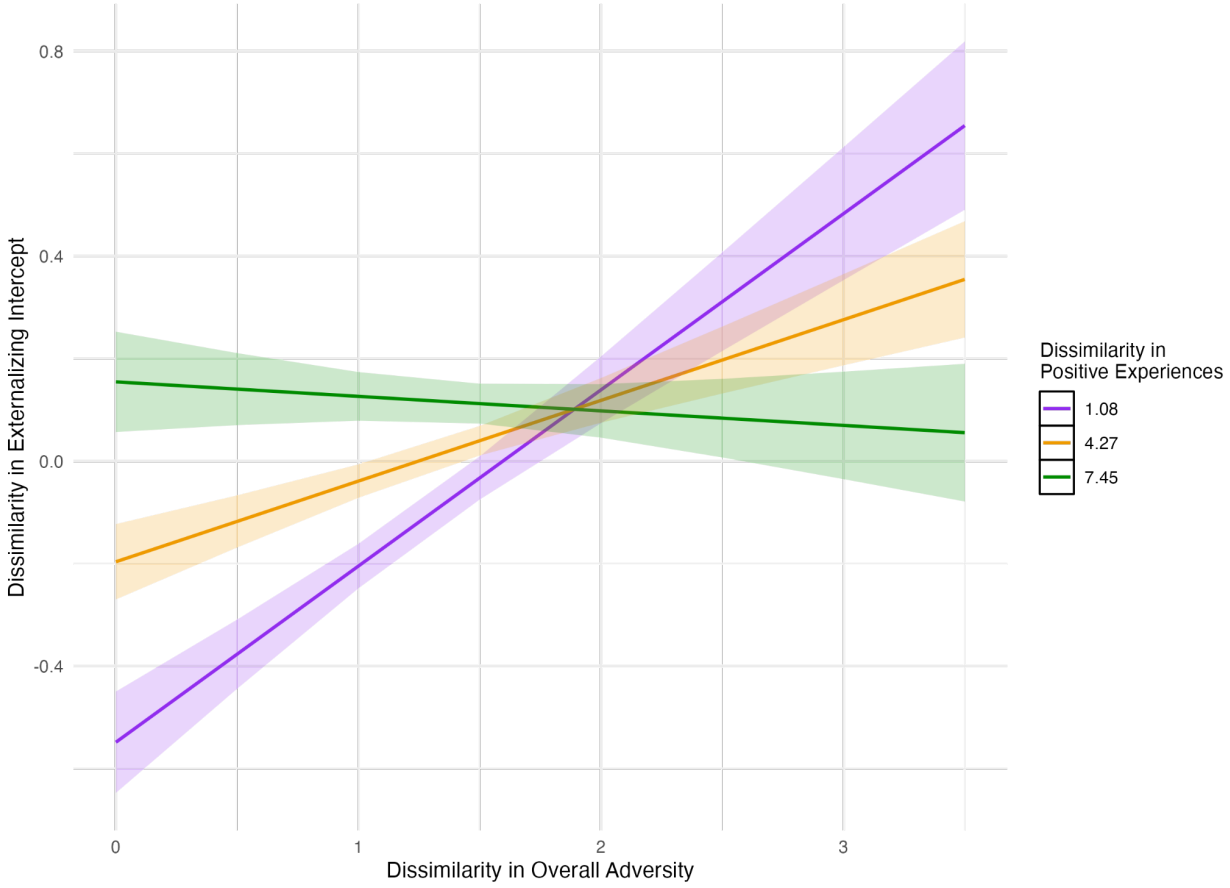
	OA	OA*BCEs	Dimensional	Threat*BCEs	Dep*BCEs
Intercept	0.000 (0.014)	0.016 (0.014)	0.000 (0.014)	0.003 (0.014)	0.021 (0.014)
	p = 1.000	p = 0.267	p = 1.000	p = 0.824	p = 0.152
BCEs	0.100*** (0.018)	0.101*** (0.018)	0.093*** (0.019)	0.089*** (0.018)	0.098*** (0.019)
	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001
Overall Adversity (OA)	0.070*** (0.019)	0.087*** (0.019)			
	p = <0.001	p = <0.001			
Threat			-0.025 (0.024)	-0.018 (0.025)	-0.014 (0.025)
			p = 0.330	p = 0.491	p = 0.601
Deprivation (Dep)			0.105*** (0.019)	0.110*** (0.019)	0.108*** (0.019)
			p = <0.001	p = <0.001	p = <0.001
Interaction		-0.103*** (0.016)		-0.053*** (0.014)	-0.108*** (0.018)
		p = <0.001		p = <0.001	p = <0.001
R2	0.017	0.031	0.022	0.026	0.035
R2 Adj.	0.017	0.030	0.022	0.025	0.035
Num.Obs.	4950	4950	4950	4950	4950
Permuted Term	BCEs	OA*BCEs	BCEs	Threat*BCEs	Dep*BCEs
Permuted p-value	<.001***	<.001***	<.001***	<.001***	<.001***
Adjusted p-value	<.001***	<.001***	<.001***	<.001***	<.001***

Note. Standardized estimates for regression models where outcome is externalizing intercept dissimilarity are shown in each column. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. Dep = deprivation.

The overall adversity IS-RDM also exerted a significant main effect on the Externalizing IS-RDM while controlling for the BCEs IS-RDM. Additionally, there was a significant negative interaction between the overall adversity IS-RDM and the BCEs IS-RDM, such that dissimilarity in positive experiences neutralized the association between dissimilarity in overall adversity and externalizing symptoms (see Figure 14).

Figure 14

Interaction between OA and BCEs IS-RDMs Relating to the Externalizing IS-RDM



Note. The relationship between overall adversity and externalizing intercepts is dependent on dissimilarity in positive experiences.

The Deprivation IS-RDM exerted a significant main effect on the Externalizing IS-RDM while controlling for the BCEs and Threat IS-RDMs, and it significantly interacted with the BCEs IS-RDM in predicting the Externalizing IS-RDM. Although the Threat IS-RDM did not exert a significant main effect when controlling for the BCEs IS-RDM and Deprivation IS-RDM, it did significantly interact with the BCEs IS-RDM in predicting the Externalizing IS-RDM.

D1 model comparison tests indicated that adding interactions to the externalizing models provided a significantly better fit to the data than the corresponding models without interaction terms for each the OA IS-RDM (F[1, 4, 4946] = 41.41, p = .003), the Deprivation IS-RDM (F[1,

4, 4945] = 35.21, p = .004), and the Threat IS-RDM (F[1, 4, 4945] = 14.06, p = .020).

3.5.5. Direct model relationships

Follow-up analyses with direct estimates examined whether total scores on the BCEs questionnaire predicted random intercepts and slopes of internalizing and externalizing symptoms. Greater total BCEs and PCEs were significantly associated with flatter child-reported internalizing slopes (β = –0.25, SE = 0.07, t[68.75] = –3.56, p < .001) when controlling for child-reported intercepts. Adding BCEs total score to the child-reported intercept model increased the adjusted r-squared by .055, and D1 model comparisons indicated an improvement in model fit (F[1, 4, 970] = 12.68, p = .024).

Higher BCEs and PCEs were also significantly associated with lower parent-reported internalizing intercepts (β = –0.35, SE = 0.10, t[88.07] = –3.63, p < .001, adj. R^2 = .114) and with lower externalizing symptom intercepts (β = –0.30, SE = 0.10, t[92.41] = –3.07, p = .003, adj. R^2 = .080).

4. Discussion

These studies examined how intersubject dissimilarity in adversity and positive experiences relates to the stable differences of internalizing and externalizing symptoms, as well as differences in trajectories of internalizing symptoms, across adolescence. By leveraging both child- and parent-reported outcomes, I aimed to understand whether youth with more similar psychosocial environments also show more similar patterns of mental health across time.

4.1. Effects of the Psychosocial Environment

Across both studies, adversity was more consistently associated with externalizing

symptoms compared to internalizing symptoms. Associations with parent-reported internalizing intercepts were minimal, suggesting that while individual differences in adversity may be weakly linked to parent-reported internalizing symptoms, other factors likely play a more substantial role. No associations were found between adversity and child-reported internalizing slopes in models that controlled for child-reported intercepts. Overall adversity (OA) was, however, associated with externalizing intercepts; higher dissimilarity in adversity corresponded with greater dissimilarity in externalizing symptoms. There was also a modest interaction between adversity dimensions where similarity in deprivation appeared to strengthen the relationship between threat and externalizing dissimilarity, and dissimilarity in deprivation appeared to weaken that relationship.

Although an association between adversity and internalizing symptoms was expected, several factors could explain its absence in this sample. Notably, the COVID-19 pandemic began during Wave 3 of this longitudinal study and has been shown to disproportionately affect adolescent girls' mental health (Benton et al., 2021). While this sample was not recruited to represent a clinical population, the average CES-DC score across assessments was 16.7, indicating elevated depression symptoms in this sample. For context, researchers have used thresholds of 16 and 21 on the CES-D to indicate high symptom levels or potential depression diagnoses (Shean & Baldwin, 2008). The pandemic may have acted as a shared environmental stressor, potentially obscuring the effects of adversity-related individual differences on internalizing symptoms. This is consistent with findings by Rakesh and colleagues (2025), who reported an unexpected reversal in the association between SES and internalizing symptoms during the pandemic, with higher SES predicting greater symptom increases. It is also possible that, in this sample, externalizing symptoms reflect more intense distress and are therefore more

closely aligned with experiential adversity than internalizing symptoms.

Another interpretation is that there simply is no strong relationship between individual differences of internalizing symptoms and adversity history. One study using a community sample found that ACEs were most strongly associated with violence and suicidality (Meeker et al., 2021). Though suicidality was not included in the mental health outcomes analyzed here, these findings do somewhat align with the observed stronger associations for externalizing differences. Additionally, Miller and colleagues (Miller et al., 2018a) identified that, while threat experienced before age six did predict internalizing and externalizing problems at age 17, deprivation only predicted externalizing problems, which are partially consistent with these results. Notably, their measurement of threat was limited to early childhood and incorporated data gained from observed parent-child interactions in the home and reports of harshness or abuse in interviews with mothers. This approach differs substantially from this study and may offer a more nuanced assessment of how early threat contributes to the development of internalizing symptoms.

In striking contrast to adversity, dissimilarity in positive experiences predicted greater differences in all youth mental health outcomes examined, including child-reported internalizing slopes, parent-reported internalizing intercepts, and externalizing intercepts. This is especially significant given that adversity is traditionally emphasized as a dominant predictor of youth mental health. That positive experiences demonstrated equal or greater associations across all outcomes suggests that they may play just as critical, if not more so, a role in the mental health of some adolescent populations.

Interactions between adversity and positive experiences emerged in externalizing models, indicating both additive and interactive effects. Specifically, differences in adversity were no

longer associated with differences in externalizing symptoms when adolescents also differed in their level of positive experiences. This highlights a possible overriding influence of positive environments in the context of a community sample.

Examining dimensional effects of adversity also yielded some useful insights. Interactions between threat and deprivation showed that similarity in both constructs increased the similarity in externalizing symptoms. Conversely, dissimilarity in measures of deprivation (including proximal measurements of both material and psychosocial components), weakened the relationship between dissimilarity in threat and externalizing symptoms. This finding echoes research on how low SES, likely due to reduced access to fewer resources, or neglect can obscure the direct impact of traumatic experiences on mental health outcomes.

4.2. Methodological Considerations

Although adversity was measured earlier in development and thus might be expected to have a stronger relationship to proximal mental health symptom endorsements and changes, cumulative positive experiences, which were assessed after longitudinal mental health symptoms, showed equal or stronger associations with mental health. This finding may underscore their potential promotive or protective role, though the lack of measurement specificity in timing of those positive experiences provides other possible interpretations (see Limitations section for further discussion of timing differences).

An interesting pattern emerged when comparing informant reports: parent-reported internalizing intercepts were not strongly related to child-reported intercepts, but were mildly related to child-reported slopes over time. This suggests that while caregivers may not align with early adolescents' self-report of internalizing symptoms, parents' perceptions do correspond with how symptoms evolve over time. Child-reported early internalizing symptoms were strongly

associated with their own slopes, reinforcing the notion that these earlier self-perceptions are more predictive of developmental trajectories than caregiver perceptions. The data also did not indicate much variation in change in parental reports over time, measured here through the CBCL, whereas the composite score representing child internalizing problems did produce variation in trajectories. Discrepancies between parent and child reported internalizing problems are common in the literature (Weissman et al., 1980), with adolescents often self-reporting higher symptoms (Sourander et al., 1999). Given the internal nature of these symptoms, it is likely that self-reports more accurately capture the adolescent psychological experience, particularly when they reflect meaningful change over time beyond age-related effects.

Although the use of intersubject representational similarity analysis (IS-RSA) provided a novel framework for examining individual differences in adversity and positive experiences, this second-order modeling approach produced smaller effects compared to traditional direct measurement models. Specifically, models using summary scores (e.g., CTQ total or total positive experiences) as predictors yielded stronger effect sizes and better model fit than their IS-RDM counterparts. This may suggest that cumulative or total scores more effectively capture overall risk and protective influences on adolescent mental health compared to multivariate similarity patterns. Alternatively, it may reflect a methodological limitation of this IS-RSA application—namely, that representing mental health outcomes as differences along a single composite score can obscure meaningful variation across symptom domains, reducing sensitivity.

The positive experiences (BCEs) IS-RDM was almost completely driven by differences in total scores, as it was composed of fewer distinct measurements compared to the adversity IS-RMDs, and the total score was not transformed to match the scale of other features. Follow-up

analyses that transformed the total score (e.g., dividing by the number of items to constrain its range to 0–1) produced similar results to the reported results, suggesting robustness but also reinforcing that this IS-RDM closely mirrored a direct total score comparison.

Additional work could further evaluate when and how intersubject similarity models offer added value beyond more traditional, variable-centered approaches in these domains. IS-RSA offers unique insights that complement variable-centered approaches. Population-level associations do not always map cleanly onto inter-individual variation, and IS-RSA allows for a flexible examination of how shared or divergent experiences may shape developmental outcomes. Its utility may be particularly strong for capturing nuanced pairwise differences in exposure patterns (e.g., chronicity, timing, intensity) and for modeling complex, multivariate outcomes (e.g., neural development, symptom profiles). Future studies could refine how experiential similarity is operationalized, identify which constructs are best suited to intersubject modeling, and clarify the conditions under which IS-RSA adds value. Combining IS-RSA with idiographic or intraindividual approaches (e.g., dynamic structural equation modeling) may offer a more comprehensive framework for understanding how individual-level processes unfold and contribute to interpersonal differences across key developmental periods.

4.3. Theoretical and Clinical Implications

Overall, the findings support a differentiated view of how life experiences correspond to youth psychopathology. Here, externalizing symptoms appear more closely tied to both positive and negative experiences, while internalizing symptoms appear to be only tied to positive experiences. A dimensional adversity framework—particularly the distinction between threat and deprivation—adds nuance to how these factors interact to predict externalizing symptoms.

Most importantly, the present findings highlight the independent role of positive

experiences—not only in relation to reduced mental health symptoms, but also in shaping the association between adversity and mental health outcomes. This has significant implications for prevention and intervention strategies, particularly within general population settings where adversity may be present but not severe, and where opportunities for positive experiences may vary. For instance, when supporting an adolescent who is experiencing emotional distress and school-based bullying, it may be more effective to prioritize the facilitation of positive experiences (e.g., fostering supportive interpersonal relationships) rather than focusing solely on the reduction of stressor exposure. Although such decisions must be made with consideration of safety, individual preferences, and contextual factors, these findings reinforce the idea that enhancing access to positive experiences may serve as a critical target for intervention.

Many evidence-based therapeutic approaches do include components that can encourage exposure to positive experiences, such as behavioral activation within cognitive behavioral therapy, ABC PLEASE within dialectical behavior therapy, or value-based action in acceptance and commitment therapy. These elements are typically framed as short-term behaviors designed to improve mood or support emotion regulation, and are rarely presented as developmentally grounded targets informed by adolescent-specific research. In the present study, the positive experiences most strongly associated with individual differences included family- and community-centered experiences (e.g., receiving emotional support during stress, participating in community traditions, and having predictable routines), as well as a sense of comfort with oneself. In contrast, opportunities for fun—which are frequently emphasized in such therapeutic strategies—were least associated with overall differences in positive experience profiles. These findings suggest that while engaging in enjoyable activities remains a well-researched, valuable therapeutic tool, a broader focus that also incorporates socially grounded, developmentally

salient experiences may also provide important benefits for adolescent mental health. While many interventions for younger children often emphasize the importance of family- and community-based support, this focus is less commonly extended to adolescents, though other research suggests the continued importance of these factors for adolescents with a history of maltreatment (Cheung et al., 2017). Future experimental work, both in community and clinical populations, is needed to inform the development of targeted, developmentally-informed prevention and intervention strategies.

4.4. Limitations

Several limitations of this study warrant consideration. Temporal differences in the measurement of adversity and positive experiences complicate interpretation. Adversity measures were assessed earlier in development compared to mental health trajectories, while positive experiences were assessed later. Some positive experiences may have co-occurred with or followed mental health outcomes, and this confounds inferences about the relative influence of how positive experiences might coincide with adverse experiences. Due to the study design, the data cannot reveal whether differences in positive experiences were established before, alongside, or after mental health divergence. Perhaps, and very possibly, differences emerged throughout each of those periods in dynamic, bi-directional relationships.

As stated earlier, participants in the study's community sample were not selected for elevated adversity or psychopathology, limiting generalizability to higher clinical or risk-exposed populations. Effects of adversity—and especially the interactive or buffering role of positive experiences—may manifest differently in populations with greater exposure to threat, deprivation, and, not measured here, unpredictability. Future studies in high-adversity samples are needed to extend these findings.

This sample was not large enough to examine these specific relationships among different social minority identities, such as racial and ethnic identities or sexual and gender minority identities. This is a major limitation to the generalizability of this study, especially given that socially and economically marginalized youth face heightened rates of adverse exposures. For instance, Black families in the U.S. experience even greater material hardship, which is related to youth behavioral challenges (Ibekwe-Okafor et al., 2023), and Native American children face disproportionately greater exposures across a broad array of adversities (T. M. Jensen et al., 2024). Additionally, legislation negatively targeting LGBTQ+ populations in the U.S., particularly that targeting transgender and gender-diverse people, is increasing exponentially (Movement Advancement Project, 2023) at a time when younger generations are identifying as sexual and gender minorities at higher rates than ever (Twenge et al., 2024). These chilling sociopolitical and economic conditions compound developmental risk and underscore the urgency for focused research that can support the well-being these populations.

Importantly, communities facing systematic oppression also demonstrate incredible resilience (Linklater, 2020), and the acknowledgement of collective and individual strengths are equally important to the acknowledgement of historically-compounded inequities (Linklater, 2020; Mueller et al., 2021). Future work should also continue to examine how resilience is fostered within cultural contexts while attuning to the influence of life experiences, and advocate for policies and interventions that support those facing multiple, intersecting forms of oppression. This line of scientific inquiry has developed across many fields, and the fields of adversity and resilience science can be strengthened by highlighting and integrating existing frameworks that have examined individual and community healing, including liberation psychology, Black psychology, queer psychology, and intersectional theory (Freeman-Coppadge

& Langroudi, 2021; French et al., 2020; Hudson & Romanelli, 2020; McNeal et al., 2024; Parmenter et al., 2024).

4.5 Additional future directions

In addition to future areas of scientific research that have already been proposed, these findings can be better characterized by elucidating the mechanisms linking similarity in life experiences to similarity in mental health trajectories. Study designs that allow for the examination into possible mediators such as self-regulation, social support, identity development, or stress physiology may help explain why certain experiential patterns cluster with particular symptom trajectories. Integrating neural or cognitive data into intersubject models could also advance efforts to map individual experience to brain development and behavior over time

5. Conclusion

Taken together, this study highlights the complex, multifaceted ways in which adolescents' early life experiences relate to their mental health trajectories. By leveraging an intersubject dissimilarity approach, we found that both adversity and positive experiences contribute meaningfully—but differently—to individual variation in internalizing and externalizing symptoms. While adversity was more consistently linked to externalizing outcomes, positive experiences emerged as robust predictors across domains. These findings suggest that fostering positive environments may be an especially powerful and underutilized tool for promoting youth mental health. Future research attending to both risk and resilience processes will be crucial in advancing personalized approaches to youth mental health.

CHAPTER III: ADVERSE EXPERIENCE & BRAIN ASSOCIATIONS

This work, entitled Comparing the Multivariate Relationships of Conceptual Adversity Models and Structural Brain Development in Adolescent Girls: A Registered Report, was co-authored with T. Cheng, J. Flannery, K. Mills, P. Fisher, C. McCann, and J. Pfeifer. I was the lead author of this publication and responsible for the writing of the manuscript, and edits provided by co-authors. I led the study design and analysis with input and assistance from co-authors.

1. Introduction

The lifetime burden of childhood adversity is rooted in its pronounced impact on human development and well-being, often to the detriment of one's long-term physical and psychological health. Most children and adolescents in the United States experience at least one form of adversity, and researchers estimate that adversity contributes to a third of all psychiatric disorders (Green et al., 2010). Although the definition of childhood adversity has greatly varied across time and research groups which typically draw upon elements of poverty, maltreatment, trauma, and toxic stress (Brooks-Gunn et al., 1987; Perry et al., 1995; Shonkoff et al., 2012), this study will use the definition provided by McLaughlin (2016), which purports that adverse experiences are violations of the expectable environment that may (but not necessarily) result in chronic or acute stress responses.

While the field has made significant advancements in understanding the long-term effects of childhood adversity on mental and physical health (Berens et al., 2017; Gunnar et al., 2006; Turner & Lloyd, 1995), the precise neurodevelopmental mechanisms through which different types of adversity "get under the skin" remain an open question. A preponderance of evidence

suggests that neural development is a crucial mechanism through which experience is biologically embedded. Still, the specific neural mechanisms likely vary by several situational elements, including what is experienced and when (Nelson & Gabard-Durnam, 2020). Some of the neurodevelopmental mechanisms of adversity may be adaptive, supporting the ability of children and adolescents to calibrate their functioning and behavior toward adverse environments (Ellis, Abrams, et al., 2022). Understanding how childhood adversity influences the brain will influence how adversity is measured and how it might best be clinically addressed to support positive development.

1.1. Conceptual Models of Adversity

Historically, researchers have measured the impact of individual experiences on development (e.g., the effect of physical abuse on psychopathology or financial instability on cognitive development). This method provided foundational evidence within the field that implicates childhood adversity in altered physical and mental health outcomes (Bremner et al., 1997; Chase-Lansdale et al., 1995; Mullen et al., 1993; Springer et al., 2007). Yet, this design does not inherently account for either the comorbidity of adverse experiences or the shared mechanisms that stem from similar experiences. To increase ecological validity and account for potentially shared developmental risks, researchers have proposed conceptual models of adversity built upon the multitude of experiences in one's life that could impact development, including the social safety theory (Slavich et al., 2019) and the adaptive calibration model (Del Giudice et al., 2011), among others. This study will investigate two primary models of adversity: the cumulative risk and dimensional models.

The cumulative risk model, famously linked to the landmark Adverse Childhood Experiences study that reported accumulating adverse experiences incrementally increase the

risk for disease (Felitti et al., 1998), assumes that all adverse experiences exert an accruing effect on development processes (Evans et al., 2013). Researchers using this model often summarize adverse experiences to operationalize cumulative risk (Evans et al., 2013; Felitti et al., 1998), though others have critiqued this operationalization since it fails to account for other essential features, such as severity or chronicity of experience (Smith and Pollak, 2020). Accrued adverse experiences are theorized to activate a global stress response and result in greater allostatic load, imparting physical and psychological consequences (Evans et al., 2013). By creating a clear link between the number of adverse experiences and the risk for disease and mental illness, this model has been beneficial for creating screening tools for intervention delivery that can be used in pediatric visits (Koita et al., 2018). The proposed global stress mechanism, however, may not capture the effect of some adversities that might not activate a stress response (e.g., impoverished language exposure; Nelson & Gabard-Durnam, 2020). Furthermore, many studies suggest that all experiences are not necessarily equal in their effect; instead, their influence on development may depend on environmental variables other than accumulation alone, like the type or timing of an event (Bick & Nelson, 2016; Edmiston et al., 2011; Fox et al., 2010).

Dimensional models of adversity assert that particular features of experience are linked with specific developmental processes and consequentially exert a unique influence on neurobiology (McLaughlin, Sheridan, & Lambert, 2014). One such model, the Dimensional Model of Adversity and Psychopathology (DMAP), posits that threat experiences may exert a greater impact on socio-emotional development. In contrast, deprivation may have a greater influence on cognitive development. Specific adverse experiences, such as poverty, do not necessarily convey a presence of either threat or deprivation. Still, some evidence suggests that

in the United States, poverty tends to track with outcomes associated with deprivation (Sheridan et al., 2020).

However, whether these dimensions are natural kinds of discretely modifying one's biology is still a subject of scientific debate (Smith & Pollak, 2020). In Smith and Pollak's critique, the authors question whether today's social and political categorizations of adversity match biologically meaningful distinctions and, moreover, whether any single experience can be cleanly assigned to either a threat or deprivation category. For example, exposure to something threatening (e.g., physical abuse from a parent) also tends to inherently contain aspects that could be classified as deprivation (e.g., lack of sensitive caregiving). Differences in how threat and deprivation are operationally defined across studies highlight the challenge of classifying experiences accordingly. Additionally, the co-occurrence of different types of adversities and the potential for co-occurrence to create its unique impact create further statistical and interpretational challenges when unaddressed in modeling strategies.

Despite such complications presented by attempting to break down complex experiences, recent studies using this framework provide various levels of support for a dimensional approach when evaluating outcomes related to brain function (Sheridan et al., 2017; Young et al., 2022) and connectivity (Chahal et al., 2022; Cheng et al., 2021; Goetschius et al., 2020) as well as behavioral outcomes such as emotion regulation, cognitive control, and psychopathology (Lambert et al., 2017; Miller et al., 2018a; Sheridan et al., 2017, 2020; Young et al., 2022). Reviews of studies that solely examined one dimension or another suggest that threat and deprivation are each uniquely associated with brain structure (Colich et al., 2020; McLaughlin et al., 2019). Still, many of these studies do not provide unequivocal support for DMAP, and others have suggested that a single adversity factor can best explain variance in later brain (Gehred et

al., 2021) and behavioral outcomes (Bignardi et al., 2022; Carozza et al., 2022). While there are undoubtedly other salient dimensions of adversity that track with health and behavioral outcomes (e.g., environmental unpredictability; Brumbach et al., 2009; Doom et al., 2016), given the depth of literature on the specific neurophysiological pathways for threat and deprivation, we will focus on characterizing the relative advantages of the DMAP and cumulative risk model with respect to how they relate to structural brain trajectories.

1.2. Adversity and Neurodevelopment

A recent systematic review of adversity and neural development suggests that the DMAP (compared to an undifferentiated approach) may better explain some structural brain differences between individuals who have experienced childhood adversity and those who have not (McLaughlin et al., 2019). The authors report that threat exposures and deprivation exposures have been associated with differences in regional thickness or volume within unique neural networks. Across the reviewed studies, threat exposures tend to be more closely associated with the fronto-amygdala and hippocampal circuit structure, and deprivation exposures tend to be more closely associated with the frontoparietal network structure. Specifically, they found a majority of studies that identified a negative association between threat and amygdala volume (whereas deprivation and amygdala volume findings were mixed) and threat and medial prefrontal cortical volume or thickness (whereas the majority of studies identified a null association with deprivation and medial prefrontal cortical structure). Similarly, most studies identified negative associations between threat and hippocampal volume, while most identified a null association between deprivation and hippocampal volume. Conversely, across reviewed studies, deprivation was unanimously associated with reductions in thickness or volume in the

dorsolateral prefrontal cortex and reductions in thickness in the superior parietal cortex (whereas threat was not consistently associated with differences in these regions).

This review suggests that inconsistent brain-adversity associations in the literature are partly driven by the failure to differentiate between the unique influence of these dimensions (McLaughlin et al., 2019). Subcortical differences (particularly in the hippocampus and amygdala), for example, have been extensively studied between adversity-exposed and unexposed individuals, yielding mixed results. A 2017 meta-analysis found a small effect for decreased hippocampal volume but no amygdala differences in non-clinical adults who had been exposed to adversity in childhood (Calem et al., 2017). Yet, hippocampus and amygdala volume in childhood and adolescence appear to be consistently associated with threat exposure (McLaughlin et al., 2019). An additional explanation of these inconsistent subcortical effects incorporates developmental timing. One review has suggested that both the timing of exposure and timing of measurement may influence observed associations, noting that the expression of the effect of adversity could vary by age and emphasizing the importance of examining sensitive periods of development (Tottenham & Sheridan, 2010).

1.3. Adversity and Adolescence

Research on adolescence—a time of considerable structural brain development and a possible sensitive period (Fuhrmann et al., 2015)—could provide insight into the development of neural processes relevant to widely studied outcomes of adversity, including the onset of psychopathology. Widespread cortical thinning during adolescence is instrumental in improving cognitive and socioemotional processing (Dahl et al., 2018; Fuhrmann et al., 2015; Mills et al., 2016; Tamnes et al., 2017). Though other neural metrics, such as surface area, have been associated with adverse exposures in cross-sectional samples, cortical thinning has been shown

to account for the bulk of volumetric change in the brain during adolescence and remains a crucial measurement of interest in characterizing rates of brain development. For instance, accelerated brain development could be measured through accelerated cortical thinning during adolescence or by markers of early deceleration, indicated by an earlier plateau of cortical thickness. The stress acceleration hypothesis, developed to explain the accelerated development of neural circuits in response to a lack of sensitive parenting in early childhood, suggests that accelerated development is an adaptive response that encourages adult-like functioning in stressful environments (Callaghan & Tottenham, 2016). However, while protective in certain contexts, this acceleration may also lead to negative psychological effects, such as poor emotional regulation in adulthood (Herzberg & Gunnar, 2020).

While substantial evidence has identified early life (often operationalized as ages 0-3 years) as a period of brain development that is particularly sensitive to adversity (Bick & Nelson, 2016; Hambrick et al., 2019; Heim et al., 2019; Pollak, 2005; Shonkoff & Garner, 2012), research suggests that adolescence may be a developmental period of enhanced opportunity for resilience (Bonnie & Backes, 2019; Malhi et al., 2019; Masten et al., 1999). The rapid biological growth and ecological change that characterizes adolescence provide a foundation for higher-level cognitive, motivational, and social processing (Dahl et al., 2018; Fuhrmann et al., 2015). Programs that promote positive parenting of adolescents and increase knowledge about stress and its reduction provide promising examples of ameliorating maladaptive development through intervention (Brody et al., 2017; Lupien et al., 2013).

However, this developmental period also conveys its unique risks. The chronic continuation of early adversity or experience of adversities specific to adolescence can impede the opportunity for resilience (Ganzel et al., 2013; Romeo, 2017; Shiner et al., 2017). The

adolescent brain is uniquely tuned towards social stimulation and identity exploration, and there is reason to believe that experiences that derail those developmental goals (e.g., bullying or lower perceived status) could impart their own neurophysiological risk (Rudolph et al., 2021; Tzanoulinou & Sandi, 2017). Certain types of adversity (e.g., sexual assault) also occur at higher rates in adolescence (Gewirtz-Meydan & Finkelhor, 2020). In this period, the onset and rates of mental health disorders - including anxiety, eating, substance use, and mood disorders - also increase, which may be related to endemic biological and behavioral changes (Byrne et al., 2017; Costello et al., 2011; Kessler et al., 2005; Phillips et al., 2005; Ullsperger & Nikolas, 2017). Moreover, individuals with a history of adversity have a higher risk of experiencing further adversities in adolescence (Cassels et al., 2018; Pearlin, 2010; Sterzing et al., 2019).

1.4. Purpose of Current Study

To build upon previous work characterizing associations between adverse experiences and brain structure, this study aims to examine the relationship between the history of adversity and changes in cortical thickness and subcortical volume during adolescence. Although informative about between-person differences, the single time point neural measurements in most reviewed studies cannot address questions regarding within-person brain development, which is better related to outcomes of interest like cognitive processing (Shaw et al., 2006). Of the few studies that have characterized the relationship between dimensions of adversity and metrics of brain development, several have found evidence for dimensional associations with resting state network development in adolescence (Chahal et al., 2022; Rakesh et al., 2021). These longitudinal findings neither completely support nor refute the DMAP hypotheses and highlight the need for further longitudinal work with various neurodevelopmental metrics. Additionally, associations between dimensions of adversity and neural development have been

shown to differ by biological sex (Rakesh et al., 2021). Furthermore, a recent call in the literature emphasizes the need to examine sex-specific effects of adversity on neurodevelopment to advance understanding of female development and correct historical bias toward male development in translational research (Bath, 2020).

As described in the methods, our modeling strategy abstracts from these direct estimates. It does not inform the directionality of neurodevelopmental effects (e.g., whether adversity produces accelerated or early deceleration of cortical thinning). Instead, we use a multivariate approach to ask a simple but essential question: Do individuals with similar adverse experiences also have similar brain development in adolescence and does that relationship change depending on the structuring of adversity?

1.5. Strengths of a Multivariate Approach

Measuring adversity has provided a difficult challenge for researchers in the field. Even with a clear working definition of adversity, capturing all types of adverse experiences in a single measurement tool can be nearly impossible. Furthermore, exposures that could be similar in their dimensionality may not be equal in their severity. For example, witnessing a physical attack and being the recipient of a physical attack are both considered threatening but might vary in their degree of threat. In traditional analyses, one can assign a predetermined severity weighting or determine participants to be threat-exposed or -unexposed. To examine these relationships through a novel lens, we propose to use inter-subject representational similarity analysis (IS-RSA) to determine how representational structures of adversity, designed to reflect the cumulative risk model and DMAP, relate to representational structures of adolescent brain development, as characterized by changes in cortical thickness and subcortical volume, in frontotemporal and frontoparietal regions, the amygdala, and the hippocampus.

Representational similarity analysis is an analytic framework that abstracts from direct measurements to test hypotheses in a second-order geometrical space (Kriegeskorte et al., 2008; Kriegeskorte & Kievit, 2013; Popal et al., 2019; Stolier et al., 2020). While traditionally, this method has been used to examine within-person brain activation, we intend to use this technique to examine inter-subject similarities of structural brain development. IS-RSA has previously been used to investigate individual differences in behavior and brain connectivity observed during neuroimaging tasks (Chen et al., 2020; Finn et al., 2020; van Baar et al., 2019). This study will use IS-RSA to investigate individual differences in behavior and brain development. This technique is highly flexible and allows for the use of granular data and summary scores to calculate inter-subject similarity, which preserves the multidimensionality inherent in the sample's exposures to adversity without bestowing additional importance on different types of exposures. Similarly, this multivariate approach is agnostic to how effects are distributed in the brain, allowing researchers to examine relationships while remaining agnostic to the precise mapping of exposures to brain regions. Multivariate approaches to neural data, such as IS-RSA, have been shown to be more sensitive and result in more stable correlation effects in functional neuroimaging compared to traditional univariate techniques (Grady et al., 2020; Lukic et al., 2002).

1.6. Strengths of a Longitudinal Approach

In addition to modeling adversity in a multifaceted way, this study is poised to examine its effects over time, which allows for more accurate inferences of neural development and requires fewer participants to observe structural change than a cross-sectional design (Marek et al., 2022; K. L. Mills & Tamnes, 2014; Pfeifer et al., 2018). Since the effects of adversity are biologically embedded and reified across time, using a within-subjects design allows us to

investigate how such experiences influence individual development. Longitudinal approaches allow for prospective reports of childhood adversity, which are more likely to reflect neural development than retrospective reports (Gehred et al., 2021a; Widom et al., 2004). Moreover, this study has been designed to contain multiple time points from adolescence where cortical thickness decreases with age (Frangou et al., 2022; Tamnes et al., 2017).

In this study, we will use IS-RSA to investigate the effects of adversity on structural development in adolescence and evaluate the ability of conceptual model representations to recover brain-behavior relationships. We expect that both the DMAP and cumulative risk representations will be significant predictors of neural development, and we predict that the DMAP will predict more variance in structural trajectories compared to the cumulative risk model. We also expect threat and deprivation to be uniquely associated with the fronto-amygdala and hippocampal circuit and frontoparietal network, respectively. Here, we seek to replicate emerging findings of differential influences and extend this work to examine neurodevelopment in a key period of life.

2. Methods

2.1. Sample Characteristics

This project uses four timepoints of data gathered through the Transitions in Adolescent Girls* (TAG) study and a fifth timepoint of data collected through a substudy (referred to as SOS) with the sample (Barendse et al., 2020). The study was designed to assess pubertal development and mental health in adolescent girls, so recruitment targeted students registered as female.

Prior analyses with these data had yet to examine changes in brain structure. Only one analysis inspected measurements of structural brain data within this sample. The first wave of structural scans was processed and analyzed for a separate study on the association between pubertal hormone concentration and cortical thickness and subcortical volume (Vijayakumar et al., 2019). Out of the adversity questionnaires, only socioeconomic status measurements were used in prior analyses (specifically an examination of self-perceived scholastic competence across adolescence).

189 adolescents aged 10.00 to 13.00 years and their caregivers from the Lane County community in Oregon, USA were recruited primarily through recruitment letters issued through schools (all districts in the greater Eugene/Springfield area) to all families with children registered in school as female students within our age range of interest, and to a smaller extent through community flyers and word-of-mouth. In this sample, seven participants were excluded (see Table 11) and eight withdrew before their first session, resulting in 174 adolescent girls* (in the first wave, 171 identified as girls and three as nonbinary; we confirmed that youth identifying as nonbinary were assigned female at birth). The study was approved by the University of Oregon Institutional Review Board, and assent and informed consent were obtained from the adolescents and their caregivers before inclusion in the study. No positive controls were included because this study was not a controlled experimental design. Distributions of ethnic and racial identity in the sample are more diverse than the local community as measured at Wave 1; a detailed breakdown is included in Table 12.

Table 11

Inclusion and Exclusion Criteria

Inclusion criteria	Exclusion criteria
Female	Diagnosed with a developmental disability
10, 11, or 12 years old	Diagnosed with a psychotic disorder
Fluent in English	Diagnosed with a behavioral disorder, including autism
Normal or corrected-to-normal vision	Taking psychotropic medication other than stimulants
	MRI contraindications
	Report or suspect being pregnant

Table 12

Race and Ethnicity

Race	Non-Hispanic/Latina/Chicana	Hispanic/Latina/Chicana	Percent Non-Hispanic/Latina/Chicana	Percent Hispanic/Latina/Chicana
Black	0	1	0.00%	0.57%
Asian	2	0	1.15%	0.00%
NAAN	1	0	0.57%	0.00%
White	118	20	67.82%	11.49%
White and NAAN	9	2	5.17%	1.15%
White and NHPI	0	1	0.00%	0.57%
White and Middle Eastern	1	0	0.57%	0.00%
White and Asian	8	0	4.60%	0.00%
White and Asian and NHPI	0	1	0.00%	0.57%
White and Asian and NAAN	1	0	0.57%	0.00%
Black and White	8	0	4.60%	0.00%
Not Specified	0	1	0.00%	0.57%

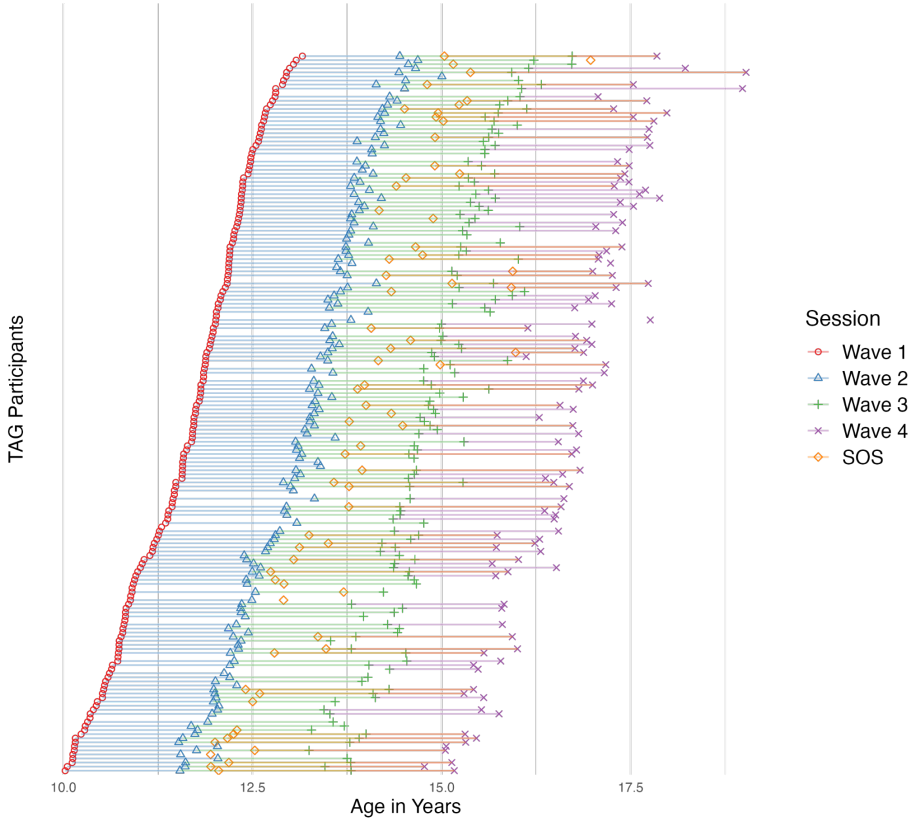
Note. 32.2% of participants identified as a racial or ethnic minority. Comparatively, 18.2% of the local county identifies as a racial or ethnic minority. NAAN = Native American and Alaskan Native; NHPI = Native Hawaiian and Pacific Islander.

Each wave of data collection took place approximately 18 months apart (range of 0.72-36.65 months due to pandemic-related disruptions). Each participant completed two laboratory visits at each wave, spaced approximately one month apart. Out of 174 participants in the first wave, 163 (93.68%) participants participated in the second wave, 150 (86.21%) participated in the third wave, and 109 (62.64%) completed the fourth wave. Questionnaires were completed at the first and second visits, and home; the MRI scan was completed during the second visit. A subsample of TAG participants (N=62) participated in an additional MRI scan for the SOS substudy that occurred largely between waves two and three (four TAG participants completed the substudy between waves three and four). All available high-quality scans from available waves were included to maximize participant inclusion. Figure 15 provides a visualization of

participation throughout the study. Structural scans from two to five timepoints (depending on image quality) were used to measure cortical thinning and subcortical volumetric change, and only data from the first corresponding timepoint of the included scans was used to measure adversity. Participants who only had a high-quality scan from fewer than two timepoints were excluded from these analyses.

Figure 15

Participation across Study Timepoints



Note. Each line represents each participant's age at the second session (the MRI session) across all time points.

2.2. Measure of Brain Structure

2.2.1. Structural scan

Prior to entering the 3T Siemens Skyra MRI scanner, participants completed a mock scan to help ease any scanner-induced anxiety. Participants watched a short, animated video while acquiring a T1-weighted 3D MP-RAGE structural scan. 176 slices of 1 mm isometric voxels were collected over 5 minutes and 59 seconds (FOV = 256 mm, TR = 2500 ms, TE = 3.41 ms, flip angle = 7 degrees).

2.3. Measures of Adversity

2.3.1. Childhood Trauma Questionnaire (CTQ)

We administered the CTQ form to adolescent participants with 32 items, but only the standard 25 items from the validated short form were used for these analyses. It has five subscales: physical abuse, emotional abuse, sexual abuse, emotional neglect, and physical neglect. The CTQ has demonstrated high convergent validity (Bernstein et al., 1994; Bernstein et al., 1997) with therapist ratings and trauma interviews and exhibits high levels of internal consistency across age groups (Bernstein et al., 2003; Scher et al., 2001; Spinhoven et al., 2014). Initial test-retest reliability coefficients vary between .79 and .86 across 2-6 months (Berenstein et al., 2003) and .44 to .67 across five years (Cammack et al., 2016). Given that researchers have noted concerns of the internal consistency of the physical neglect scale in Dutch, Swedish, and Canadian samples (Gerdner & Allgulander, 2009; Paivio & Cramer, 2004; Thombs et al., 2009), we conducted a sensitivity analysis and reported how this inclusion affected the results. We also

did the same for the emotional neglect scale, comprising five positive, reverse-coded items (e.g., "someone in my family made me feel important or special").

2.3.2. Socioeconomic status (SES) measurement

We used a single-item caregiver report on whether their child received free lunch at school since both household income and family size influence eligibility. We also included caregiver education (highest report if a participant had more than one caregiver) which indicates if caregivers received no high school diploma (or equivalent); high school diploma (or equivalent); some college credit (no degree), trade, technical, or vocational training; associate degree, undergraduate degree, or postgraduate degree. Finally, we used participants' residential addresses to create an index of neighborhood deprivation based on the computation provided by Messer et al. (2006) that incorporates census data on income, education, employment, housing, and occupation.

2.3.4. Youth Risk Behavior Survey (YRBS)

Two items from the YRBS were used to assess if the participant had ever been bullied on school property or electronically (i.e., through email, chat rooms, instant messaging, websites, or texting). Data was also summed to provide one composite measurement.

2.3.5. Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS)

The following events evaluated in the K-SADS Post-Traumatic Stress Disorder module were used to assess further experiences of trauma: a significant car accident that resulted in injury and required medical intervention, other accident that resulted in injury and required medical intervention, witnessed a fire that caused significant property damage or moderate-

severe physical injuries, witnessed a natural disaster that caused significant devastation, witness to threatening or violent crime, a victim of threatening or violent crime, terrorism-related traumatic event, war zone traumatic event, witness to domestic violence, physical abuse, and sexual abuse. These events were also summed to create a composite score.

2.4. Rates of adverse exposures

Within the full sample at wave 1, 35% reported receiving free lunch, 27% experienced at least low levels of abuse (according to CTQ abuse thresholds), 20% reported experiencing at least one traumatic event on the K-SADS, and 35% had been bullied either in-person or online. See Table 13 for the descriptive statistics of the CTQ subscales before imputation and Figure 16 for the subscale score distributions. These occurrence rates mirror those in another study with a community sample of 142 adolescents (Rakesh et al., 2021).

Table 13

CTQ Descriptive Statistics

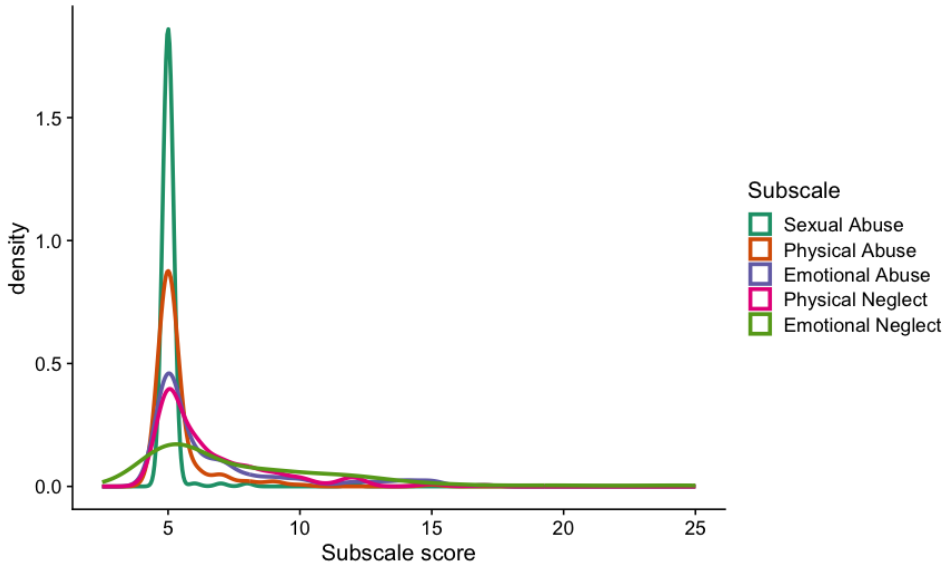
Subscale	Mean	Median	Standard Deviation
Physical Abuse	5.34	5	.89
Sexual Abuse	5.05	5	0.38
Emotional Abuse	6.61	5	2.66
Physical Neglect	6.73	6	2.35
Emotional	7.8	6	3.84

Neglect

Note. The average, median, and standard deviations of the sum scores for each CTQ subscale. CTQ subscale sums can range between 5 and 25.

Figure 16

CTQ Subscale Score Distributions



2.5. Proposed Analysis Pipeline

2.5.1. Processing of adversity data

Because our analyses cannot accommodate missing data, if three or more items within a CTQ subscale (all are composed of either five items) or three or more items of the K-SADS events list (out of ten items) were missing, we substituted the missing data with corresponding responses from the next closest timepoint. If these data were also missing from the next closest timepoint, that participant was excluded. If fewer than three items on the K-SADS events list and/or fewer than three items on the CTQ subscale were missing, these data were imputed using

the missforest algorithm with the following predictors: age, race and ethnicity, caregiver education, caregiver income, school of attendance, sum of traumatic life events (for CTQ imputation), and CTQ total score (for K-SADS imputation). Analyses with data substitutions will be compared to analyses with imputing all missing adversity data regardless of a number of items missing to determine whether this method significantly changes results. The number of data substitutions and imputations are reported in the results.

Since both conceptual models recognize the importance of incorporating accumulation and severity of adversity, inter-subject similarity for both dimensional and cumulative exposures was calculated using individual item responses and summary scores. This calculation method allowed for the total scores and variation in experiences to factor into inter-subject similarity. Data was grouped as shown in Table 14. Further details are provided in the statistical analysis section.

Table 14

Measure Groupings

Threat	Deprivation	Cumulative Risk
Physical, sexual, and emotional abuse items and summary scores	Physical neglect items and summary score	CTQ items and summary score
Electronic and in-person bullying and composite score	Emotional neglect items and summary score	Electronic and in-person bullying items and composite

Measure Groupings

		score
Traumatic events items and summary score	Free lunch	Free lunch
	Caregiver education	Traumatic events items and summary score
	Neighborhood deprivation index	Neighborhood deprivation index
		Caregiver education

2.5.2. Processing of neural data

Preprocessing of the structural scans, including the initial steps of skull stripping (Ségonne et al., 2004) and intensity correction (Sled et al., 1998), were completed with the FreeSurfer (v 7.2.0) imaging analysis suite (http://surfer.nmr.mgh.harvard.edu/) for all waves. This automatic process reconstructs models of the pia surface and gray and white matter boundary after segmentation (Fischl et al., 2002, 2004), tessellation, and topological correction (Fischl et al., 2001; Ségonne et al., 2007).

Two trained coders visually inspected anatomical scans for artifacts associated with participant motion (i.e., blurring or ringing). Motion artifacts can negatively bias cortical estimates, necessitating a rigorous and transparent quality control process (Backhausen et al.,

2016). The scans were rated on a four-point scale for evidence of motion, with 4 representing major amounts of motion disturbances, 3 indicating moderate amounts of visible motion in multiple areas or across a number of slices, 2 indicating minor motion disturbances across a few slices, and 1 indicating a sharp image with no visible motion artifact. Scans were also being rated for accurate white and pial surface boundaries on a four-point scale, with 4 indicating missing or entirely inaccurate surface boundaries, 3 indicating inaccurate boundaries in multiple areas, 2 indicating poor boundaries in limited areas, and 1 reflecting good surface boundaries. To diminish the impact of motion artifacts, scans that received scores of 3 or higher for the motion or surface rating were excluded from analyses. Scans that received scores of 2 for either rating were further inspected to ensure that regions specific to these analyses were not visibly affected.

After completing manual quality control, 111 participants were deemed eligible for inclusion in these analyses by having at least two time points of usable neuroimaging data. Of the included participants, 33 had two usable scans, and 78 had three or more usable scans. Of those excluded participants, just over half (n = 36) had only one high-quality scan across waves.

After the initial quality inspection, scans were run through FreeSurfer's longitudinal pipeline (Reuter et al., 2012), and scans that received a 3 or 4 rating on surface boundaries were re-examined to assess whether surface reconstruction improved after using an unbiased within-subject template which informs the standard processing steps. Cortical thickness was estimated using the smallest distance between the gray and white matter boundary and the gray and pial surface boundary at each vertex (Fischl & Dale, 2000). Cortical thinning estimates were estimated by calculating the annualized percent changes in cortical thickness for individuals with at least two timepoints of usable scans. We used the same equation as Mills et al. (2021) to calculate the annualized percent change for each region between each scan:

$$\frac{\left(\left(\frac{Brainmeasurement_{x+1} - Brainmeasurement_{x}}{\left(\left(Brainmeasurement_{x} + Brainmeasurement_{x+1}\right)/2\right)}\right) \times 100\right)}{Age_{x+1} - Age_{x}}$$

Annualized percent change scores were then averaged for participants with three or more usable scans to create a single change score.

The following regions of interest (ROIs) examined in this study were chosen to reflect regions in the fronto-amygdala and hippocampal circuit and frontoparietal network described in McLaughlin et al.'s 2019 review: amygdala, hippocampus, rostral anterior cingulate cortex, medial orbitofrontal cortex, rostral middle frontal cortex, and superior parietal cortex. We used FreeSurfer's anatomical masks for the amygdala and hippocampus. Cortical regions were defined by selecting these parcellations from the Desikan-Killiany atlas, given its use in the relevant literature and theoretical specificity. For hypotheses 1 and 2, the inter-subject similarity was based on each ROI's average annualized percent change scores, resulting in one representation of structural brain development. For hypothesis 3, inter-subject similarity was based on average annualized percent change scores in two separate groups of ROIs. One group, expected to be associated with threat, included the amygdala, hippocampus, rostral anterior cingulate cortex, and medial orbitofrontal cortex. The other group, expected to be associated with deprivation, included the rostral middle frontal cortex and superior parietal cortex.

2.6. Hypotheses

1. It was hypothesized that each inter-subject adversity representation, as organized by both the cumulative risk model and the DMAP (threat and deprivation), will be significant predictors of the similarity representation of structural brain development. Specifically, similarity across all adverse experiences would significantly predict similarity in

structural brain development (cumulative model); also, similarity in threatening experiences and similarity in deprivation experiences would together significantly predict similarity in structural brain development (dimensional model). We further hypothesized that both predictors in the dimensional model equation would have significant beta coefficients.

2. If the DMAP better delineates neurophysiological development, it was hypothesized that patterns of inter-subject similarity of threat and deprivation would predict more variance in trajectories of structural brain development than similarity along one cumulative adversity pattern.
3. If the theoretical neural specificity of the DMAP is supported, it was expected that similarity in threat experiences would better predict similarity in fronto-amygdala and hippocampus circuit development compared to similarity in all adverse experiences or similarity in deprivation. Likewise, similarity in deprivation experiences would better predict similarity in the frontoparietal network development than similarity in all adverse experiences or similarity in threat.

2.7. Statistical Analyses

To test these hypotheses with IS-RSA, we built inter-subject representational dissimilarity matrices (IS-RDMs) of both adversity-related experiences (threat, deprivation, and overall adversity) and structural change (all ROIs and separately grouped ROIs). Inter-subject dissimilarity was calculated using Euclidean distances between participants along the measures of interest to create a matrix, the height and width corresponding to the number of participant comparisons, and symmetric along the diagonal. Adversity data was transformed so that experiences that were measured with a greater number of items (e.g., physical abuse) did not

have greater weight when calculating similarity than others. Items were divided by the number of items per experience (e.g., each of the five physical abuse items were divided by five). All items were transformed to a percent of the maximum score so that continuous variables were not weighted more heavily than categorical variables.

These matrices were then vectorized above the diagonal, and the adversity matrices were included in regression models used to predict the structural change matrix. To test the first hypothesis, the structural change matrix was regressed along the threat and deprivation matrices (structural change ~ threat + deprivation) and the cumulative experiences matrix (structural change ~ cumulation). To test the second hypothesis, we added an interaction term to the dimensional model (structural change ~ threat + deprivation + threat*deprivation) to determine whether the interaction of similarity is a significant predictor of similarity in brain development. Interaction in these similarity dimensions could signify that inter-subject matching along *both* dimensions of threat and deprivation (compared to inter-subject mismatches in one or both dimensions) is helpful in predicting similarity in brain development above and beyond similarity along each dimension separately. High levels of inter-subject matching in both dimensions could occur regardless of individual reports of high or low adversity in either dimension. In other words, paired individuals with high similarity in both dimensions might have high adversity in one dimension but not in the other, high adversity in both dimensions, or low adversity in both. While the absence of a significant interaction would not disprove a dimensional model, the presence of a significant interaction would support the theory that dimensions of adversity are uniquely relevant to brain development. If this interaction term was not significant, it would not be included as a final parameter in the DMAP model. Finally, we reported the overall fit statistics of the DMAP and cumulative risk models, including adjusted R^2, AIC, and BIC.

While a comparison of fit statistics is not a direct statistical test, a greater R^2 and smaller AIC and BIC for one model over the other across all three would provide support for the corresponding conceptual model. If AIC and BIC were each at least two points lower in one model, we considered positive evidence for that corresponding model. To test the third hypothesis, predictors (threat, deprivation, cumulation) were individually associated with each outcome matrix for the two ROI groupings. A difference of dependent slopes tested differences in association magnitudes. Tests were considered statistically significant at an alpha level of 0.05.

We provided additional non-parametric assessments using a similar approach to a Mantel permutation test to complement each of the three hypothesis tests. Like the strategy Finn et al. (2020) used, we created null distributions by computing 10,000 statistics (e.g., beta values) with permuted participant IDs in one of the similarity matrices. The observed statistic was compared to the distribution to obtain a p-value.

To address the concerns of the CTQ physical and emotional neglect subscales' psychometric properties, we conducted sensitivity analyses to determine whether inclusion altered the significance of the predictors. Before creating the dissimilarity matrices, group comparisons between participants who were excluded due to poor scan quality and participants who were included were conducted for all adversity measurements (CTQ scores, traumatic events score, caregiver education, neighborhood deprivation index, bullying, and free lunch) to see whether data loss is greater among individuals with more adverse exposures. Measurements of internal consistency for all CTQ subscales were reported for included participants.

2.8. Statistical Power Analysis

Unfortunately, there is no established way to conduct a power analysis with IS-RSA, so we have examined the literature to compare the sample sizes of studies that similarly sought to identify brain-behavior relationships. The two studies that have examined experience and brain development to identify associations between dimensions of adversity and functional connectivity network stability had community sample sizes of 142 and 225 (Chahal et al., 2022; Rakesh et al., 2021). In IS-RSA analysis, each subject will have approximately 99 pairwise distance scores, resulting in a vector with thousands of inter-subject pairings in experience predicting thousands of inter-subject pairings in structural development, and consequently should have greater power than univariate analyses. Studies that have used IS-RSA to examine behavior and brain activation relied upon samples of 26 (Chen et al., 2020), 57 (van Baar et al., 2019), and 93 (with an additional holdout sample) participants to build their matrices (Finn et al., 2020). With our statistical plan, we expect our study to be well-powered to detect similar effect sizes to those reported in the previous literature.

2.9. Planned Exploratory Analyses

Additional brain regions have been empirically associated with components of threat and deprivation that were not necessarily encapsulated by the theoretically driven ROIs identified by McLaughlin and colleagues (2019). We noticed that when reported at the voxel level, observed clusters frequently reflected only a small part of the large parcel descriptions. To examine the relationship of adversity with these previously observed regions, we will build similarity matrices composed of cortical metrics within parcels of the Schaefer 400 parcellation atlas (Schaefer et al., 2018) that have been empirically associated with threat and deprivation in

addition to the amygdala and hippocampus. These "observed ROIs" were generated by examining the regions in papers cited in McLaughlin and colleagues' 2019 review that provided cortical thickness or volumetric results in samples with exposure to threat or deprivation (Butler et al., 2018; Edmiston et al., 2011; Gold et al., 2016; Hanson et al., 2010; Hodel et al., 2015; McLaughlin, Sheridan, Winter, et al., 2014). These ROIs are represented in Figure S1 and Figure S2 and listed in Table S1. Parcels were selected differently based on how data was reported (e.g., MNI coordinates, brain image slice with highlighted regions, flat map representations, etc.). If a coordinate was provided that fell at the border between parcels, we selected the parcel that was most consistent with the label or term the authors used. If regions were not clearly delineated in a paper, discussions were had to achieve consensus on an appropriate parcel. To mirror our theoretically driven hypothesis one, this brain similarity matrix will be regressed upon the same matrices operationally defined by the cumulative and dimensional models.

Furthermore, to test whether these effects are specific to those previously studied regions and evaluate how well they compare to other parcellation combinations, we plan to create two distributions (one for the DMAP and one for the cumulative risk model) wherein random parcellations from the whole brain are used to build the brain similarity matrix. Beta coefficients from the predictor variables will be logged as we iterate through the 10,000 randomly generated brain similarity matrices to generate distributions. We will report the placements of beta coefficients from our cumulative model and dimensional model tests on the distribution to illustrate their predictive capacity compared to random parcellations.

Finally, we will conduct a latent class analysis to characterize the common experiences of adversity in this community sample. First, using the same adversity data, we will determine how many and what kinds of profiles emerge amongst our sample. We will assess whether the data

naturally cluster around dimensions such as threat and deprivation and test whether profiles are each associated with development in each brain region of interest individually.

2.10. Deviations from Stage 1 manuscript

Several minor deviations and clarifications from the original Stage 1 manuscript were made during the completion of this study. When processing the adversity data, we needed to impute one participant's income and substitute six participant's income. We also used parent-reported race and ethnicity if the child report was unavailable. We did not have information about school of attendance for many participants, so we did not use this variable during imputation. Additionally, we stated that we would use addresses provided at wave 1, however, that number was relatively small, so we also used addresses if they were provided retrospectively at later dates. When processing the neuroimaging data, we used FreeSurfer v 7.2 instead of v 6. We used FreeSurfer's masks for the amygdala and hippocampus instead of the Harvard-Oxford masks created in our lab when, upon visual inspection, we found that FreeSurfer's masks were better aligned with the underlying subcortical anatomy.

2.11. Transparency and openness

This study's analytic plan was pre-registered and refined through the registered report process. Statistical analyses were completed using version 4.3.0 of R (R core team, 2023), the *tigris* (*v2.0.3*; Walker, 2023), *ndi* (*v.0.1.4*; Buller, 2022), *tidycensus* (*v1.4.1*; Walker & Herman, 2023), *missForest* (*v.1.5*; Stekhoven, 2022; Stekhoven & Buehlmann, 2012), and the *poLCA* (*v.1.6.0.1*; Linzer & Lewis, 2011) packages. Code for the main analyses and data are available at https://osf.io/bpzju/ (Barrett et al., 2023).

3. Results

3.1. Missing data

The number of participants whose data were substituted from the next closest wave and the number of participants whose data were imputed is reported in Table 15. Most participants needing data imputed for the CTQ only missed one item. No participants needed to be excluded due to unavailable data from the next closest wave.

Table 15

Addressing Missing Data

Measure	Substitutions	Imputations
YRBS	*12*	*1*
Caregiver education	*4*	*0*
Free Lunch	*3*	*0*
KSADS items	*3*	*0*
CTQ items	*12*	*34*
Income	*6*	*1*

Note. The number of participants who needed substitutions or imputations is listed for each measure.

3.2. Effects of data exclusion

Group comparisons were conducted to determine whether the participants included in these analyses differed in their experiences of adversity compared to the participants who did not

have enough usable neuroimaging data to be included. We found that included participants had lower NDI scores (mean = -0.0056) than excluded participants (mean = 0.0285), indicating that they had lower amounts of neighborhood deprivation on average; $t(106.07) = 3.17$, $p = .002$, 95% CI [0.01, 0.06]. They did not differ in rates of receiving free lunch, getting bullied, trauma exposure, emotional abuse, physical abuse, sexual abuse, emotional neglect, physical neglect, or parental education.

3.3. Internal consistency of CTQ subscales

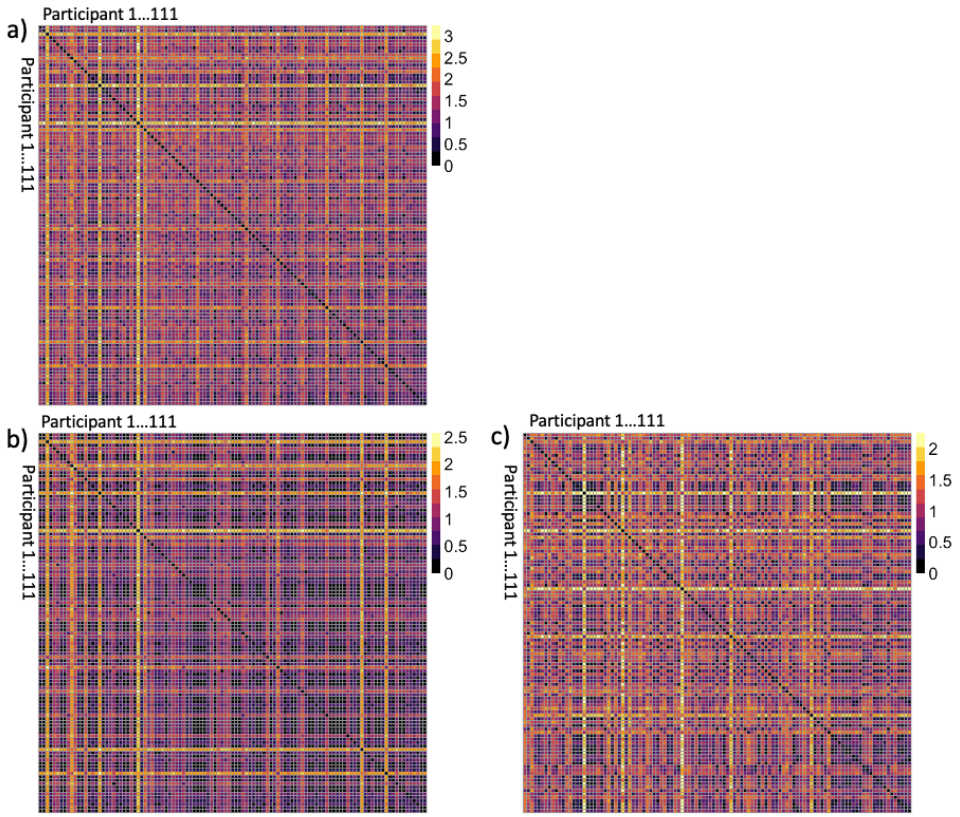
The internal consistency of the CTQ subscales was assessed by Cronbach's alpha coefficients. Similar to other researchers, we found poor internal consistency for the physical neglect subscale of the CTQ ($\alpha = 0.44$). We could not determine the internal consistency values for sexual abuse due to the lack of variability in this sample. All other CTQ subscales demonstrated acceptable levels of internal consistency and were as follows: physical abuse ($\alpha = 0.68$), emotional abuse ($\alpha = 0.77$), and emotional neglect ($\alpha = 0.82$).

3.4. Inter-subject adversity matrices

The IS-RDMs for each adversity configuration are shown in Figure 17. The overall adversity IS-RDM was positively associated with the threat IS-RDM ($r = 0.82$) and deprivation IS-RDM ($r = 0.76$). The threat and deprivation IS-RDM were also positively associated, though to a lesser degree ($r = 0.30$).

Figure 17

Adversity Inter-subject Representational Dissimilarity Matrices



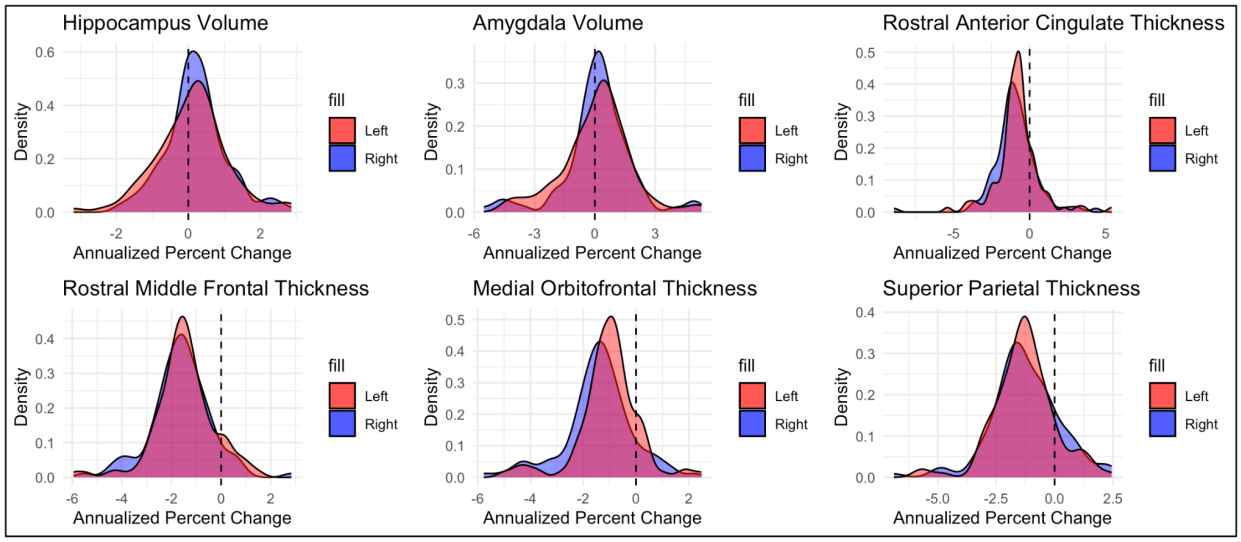
Note. Inter-subject dissimilarities (in Euclidean distance) across a) all adversity, b) threat, and c) deprivation are displayed. Higher values indicate further distance between participants in their responses to adversity items.

3.5. Brain development matrix

Annualized percent change scores demonstrate thinning in the rostral anterior cingulate cortex, rostral middle frontal cortex, medial orbitofrontal cortex, and superior parietal cortex. See Figure 18 for the annualized percent change score distributions for each ROI. The IS-RDM for annualized percent change in all ROIs is shown in Figure 19.

Figure 18

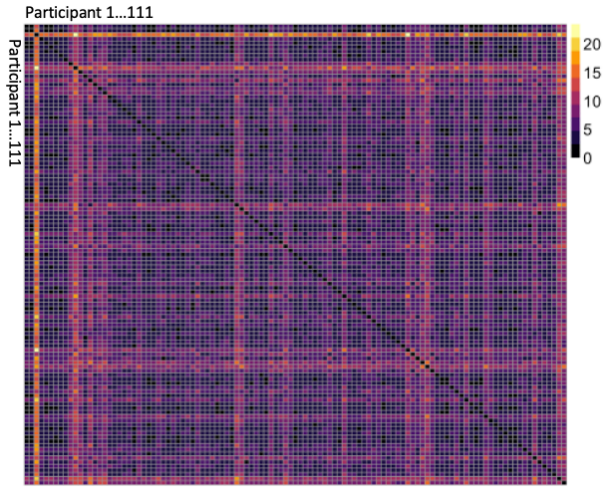
Distributions of Annualized Percent Change Scores



Note. Annualized percent change scores for each region of interest in both the right (blue) and left (red) hemispheres.

Figure 19

Inter-subject Dissimilarity Matrix for Annualized Percent Change across Regions of Interest

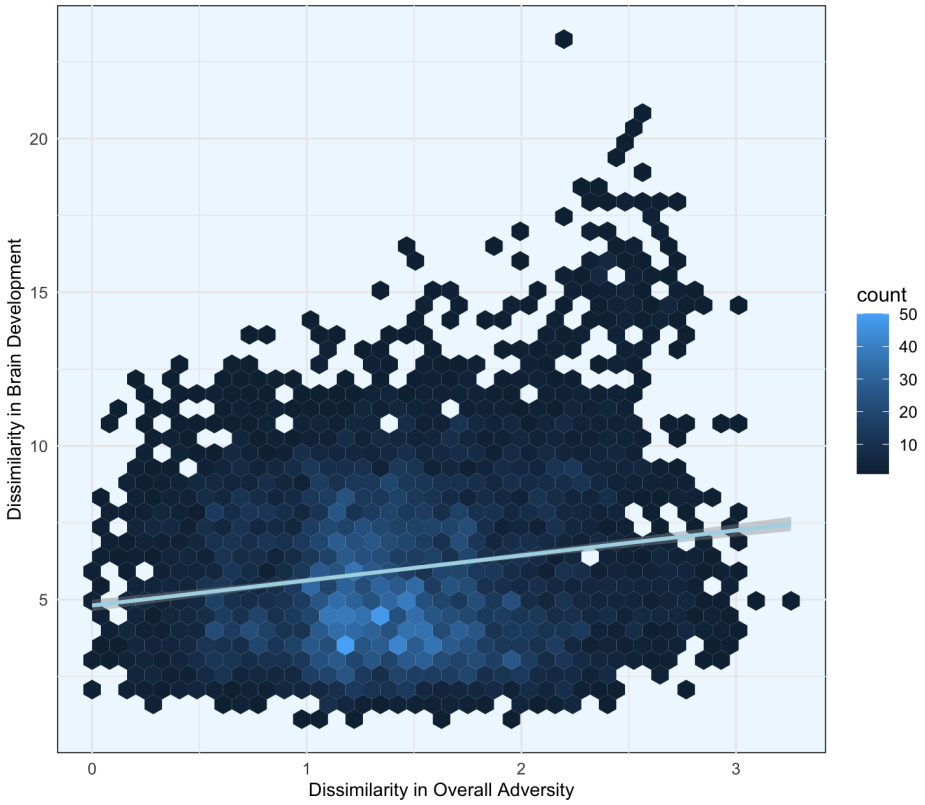


3.6. Hypothesis 1

We hypothesized that similar experiences in each of the three adversity groupings (threat IS-RDM, deprivation IS-RDM, and overall adversity IS-RDM) would significantly predict inter-subject similarity in structural brain development. Consistent with our hypothesis, the dimensional model accounted for a significant amount of variance in inter-subject similarity in brain development (adjusted $R^2 = 0.03$, $F(2, 6102) = 102.9$, $p < 0.001$) and each predictor variable, including the threat IS-RDM ($\beta = 0.17$, $t(6102) = 12.81$, $p < 0.001$) and deprivation IS-RDM ($\beta = 0.03$, $t(6102) = 2.36$, $p = 0.01$), demonstrated significant associations with the brain structure IS-RDM. In other words, participants with dissimilarity in threat and dissimilarity in deprivation would also have dissimilarity in their structural brain development in the front-amygdala and hippocampal circuit and frontoparietal network. A simple linear regression analysis also revealed that overall adversity IS-RDM was positively associated with the structural brain IS-RDM ($\beta = 0.17$, $t(6103) = 13.09$, $p < 0.001$). In simpler terms, this suggests that participants with similarity in all adversity were also more similar in their structural brain trajectories. See Figure 20 for a visualization of this relationship.

Figure 20

Dissimilarity in Overall Adversity vs. Dissimilarity in Brain Development



Note. The regression line demonstrates a small, positive relationship between dissimilarity in overall adversity and dissimilarity in brain development. The x- and y-axis each represent inter-subject Euclidean distance.

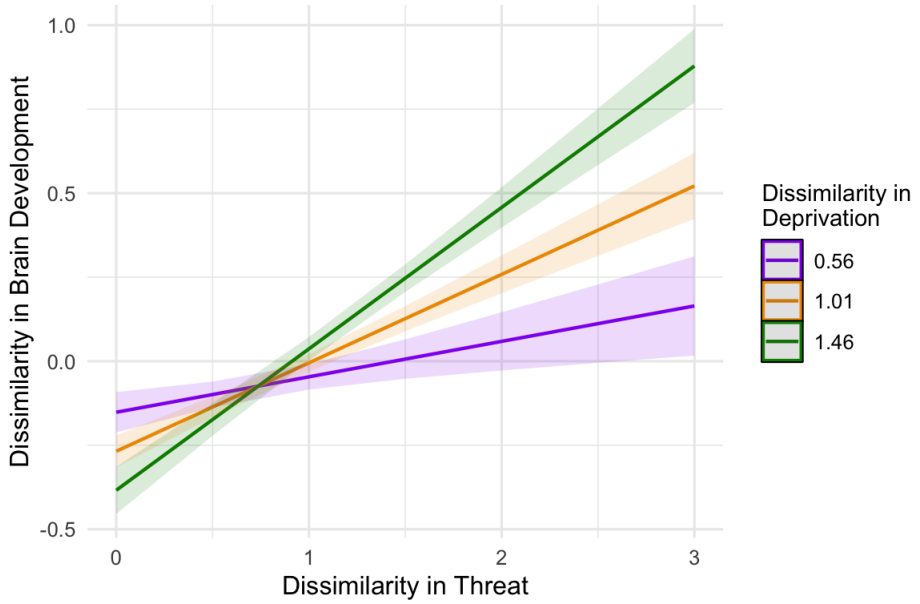
3.7. Hypothesis 2

We also hypothesized that the interaction in inter-subject similarity between threat and deprivation would uniquely predict structural brain development. The results were consistent with this hypothesis and suggest that participants who have dissimilarity in threat and dissimilarity in deprivation are most dissimilar in structural brain development compared to those dissimilar in just one dimension (see Figure 21). Put differently, dissimilarity in brain development amongst participants increases at a higher rate with the increase in dissimilarity in threat exposures for participants with dissimilar exposure to deprivation. Since this interaction

was significant (β = 0.09, $t(6101) = 7.82$, $p < 0.001$), it was included as a predictor in the final dimensional model. This dimensional model explained a significant, though small, portion of the variance in inter-subject similarity in structural brain development (adjusted $R^2 = 0.04$, $F(3, 6101) = 89.67$, $p < 0.001$). After accounting for the interaction in the model, both the deprivation IS-RDM (β = 0.02, $t(6101) = 2.15$, $p = .03$) and threat IS-RDM (β = 0.15, $t(6101) = 11.52$, $p < 0.001$) predicted further dissimilarity in the brain trajectories. Based on our inference criteria of a greater adjusted R^2 and at least 2 points lower BIC and AIC, this dimensional model (adjusted $R^2 = 0.04$, AIC = 17071, BIC = 17104) better predicted inter-subject similarity in structural brain development than the cumulative model (adjusted $R^2 = 0.03$, AIC = 17161, BIC = 17181).

Figure 21

Interaction between Threat and Deprivation



Note. At higher dissimilarity in deprivation, higher dissimilarity in threat predicts more dissimilarity in brain development.

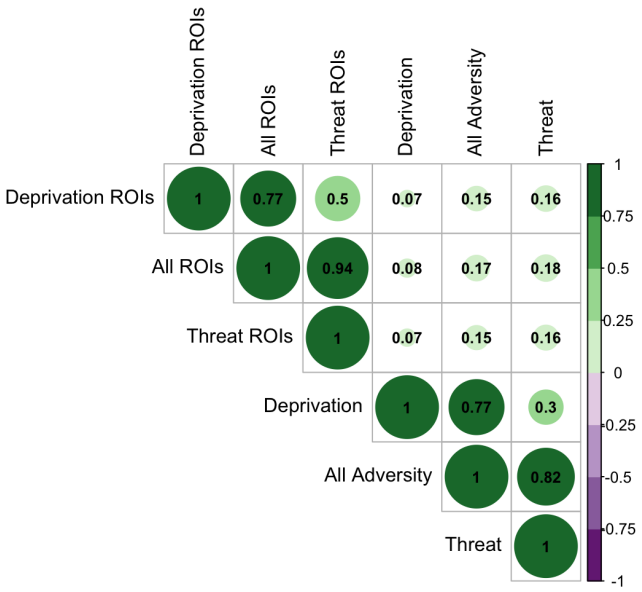
3.8. Hypothesis 3

In our third hypothesis, we predicted that the inter-subject similarity in threat experiences would best predict inter-subject similarity, specifically in fronto-amygdala and hippocampus circuit development. Indeed, threat IS-RDM had the largest correlation with the fronto-amygdala and hippocampus IS-RDM (as compared to the deprivation IS-RDM and overall adversity IS-RDM). A comparison between overlapping correlations based on dependent groups revealed a significant difference between these correlations.

Though there was also a significant difference between adversity groupings for the frontoparietal network IS-RDM, the deprivation IS-RDM was the smallest correlation. This finding was inconsistent with our hypothesis that the inter-subject similarity in deprivation experiences would best predict inter-subject similarity in frontoparietal network development. Correlations between all IS-RDMs are shown in Figure 22.

Figure 22

Correlations between Intersubject Dissimilarity Matrices



Note. Adversity and ROI RDMs are all positively correlated. Threat IS-RDM is most associated with each ROI RDM.

3.9. Permutation tests

The permutation tests for all three hypotheses supported the findings from the null hypothesis significance testing. Full details are provided in the Supplementary Information.

3.10. Sensitivity analyses

We conducted sensitivity analyses to estimate the impacts of including physical and emotional neglect subscales on the results. We found that analyses were mainly robust to these model adjustments. Without including physical neglect in the overall adversity and deprivation IS-RDMs, the deprivation IS-RDM was no longer a significant predictor of the brain development IS-RDM in hypothesis one. However, all other analyses were robust to the exclusion. Without including emotional neglect in the overall adversity and deprivation IS-RDMs, we found that the overall adversity IS-RDM, rather than the threat IS-RDM, was most

associated with the hypothesized threat-related ROIs. All other results exhibited quantitative and qualitative consistency with the proposed analyses.

When data was all imputed instead of making the predetermined substitutions from the nearest wave for high percentages of missing data, we observed two differences in the results. Though the deprivation IS-RDM was a significant predictor of brain development, it was in the opposite direction as hypothesized. Additionally, the interaction between threat IS-RDM and deprivation IS-RDM was insignificant. The effects of using these variables and methods are addressed in the Discussion.

3.11. Results Summary

Table 16 provides a summary of all results from the proposed analyses as well as the results of the three sensitivity analyses. The majority of the results were in line with the hypotheses.

Table 16

Results of Preregistered Hypothesis

Hypothesis	Proposed analyses	Analyses without physical neglect	Analyses without emotional neglect	Analyses without wave substitutions
1. Threat is a significant predictor	Yes	Yes	Yes	Yes

1. Deprivation is a significant predictor	Yes	**No**	Yes	**No**
1. All adversity is a significant predictor	Yes	Yes	Yes	Yes
2. Interaction between threat and deprivation	Yes	Yes	Yes	**No**
2. Dimensional model explains more variance	Yes	Yes	Yes	Yes
3. Threat is more strongly associated with theoretical ROIs	Yes	Yes	**No**	Yes
3. Deprivation is more strongly associated with theoretical ROIs	**No**	**No**	**No**	**No**

Note. 'Yes' indicates the result aligned with the hypothesis for the specified analysis, and 'No' indicates that the result did not support the hypothesis for the specified analysis in either the direction or expected effect.

3.12. Exploratory analyses

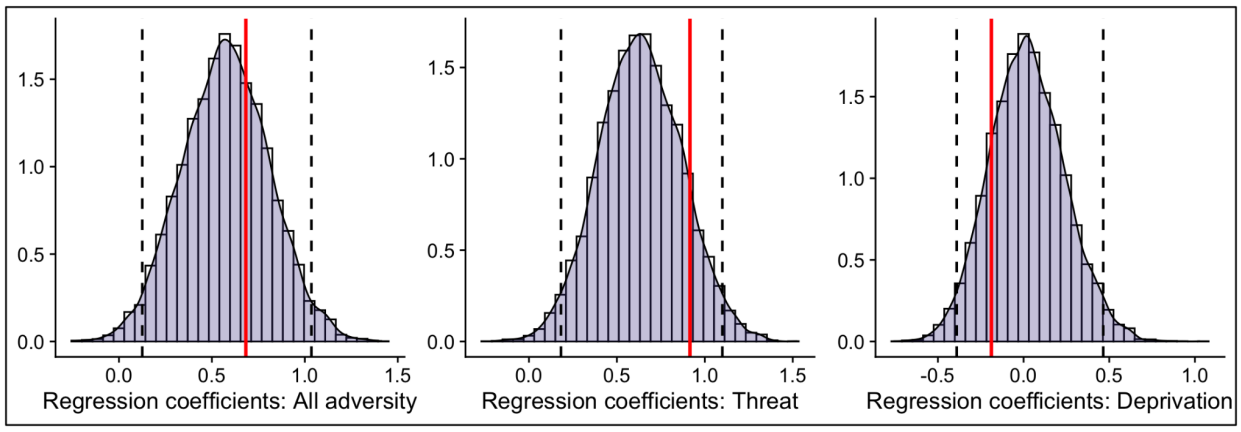
We created a new structural brain development IS-RDM of "observed ROIs" and completed the same analyses as in hypothesis one. In the dimensional model predicting the observed ROI IS-RDM, the threat IS-RDM was a positive predictor ($\beta = 0.91$), while the deprivation IS-RDM appeared to have a smaller effect ($\beta = -0.18$). Overall, this dimensional model explained limited variance in this outcome ($R^2 = 0.010$). In the cumulative model, the overall adversity IS-RDM predicted similarity in the observed ROI IS-RDM ($\beta = 0.68$), and the variance explained by the predictor was also limited ($R^2 = 0.007$).

To explore whether the effect of adversity on brain development is localized to these observed regions or extends more widely in the brain, we created a distribution of beta coefficients for each predictor in the adversity model by randomly selecting parcellations from the whole brain Schaeffer 400 atlas and creating a new structural brain development IS-RDM 10,000 times. When compared to this distribution, the relationships between each adversity IS-RDMs and the observed ROI IS-RDM were not significantly stronger, though the threat IS-RDM does appear to be nearing the threshold (see Figure 23). This result suggests that the observed ROIs do not reflect a unique effect of adversity on brain development compared to any ROIs. Of note, the distributions of threat and all adversity beta coefficients indicate a general positive effect that is significantly different than 0, whereas the distribution for deprivation beta coefficients is centered around 0. This suggests that our measurement of dissimilarity in

deprivation does not capture a widespread effect on brain development when controlling for dissimilarity in threat.

Figure 23

Distributions of Randomly-Selected Parcellations



Note. Annualized percent change scores for randomly selected parcellations from the whole brain were used to create 10,000 new structural brain development IS-RDMs. These were regressed along each adversity IS-RDM. The unstandardized regression coefficients from the models with the "observed ROIs" are marked by the red lines.

Finally, we conducted a latent class analysis to explore adversity groupings in this sample. Adversity data were binarized to increase interpretability. CTQ subscales were recoded to indicate whether participants met the standard thresholds for low levels of abuse or neglect (Bernstein & Fink, 1998). K-SADS endorsements were used to indicate whether a participant had experienced a traumatic event. Neighborhood deprivation indices were recoded to indicate whether participants were above or below the average index score in Oregon. Parental education data were recoded to indicate whether a participant's caregiver obtained a college or other advanced degree. The free lunch and bullying variables were already binary. With these

categorical variables, we fit models of two to six latent classes. Fit statistics for each model are reported in Table 17.

Table 17

Fit Statistics of Latent Class Models

Classes	AIC	BIC	Log-Likelihood	Entropy
2	1171.3	1233.6	-562.6	5.1
3	1169.9	1264.7	-550.0	4.9
4	1169.7	1297.0	-537.8	4.8
5	1177.8	1337.7	-529.9	4.8
6	1188.6	1381.0	-523.3	4.7

The two-class model, which generally classified participants into either high or low adversity groups, had the lowest BIC of all models. However, the three-class model also had a lower AIC and a higher log-likelihood (while still relatively lower BIC than the four-, five-, and six-class models). The three-class model identified a group characterized by lower parental education and free school lunch qualification (and low levels of other adversity indicators), a group characterized by endorsements of both threat and deprivation, and a group characterized by low levels of adversity overall. We examined both the two- and three-class models to see how

these representations were related to the hypothesized regions of interest: amygdala, hippocampus, rostral anterior cingulate cortex, medial orbitofrontal cortex, rostral middle frontal cortex, and superior parietal cortex. Mean annualized percent change values in each of the regions of interest did not differ between classes for either the two- or three-class models. The details of these class comparisons for each ROI are noted in the Supplementary Information.

4. Discussion

In this registered report, our primary objective was to assess the influence of threat, deprivation, and overall adversity on the structural trajectories of two neural systems: the frontoparietal network and the fronto-amygdala and hippocampus circuit. To investigate these relationships, we employed inter-subject representational similarity analysis (IS-RSA), an analytical approach more commonly used in functional neuroimaging studies (Finn et al., 2020). This methodology allowed us to examine whether multivariate representations of individualized experiences could effectively predict idiosyncratic variations in the developmental trajectories of specific brain regions previously implicated in the literature (McLaughlin et al., 2019). We chose this approach because it leveraged the available data and accounted for the intricate nature of individuals' experiences of adversity and subsequent brain development. In the context of our investigation, we sought to determine whether categorizing adverse experiences according to the dimensions of threat and deprivation would yield more accurate predictions of the developmental patterns across these brain regions compared to an overall grouping of these adversities. In summary, our findings substantiate our hypotheses and underscore the significance of dimensional grouping in understanding the impact of adverse experiences on brain development.

4.1. Effect of dimensional approach

Consistent with the Dimensional Model of Adversity and Psychopathology (DMAP), our results demonstrated a significant relationship between inter-subject similarity in threat experiences and inter-subject similarity in brain development, after controlling for inter-subject similarity in deprivation experiences. Specifically, we observed that greater dissimilarity in exposure to threat-related adversities corresponded to greater dissimilarity in the annualized percent change across all examined regions. This same effect held for inter-subject similarity in deprivation experiences, such that higher dissimilarity in exposure to deprivation adversities was associated with higher dissimilarity in brain development. These results suggest that incongruence in the experiences of adversity along distinct dimensions serve as predictive indicators of incongruence in brain development among individuals.

Furthermore, our analysis revealed a significant interaction between dissimilarity in threat and dissimilarity in deprivation, indicating that individuals with incongruent experiences in both of these dimensions exhibited the highest levels of dissimilarity in brain development. To clarify, when participants had divergent experiences in both threat and deprivation, it resulted in a more pronounced mismatch in their structural brain development. Such discrepancies in a dimension could occur due to varying levels of exposure, where one individual had a substantial exposure while another had minimal exposure, or because both individuals had experiences within that adverse dimension, but their specific exposures did not overlap. For instance, if one participant exclusively encountered physical abuse while another solely experienced peer bullying, these non-overlapping experiences would contribute to an increased dissimilarity in threat experiences. This interaction underscores the significance of considering both threat and

deprivation dimensions when investigating their respective effects on brain structure, emphasizing the nuanced and complex interplay between different adverse exposures.

We constructed a model employing a single inter-subject representational dissimilarity matrix (IS-RDM) to encompass all measured adversities, aiming to reflect the cumulative risk model while introducing an essential departure from conventional approaches. Historically, the cumulative risk model has often been operationalized using a single summary score that quantifies total adversity exposure (Evans et al., 2013; Lacey & Minnis, 2020). This summary score approach, however, tends to overlook the clustering of adversities, a limitation frequently criticized in the context of the cumulative risk model (Lacey & Minnis, 2019; McLennan et al., 2020). By incorporating item-level data into the IS-RDM, our model considered inter-subject similarity influenced not only by the overall quantity of adversity experienced but also by the specific endorsements of individual adversities. Like other cumulative risk approaches, two participants with divergent levels of adverse exposures would exhibit greater dissimilarity. Yet, our approach had the added capability of capturing dissimilarity among participants with high levels of adversity but dissimilar exposure profiles. Consequently, while this approach did not impose predetermined theoretical dimensions of adversity, it accommodated more dimensionality in the model than a conventional summary score. In our analyses, this model successfully predicted variations in brain development, but it was slightly outperformed by the dimensional model, thereby reinforcing the argument for considering dimensionality in adversity research.

Our investigation into the theoretical neural specificity of the Dimensional Model of Adversity and Psychopathology (DMAP) yielded partial support. Specifically, we observed that inter-subject similarity in the development of the fronto-amygdala and hippocampus circuit was

predominantly associated with inter-subject similarity in threat experiences. However, an intriguing contrast emerged in the case of the frontoparietal network, where inter-subject similarity in threat experiences, rather than deprivation experiences, exhibited a stronger association with its development. This latter finding deviated from our initial hypothesis, which posited that deprivation would play a more prominent role in shaping differences in frontoparietal structure, as previously suggested in related research (McLaughlin et al., 2019). This divergence might be attributed to potential limitations in the way we measured deprivation in this study. Alternatively, it is conceivable that the impact of threatening experiences extends beyond specific regions, exerting a more global effect on structural brain development. Indeed, our analysis revealed that inter-subject similarity correlated most strongly with inter-subject similarity across all specified regions of interest (ROIs), encompassing both the frontoparietal network and the fronto-amygdala and hippocampus circuit.

Furthermore, an exploratory analysis indicated that, across all operationalizations of adversity, the relationship between adversity and brain development remained consistent regardless of whether we selected parcellations to align with associations established in prior studies or chose parcellations randomly from the entire brain. This observation aligns with recent findings by Gehred et al. (2021) and suggests that there is merit in examining the brain as a holistic entity rather than focusing exclusively on specific regions when elucidating the neurobiological repercussions of adversity. This holistic perspective may provide valuable insights into the comprehensive impact of adverse experiences on brain structure.

Notably, our exploration of a latent-class approach did not yield any discernible relationships between adversity groups and specific neural regions of interest. This result contrasts with the findings of others who have successfully utilized this technique to unveil

adversity-physiology associations (Lacey et al., 2020). In our particular sample, it was only through the preregistered intersubject similarity approach that we were able to uncover these noteworthy relationships between adversity and neural regions of interest. This discrepancy underscores the potential variability in the effectiveness of different analytical approaches in revealing the complex relationships between adversity and neurophysiology, suggesting that the intersubject similarity approach may offer unique insights in certain contexts.

The effect sizes of these results are relatively small, which is in line with well-powered brain-wide association studies (Marek et al., 2022). However, it is crucial to note that the presence of small effect sizes does not necessarily diminish the significance of these relationships, as even subtle effects can have meaningful long-term implications (Funder & Ozer, 2019). An important avenue for future research lies in extending these multivariate methods to investigate behavioral outcomes, exploring whether this approach can enhance the prediction of later functioning.

4.2. Limitations

One of the strengths of this study lies in its repeated-measures design, and future research would benefit from replicating this approach in diverse samples to strengthen the generalizability of these findings. It's important to note that the specific demographic composition of Lane County, Oregon, and the focus on adolescents assigned female at birth do impose limitations on the study's scope and its broader applicability. Though this community sample of typically developing adolescents allows us to explore the predictive capacity of adversity models on brain development in lower-adversity populations, it is important to acknowledge that this approach also constrains our ability to detect effects that might be more pronounced within higher-adversity populations.

Additionally, there are several limitations to this study's measurement of adversity. The study did not account for all possible dimensions of adversity, such as harshness and unpredictability, which could also have a significant impact on adolescent development (Brumbach et al., 2009; Ellis et al., 2022). Moreover, our measurement of deprivation was less extensive than of threat, and its refinement could involve the inclusion of additional facets of deprivation, such as medical, environmental, or educational neglect, along with an enhancement in the measurement of experiences related to physical and emotional neglect. Notably, once physical neglect was removed in one sensitivity analysis, inter-subject similarity in deprivation was no longer associated with inter-subject similarity in brain development. Therefore, further examination of this relationship using measures other than the Childhood Trauma Questionnaire would be valuable in determining the robustness of deprivation's impact on neural development.

It is also important to note that participants included in the analyses had lower neighborhood deprivation index scores than those excluded due to low scan quality, potentially introducing bias into our findings. Investigating these relationships in contexts of higher deprivation would enhance our understanding of these effects. Future directions may benefit by incorporating additional theories of stress and adversity, such as the social safety theory (Slavich, 2020) or the adaptive calibration model (Del Guidice et al., 2001), to guide both measurement and modeling approaches.

Lastly, it's important to note that these analyses, while focused on characterizing the effects of adversity on brain development, do not actively extend beyond the conventional deficit-based approach. They do not delve into exploring potential protective factors that may mitigate the negative effects of adversity (Fritz et al., 2018b; Hou et al., 2022; Malhi et al., 2019; Rutter, 2012), nor do they investigate the potential benefits or strengths that can emerge from

exposure to stress (Ellis, Abrams, et al., 2022; Frankenhuis et al., 2020). Future research should explore these facets for a more holistic and nuanced understanding of how childhood experiences impact development across diverse populations.

5. Conclusion

These findings add to the extensive and continually growing body of research on how adversity impacts brain development. This field has increasingly called for the incorporation of elements such as the developmental timing, chronicity, severity, and frequency when operationalizing adversity (Lacey & Minnis, 2019), which can help refine screening practices and progress towards individual-level resource support and care (Finkelhor, 2018; Loveday et al., 2022). Yet, one of the main advantages of simplifying adversity as a single composite score is the corresponding statistical ease and power (Evans et al., 2013). This novel application of IS-RSA exemplifies how multivariate approaches may be a promising opportunity to capture the heterogeneity and complexity in both adversity exposures and in brain development. Moreover, such multivariate approaches require far fewer participants to achieve adequate statistical power to detect brain-behavior associations (Makowski et al., 2023), offering a valuable methodological advantage.

In summary, the outcomes of this study emphasize the importance of embracing a dimensional framework when examining the impact of adversity on brain developmental trajectories. They also underscore the continued relevance of comprehensive exploration of the entire brain. A crucial avenue for future research will involve delineating the implications of these findings in the context of both adaptive and maladaptive behavior, offering a promising direction for further inquiry in this field.

CHAPTER IV: POSITIVE EXPERIENCE & BRAIN ASSOCIATIONS

1. Introduction

Although neurodevelopmental research has advanced our understanding of how adversity shapes the development of brain structure, function, and connection, much less attention has been given to the influence of positive experiences. This imbalance reflects a broader knowledge gap in the literature, and this chapter presents exploratory investigations into how both positive and negative childhood experiences contribute to structural change in adolescence, emphasizing the importance of understanding developmental processes in the context of the full range of childhood experiences.

1.1. Childhood Life Experiences and Brain Development

The brain's lengthy developmental timeline allows for considerable input and environmental adaptation (Berens et al., 2017). When exposed to adversity, that allowance spurs neural alterations that may prove maladaptive in more positive environments. Neuroimaging studies have identified links between adverse exposures and structural differences in the hippocampus, amygdala, and the prefrontal and parietal cortices (McLaughlin et al., 2019; Tottenham, 2020; Tottenham & Sheridan, 2010). Such structural neural differences at single time points have been shown in some studies to mediate the effects of adversity, mostly measured retrospectively, on internalizing symptoms (Frodl et al., 2017; S. K. G. Jensen et al., 2015; Popovic et al., 2020; Salokangas et al., 2021). Yet other studies yield conflicting evidence on the specific impact of adversity on the developing brain, resulting in a research literature pervasive with mixed effects (Gehred et al., 2021a; Haidl et al., 2023; Smith & Pollak, 2021a; Tottenham & Sheridan, 2010). A body of research on adolescence is emerging from the longitudinal

Adolescent Brain and Cognitive Development (ABCD) study, which allows researchers to examine a multitude of influences on the developing brain across the adolescent years, strengthening the implication of cross-sectional and single brain scan findings. For example, one study using data collected over two years from the ABCD study found that the number of adverse exposures assessed in youth aged 11-12 was associated with lower bilateral hippocampal volume at baseline (ages 9-10) as well as differences in volumetric change in two years after baseline assessment (Breslin et al., 2024).

Outcomes related to adverse exposures, including aspects of physiological development, are heterogeneous and difficult to predict, which is thought to be at least partially driven by differences in how adversity is conceptualized and operationalized. Areas of investigation have included examining effects of specific types of adversity, often by comparing exposed and non-exposed groups (Zeanah et al., 2003), and examining the effects of multiple adversities, often by summing together the presence or absence of different exposures (Evans et al., 2013b; Smith & Pollak, 2021b). These methods have provided important insight into individual risk factors as well as a cumulative risk factor. Dimensional models are thought to extend this work by more precisely parsing some of the outcome variability, as they propose that overarching elements of adverse exposures operate through specific biological pathways and can exert unique impact on outcomes (Ellis et al., 2009b; Ellis, Sheridan, et al., 2022; McLaughlin, Sheridan, & Lambert, 2014). They also incorporate additional important features of adversity, such as intensity, in addition to accumulation. Identified dimensions of current focus are unpredictability (Brumbach et al., 2009), threat, and deprivation. Threat is theorized to alter corticolimbic circuits (prefrontal cortices, amygdala, and hippocampus), while deprivation is theorized to alter frontoparietal regions (McLaughlin et al., 2019), though studies have also implicated deprivation in

corticolimbic circuitry (Gee, 2021). Unpredictability has also been proposed to operate through corticolimbic circuitry (Liu & Fisher, 2022), though the evidence for this theory is built primarily on animal research and behavioral studies, with increasing interest in examining its neurobiological effects in humans (Ellis, Sheridan, et al., 2022; Gee, 2021).

Given this sensitivity to the environment, it is reasonable to expect that when exposed to *positive* experiences, the brain also has potential to change in ways consistent with healthy development and positive well-being. Most investigations into how positive experiences influence brain development assess how various aspects of caregiving are associated with the developing brain, though this line of inquiry is far less explored compared to various adverse exposures (Farber et al., 2022). Several studies have investigated whether features of caregiving might buffer the effects of adversity or socioeconomic disadvantage on the developing brain. One study did not find a buffering effect when examining caregiver support and early adversities, prospectively measured in preschool, in relation to structural brain development in childhood (Luby et al., 2019), though another did identify that observations of positive parenting with adolescent boys reduced the effect of neighborhood socioeconomic disadvantage on the structural development of the dorsal frontal and lateral orbitofrontal cortices, as well as the amygdala (Whittle et al., 2017).

1.2. Interaction of Positive Experiences and Adversity

New measures of positive experiences have sparked growing interest in how the accumulation of positive experiences, rather than individual ones, affect development (Bethell et al., 2019; Morris & Hays-Grudo, 2023; Narayan et al., 2018; Narayan, Merrick, et al., 2023). Relatedly, many have begun to investigate how positive experiences might exert developmental influence when adversity has also occurred (Han et al., 2023; Kallapiran et al., 2025). Positive

experiences (e.g., predictability, safety, support, and enjoyment) during childhood often occur independently of adverse experiences, and evidence supports their contribution to health outcomes alongside adverse experiences (Han et al., 2023). To date, the vast majority of these cumulative positive experience measures have been collected retrospectively in adulthood.

Though these new, comprehensive measures have not yet been used to understand neural development, other work has examined the range of life experiences in relation to adolescent neural development. For example, in one analysis from the ABCD dataset, researchers revealed 14 robust patterns of covariation linking environmental, psychosocial, and individual factors with brain structure and function in youth aged nine to ten (Modabbernia et al., 2021). These patterns reflected diffuse associations across neural modalities (i.e., global measures of morphometry, myelination, white matter integrity, and functional connectivity), where various factors converged into patterns of positive and negative exposures. Such patterns captured relative advantages and disadvantages across aspects of neighborhood quality, parental characteristics, family functioning, anthropometrics, cognition, and psychopathology. The cross-sectional findings underscore the importance of capturing the co-occurrence of both advantageous and disadvantageous experiences and environments. They also highlight the potential of multivariate approaches in characterizing brain-environment relationships.

1.3. The Present Study

To date, no study has evaluated whether cumulative positive experiences relate to brain development alone or when controlling for the effects of adversity. This study aims to build upon emerging work by examining whether individual differences in positive and adverse experiences are patterned similarly to individual differences in adolescent structural brain changes across regions associated with adverse exposures. Experience measurements in primary analyses

include early measurement of cumulative and recent adversities and, given its relative breadth, later measurement of lifetime positive experiences. Follow-up analyses examine later measurement of cumulative adversity and early measurement of positive experiences related to social support. These analyses build off the published study examining how interindividual differences in adversity relate to brain development (Barrett et al., 2024). Here, I explore whether:

1. Individual differences in positive experiences are correlated with individual differences in structural brain changes
2. Identified associations persist when controlling for differences in adverse exposure
3. Timing and measurement differences affect these associations, despite the imperfect matching of measures over time.

I continue the multivariate approach method used in the investigation of adversity and brain development, as multivariate approaches generally have the potential to improve the reliability of effects even in smaller samples (Genon et al., 2022; Marek et al., 2022; Spisak et al., 2023).

2. Method

2.1. Sample Characteristics

This project uses up to sixth waves of data gathered through the Transitions in Adolescent Girls* (TAG; *not all study participants identify as girls) study and a seventh time point of data collected through a substudy with the sample (Barendse et al., 2020). Study recruitment occurred in 2015 and was aimed towards students registered as female in Lane Country, Oregon, USA who were aged 10 to 13 years of age. The study was approved by the University of Oregon Institutional Review Board, and assent and informed consent were

obtained from the adolescents and their caregivers, respectively, before inclusion in the study. Exclusionary criteria included included parent-reported diagnosis of select developmental disabilities (i.e., an autism spectrum disorder, cerebral palsy, or intellectual disability), diagnosis with a psychotic disorder, use of psychotropic medication other than stimulants, reported or suspected pregnancy, claustrophobia, and presence of ferromagnetic material in the body that would prohibit magnetic resonance scanning. 189 adolescents and their parents were initially recruited, and 174 passed exclusionary criteria and participated in the first wave of the study.

The sample includes a more diverse distribution of racial and ethnic identities than the surrounding community; a detailed breakdown is presented in Table 18. Table 19 also includes information on participants' gender identities, which reflect changes in identification across study waves. These social identities are fluid and may evolve over time. Notably, we did not disproportionately lose participants from racial or ethnic minority backgrounds due to attrition.

Table 18

Race and Ethnicity

Race	Non-Hispanic/Latina/Chicana	Hispanic/Latina/Chicana	Percent Non-Hispanic/Latina/Chicana	Percent Hispanic/Latina/Chicana
Black	0	1	0.00%	0.57%
Asian	2	0	1.15%	0.00%
NAAN	1	0	0.57%	0.00%
White	118	20	67.82%	11.49%
White and NAAN	9	2	5.17%	1.15%
White and NHPI	0	1	0.00%	0.57%
White and Middle Eastern	1	0	0.57%	0.00%
White and Asian	8	0	4.60%	0.00%
White and Asian and NHPI	0	1	0.00%	0.57%
White and Asian and NAAN	1	0	0.57%	0.00%
Black and White	8	0	4.60%	0.00%
Not Specified	0	1	0.00%	0.57%

Note. 32.2% of participants identified as a racial or ethnic minority. Comparatively, 18.2% of the local county identifies as a racial or ethnic minority. NAAN = Native American and Alaskan Native; NHPI = Native Hawaiian and Pacific Islander.

Table 19

Gender Identification across Five Waves

	Wave 1	**Wave 2**	**Wave 3**	**Wave 4**	**Wave 5**
Clinician-Administered Measure	% (N)	% (N)	% (N)	% (N)	% (N)
Female	98.35% (179)	96.93 (158)	93.57% (131)	88.39% (99)	85.71% (84)
Male	0.00% (0)	0.00% (0)	0.71% (1)	1.79% (2)	2.04% (2)
Other	1.65% (3)	2.45% (4)	5.71% (8)	9.82 (11)	12.24% (12)
Total N	182	162	140	112	98

Note. Gender identity as self-reported to a clinical interviewer. Sample size at Wave 1 exceeds 174 due to the collection of gender data from several adolescents prior to study exclusion.

Each wave of data collection took place approximately 18 months apart (range of 0.72-36.65 months due to pandemic-related disruptions). Each participant completed two laboratory

visits at each wave, spaced approximately one month apart. Out of 174 participants in the first wave, 163 (93.68%) participants participated in the second wave, 150 (86.21%) participated in the third wave, 109 (62.64%) completed the fourth wave, 100 completed the fifth wave (57.47%), and as of the time of preparing this manuscript, 59 completed the sixth wave (33.91%), which is still ongoing. Questionnaires were completed at the first and second visits, and home; the MRI scan was completed during the second visit. A subsample of TAG participants (N=62) participated in an additional MRI scan for the SOS substudy that occurred largely between waves two and three (four TAG participants completed the substudy between waves three and four).

Structural scans from two to five timepoints (depending on image quality) were used to measure cortical thinning and subcortical volumetric change. Participants who only had a high-quality scan from fewer than two timepoints were excluded from these analyses.

Prior analyses with these data examined relationships among two of the proposed study variables: adversity and structural brain change (Barrett et al., 2024). This study extends that work by integrating positive childhood experiences to understand how experiences relate to brain development.

2.2. Measures

2.2.1. Positive Childhood Experiences

2.2.1.1. Benevolent Childhood Experiences (BCEs) Scale

The BCEs scale (Narayan et al., 2018) includes ten binary items that assess if adolescent participants have experienced components of safety, support, predictability, and enjoyment during their childhood. Items were summed with the PCEs (see below) to

create a total score of positive experiences, such that higher scores indicate more positive experiences.

2.2.1.2. Positive Childhood Experiences (PCEs) Scale

The PCEs were seven binary items adapted by Bethell et al. (2019) that assess if adolescent participants have had specific positive interpersonal experiences, such as being able to talk to family about one's feelings, enjoying participating in community traditions, and feeling safe and protected by an adult at home. Items were summed with the BCEs scale to create a total score of positive experiences.

2.2.1.3. Social Belonging

Adolescents completed four items adapted from Yeager and colleagues' work (2016) assessing perceived social belonging (e.g., "I feel like I belong in my school/my social group at school," "I feel respected in my school/my social group at school"). Items were rated on a 5-point scale ranging from 1 (completely true) to 5 (not true at all). Scores were summed, with higher total scores indicating lower levels of perceived belonging.

2.2.1.4. Multidimensional Scale of Perceived Social Support (MSPSS)

When completing the MSPSS, adolescents indicate the degree (on a scale from 1 to 7; 1 = very strongly disagree and 7 = very strongly agree) that they have support from family, friends, and a significant other in the face of challenges (Zimmet et al., 1998). Items are summed into subscales for each familial support, friend support, and a significant other support, as well as summed into a total score.

2.2.2. Adverse Childhood Experiences

2.2.2.1. Childhood Trauma Questionnaire (CTQ)

We administered the CTQ form to adolescent participants with 32 items, but only the standard 25 items from the validated short form were used for these analyses. It has five subscales: physical abuse, emotional abuse, sexual abuse, emotional neglect, and physical neglect. The CTQ has demonstrated high convergent validity (Bernstein et al., 1994, 1997) with therapist ratings and trauma interviews and exhibits high levels of internal consistency across age groups (Bernstein et al., 2003; Scher et al., 2001; Spinhoven et al., 2014). Initial test-retest reliability coefficients vary between .79 and .86 across 2-6 months (Berenstein et al., 2003) and .44 to .67 across five years (Cammack et al., 2016).

2.2.2.2. Socioeconomic Status (SES) Measures

We used a single-item caregiver report on whether their child received free lunch at school since both household income and family size influence eligibility. We also included caregiver education (highest report if a participant had more than one caregiver) which indicates if caregivers received no high school diploma (or equivalent); high school diploma (or equivalent); some college credit (no degree), trade, technical, or vocational training; associate degree, undergraduate degree, or postgraduate degree. Finally, we used participants' residential addresses to create an index of neighborhood deprivation based on the computation provided by Messer et al. (2006) that incorporates census data on income, education, employment, housing, and occupation.

2.2.2.3. Youth Risk Behavior Scale (YRBS)

Two items from the YRBS were used to assess if the participant had ever been bullied on school property or electronically (i.e., through email, chat rooms, instant messaging, websites, or texting). Data was also summed to provide one composite measurement.

2.2.2.4. Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS)

The following events evaluated in the K-SADS Post-Traumatic Stress Disorder module were used to assess further experiences of trauma: a significant car accident that resulted in injury and required medical intervention, other accident that resulted in injury and required medical intervention, witnessed a fire that caused significant property damage or moderate-severe physical injuries, witnessed a natural disaster that caused significant devastation, witness to threatening or violent crime, a victim of threatening or violent crime, terrorism-related traumatic event, war zone traumatic event, witness to domestic violence, physical abuse, and sexual abuse. These events were also summed to create a composite score.

2.2.2.5. *Adverse Childhood Experiences (ACEs) Questionnaire*

The ACE questionnaire is a widely-used index of negative experiences in childhood (Felitti, et al., 1998). Adolescent participants responded to ten binary items related to abuse, neglect, parental separation or divorce, parental substance use, and household member challenges (e..g, problematic substance use, time in prison). The items were summed for a total score where higher scores indicate higher adverse experiences.

2.2.3. Structural Brain Scan

Prior to entering the 3T Siemens Skyra MRI scanner, participants completed a mock scan

to help ease any scanner-induced anxiety. Participants watched a short, animated video while acquiring a T1-weighted 3D MP-RAGE structural scan. 176 slices of 1 mm isometric voxels were collected over 5 minutes and 59 seconds (FOV = 256 mm, TR = 2500 ms, TE = 3.41 ms, flip angle = 7 degrees).

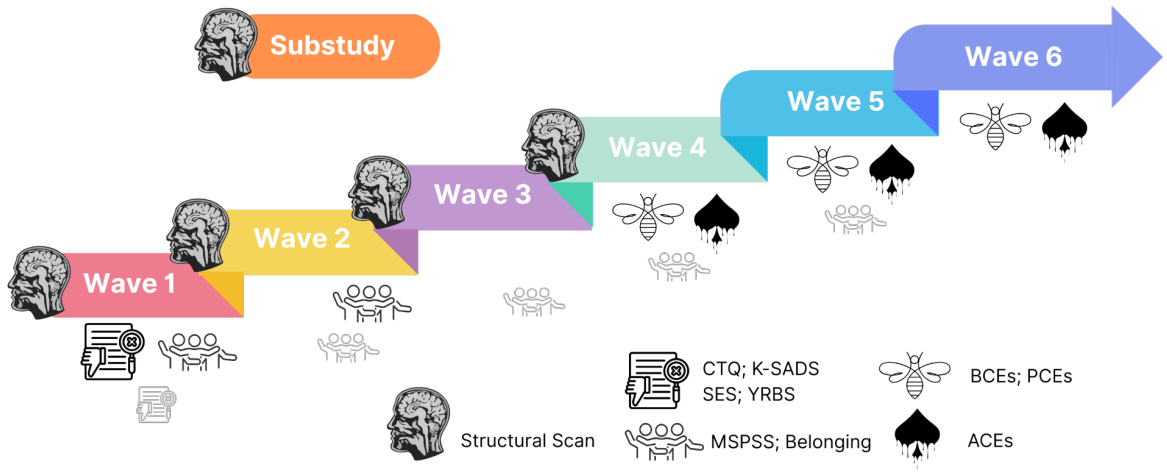
2.3. Procedure

Figure 24 displays the measures from the TAG study that were used for these analyses. For the primary analyses, adversity measurements–including CTQ data, K-SADS items, SES indicators, and the YRBS bullying items–were drawn from Wave 1. Measurements of cumulative positive experiences across childhood (i.e., BCEs and PCEs) were used from either Wave 4, 5, or 6, depending on availability. Brain imaging data were drawn from Waves 1 to 4 and the substudy that occurred generally between Waves 2 and 3. The general age range for these measurements were as follows: Wave 1 adversity data were collected between ages 10–13; later-wave measures (e.g., positive experiences) were collected between ages 15–20; and brain scans were largely collected between the former measurement points, with first scans occurring between ages 10–15 and final scans between ages 12–18 (see Figure 25).

For follow-up analyses, ACEs questionnaire data encompassing childhood adversities up through assessment time, were also drawn from Wave 4, 5, or 6, and the measurement of social belonging and MPSS data were drawn from either Wave 1 or 2, depending on Wave 1 data availability.

Figure 24

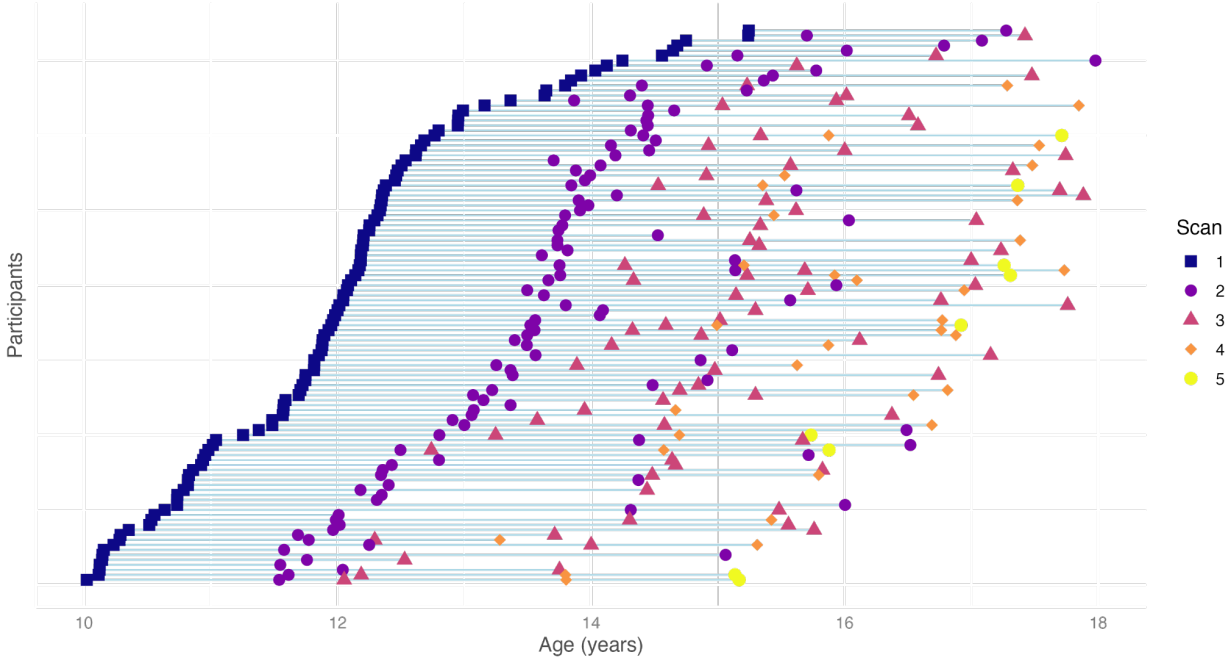
TAG Study Design: Key Timepoints and Measures Used in Analyses



Note. Semi-transparent icons represent data used as predictors for multiple imputation but not included in analyses.

Figure 25

Age at Each MRI Scan



Note. Each line represents each participant's age at the MRI session across all time points included in these analyses.

2.4. Data processing

2.4.1. Neural Data

The neural data was processed identically to the procedure reported in Barrett et al. (2024) and is as follows. Preprocessing of the structural scans, including the initial steps of skull stripping (Ségonne et al., 2004) and intensity correction (Sled et al., 1998), were completed with the FreeSurfer (v 7.2.0) imaging analysis suite (http://surfer.nmr.mgh.harvard.edu/) for all waves. This automatic process reconstructs models of the pia surface and gray and white matter boundary after segmentation (Fischl et al., 2002; Fischl et al., 2004), tessellation, and topological correction (Fischl et al., 2001; Ségonne et al., 2007).

Two trained coders visually inspected anatomical scans for artifacts associated with participant motion (i.e., blurring or ringing). Motion artifacts can negatively bias cortical estimates, necessitating a rigorous and transparent quality control process (Backhausen et al., 2016). The scans were rated on a four-point scale for evidence of motion, with 4 representing major amounts of motion disturbances, 3 indicating moderate amounts of visible motion in multiple areas or across a number of slices, 2 indicating minor motion disturbances across a few slices, and 1 indicating a sharp image with no visible motion artifact. Scans were also being rated for accurate white and pial surface boundaries on a four-point scale, with 4 indicating missing or entirely inaccurate surface boundaries, 3 indicating inaccurate boundaries in multiple areas, 2 indicating poor boundaries in limited areas, and 1 reflecting good surface boundaries. To diminish the impact of motion artifacts, scans that received scores of 3 or higher for the motion or surface rating were excluded from analyses. Scans that received scores of 2 for either rating were further inspected to ensure that regions specific to these analyses were not visibly affected.

After completing manual quality control, 111 participants were deemed eligible for

inclusion in these analyses by having at least two time points of usable neuroimaging data. Of the included participants, 33 had two usable scans, and 78 had three or more usable scans. Of those excluded participants, just over half (n = 36) had only one high-quality scan across waves.

After the initial quality inspection, scans were run through FreeSurfer's longitudinal pipeline (Reuter et al., 2012), and scans that received a 3 or 4 rating on surface boundaries were re-examined to assess whether surface reconstruction improved after using an unbiased within-subject template which informs the standard processing steps. Cortical thickness was estimated using the smallest distance between the gray and white matter boundary and the gray and pial surface boundary at each vertex (Fischl & Dale, 2000). Cortical thinning estimates were estimated by calculating the annualized percent changes in cortical thickness for individuals with at least two timepoints of usable scans. We used the same equation as Mills et al. (2021) to calculate the annualized percent change for each region between each scan:

$$\frac{\left(\left(\frac{Brainmeasurement_{x+1} - Brainmeasurement_{x}}{\left(\left(Brainmeasurement_{x} + Brainmeasurement_{x+1}\right)/2\right)}\right) \times 100\right)}{Age_{x+1} - Age_{x}}$$

Annualized percent change scores were then averaged for participants with three or more usable scans to create a single change score.

The following regions of interest (ROIs) examined in this study were chosen to reflect regions in the fronto-amygdala and hippocampal circuit and frontoparietal network described in McLaughlin et al.'s 2019 review: amygdala, hippocampus, rostral anterior cingulate cortex, medial orbitofrontal cortex, rostral middle frontal cortex, and superior parietal cortex. We used FreeSurfer's anatomical masks for the amygdala and hippocampus. Cortical regions were defined by selecting these parcellations from the Desikan-Killiany atlas, given its use in the relevant literature and theoretical specificity.

2.5. Missing Data

As this work extends the analyses in Chapter III, the adversity measures used were the same that had been imputed in Chapter III. That process included nearest-wave substitutions for high amounts of missing data (more than three items missing within the same subscale). Lower amounts of data were imputed with the missforest algorithm (Stekhoven & Bühlmann, 2012) using the following predictors: age, race and ethnicity, caregiver education, caregiver income, school of attendance, sum of traumatic life events (for CTQ imputation), and CTQ total score (for K-SADS imputation). Counts of substitutions and imputations can be found in Table 15 of Chapter III.

To address missingness in the later-wave measurements (i.e., BCEs, PCEs, and ACEs questionnaires), mode imputation was first applied to the small proportion of missing data in four items (0.9% of item data). Out of 111 participants with brain data included in these analyses, 77 had completed the later-wave questionnaires. Independent samples *t* tests comparing participants who had completed the later-wave questionnaires to those who had not (n = 34) revealed differences in half of the brain ROIs; for six out of twelve ROIs, those who completed the questionnaires showed less annualized structural change. Due to these retention effects, missing later-wave questionnaire data were imputed for those 34 participants using multivariate imputation by chained equations, which accounts for uncertainty related to missingness (MICE; (Van Buuren, 2018)). Predictor variables for imputation were selected by calculating two correlations for each target–predictor pair: one between observed values and another between the response indicator and predictor values. Predictors were retained if the larger of these absolute correlations exceed $r = .20$. The predictive mean matching method was used for all variables. The imputation model included the most recently completed social belonging and MSPSS

measurements, Wave 1 adversity composite scores (CTQ total, bullied composite, K-SADS PTSD item total, and free lunch), annualized percent change in each ROI, and social belonging and MPSS data from Wave 1 (or Wave 2 if Wave 1 was missing; see Table S2 in the Supplementary Information for the number of Wave 2 substitutions per item). One participant failed imputation and was excluded, resulting in a final analytic sample of $n = 110$ within five imputed data sets.

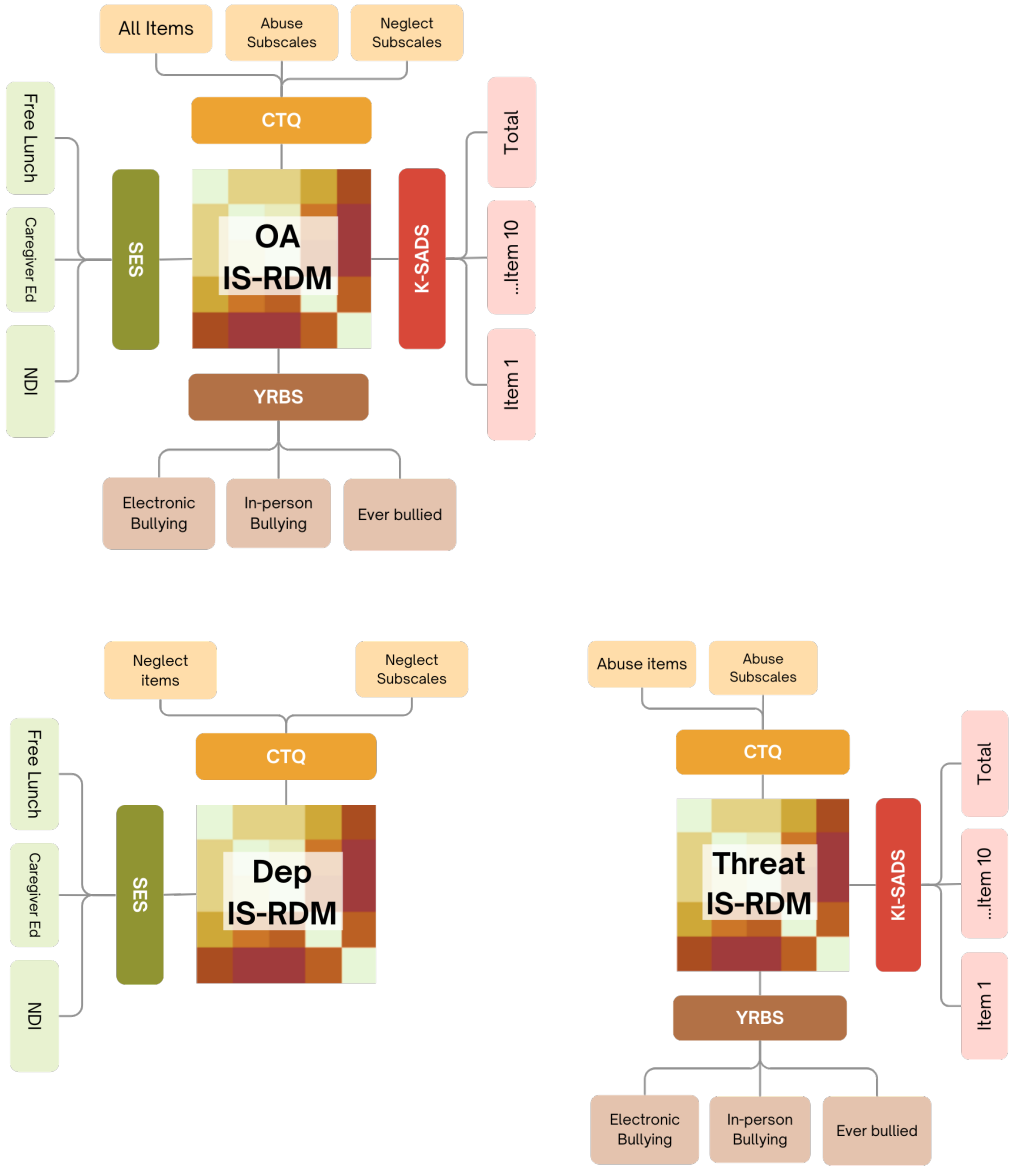
2.6. Analyses

To assess whether inter-individual differences in childhood experiences relate to coordinated or discoordinated structural brain development, I used inter-subject representational similarity analysis (IS-RSA) for this study. IS-RSA is a multivariate framework that characterizes dissimilarity between participants by constructing inter-subject representational dissimilarity matrices (IS-RDMs), which contain pairwise Euclidean distances between participants based on constructs of interest. These square, symmetric matrices reflect all possible participant comparisons and preserve the full multivariate structure of the input data.

IS-RDMs were constructed for adversity-related constructs derived from Wave 1 data: threat (Threat IS-RDM), deprivation (Deprivation IS-RDM), and overall adversity (OA IS-RDM), as illustrated in Figure 26. These adversity data were transformed so that experiences that were measured with a greater number of items (e.g., physical abuse) did not have greater weight when calculating dissimilarity than others. Items were divided by the number of items per experience (e.g., each of the five physical abuse items were divided by five). All items were transformed to a percent of the maximum score so that continuous variables were not weighted more heavily than categorical variables.

Figure 26

Grouping of Adversity Data in IS-RDMs



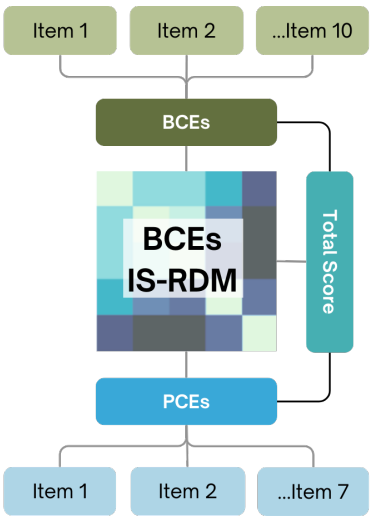
Note. The overall adversity (OA) inter-subject representational dissimilarity matrix (IS-RDM) represents all adversity measures. The Threat IS-RDM represents abuse items and subscales, trauma items and total, and bullying items. The Deprivation (Dep) IS-RDM represents neglect items and subscales, free lunch, caregiver education, and neighborhood deprivation index (NDI).

An additional construct of interest was benevolent childhood experiences (BCEs IS-

RDM), calculated using later-wave BCE and PCE items and their total score (Figure 27). Follow-up analyses incorporated an IS-RDM representing Wave 1 positive experience data—namely, belonging items and MSPSS items, subscales, and total score—as well as an IS-RDM representing later-wave ACEs data (individual items and sum score).

Figure 27

Construction of Positive Experience Data in IS-RDM



Note. The positive experiences inter-subject representational dissimilarity matrix (BCEs IS-RDM) represents all retrospectively collected BCEs and PCEs items as well as their joint total score.

Structural brain development dissimilarity (Brain IS-RDM) was calculated using the Euclidean distances between participants' average annualized percent change across ROIs: amygdala, hippocampus, rostral anterior cingulate cortex, medial orbitofrontal cortex, rostral middle frontal cortex, and superior parietal cortex.

All IS-RDMs were vectorized below the diagonal, standardized, and included in linear

regression models. IS-RDMs were calculated separately for each of the five multiply imputed data sets; regression models were run within each set and results were pooled using Rubin's rules ((Rubin, 1987)). Multivariate Wald (D1) tests were used for nested model comparisons.

Because pairwise similarity data violate independence assumptions, additional non-parametric tests were conducted, similar to Mantel permutation tests. To test individual predictors for each imputed data set, one IS-RDM was randomly sampled and regression models were rerun 2,000 times, yielding 10,000 parameter estimates across imputations. The observed model parameter was compared to this null distribution to generate permuted *p*-values. To provide a conservative test of interaction effects, both IS-RDMs were randomly sampled and their interaction estimate was compared to the null distribution. Permuted p-values were corrected for false discovery rate within each conceptually distinct set of model tests using the Benjamini-Hochberg (BH) procedure (Benjamini & Hochberg, 1995).

3. Results

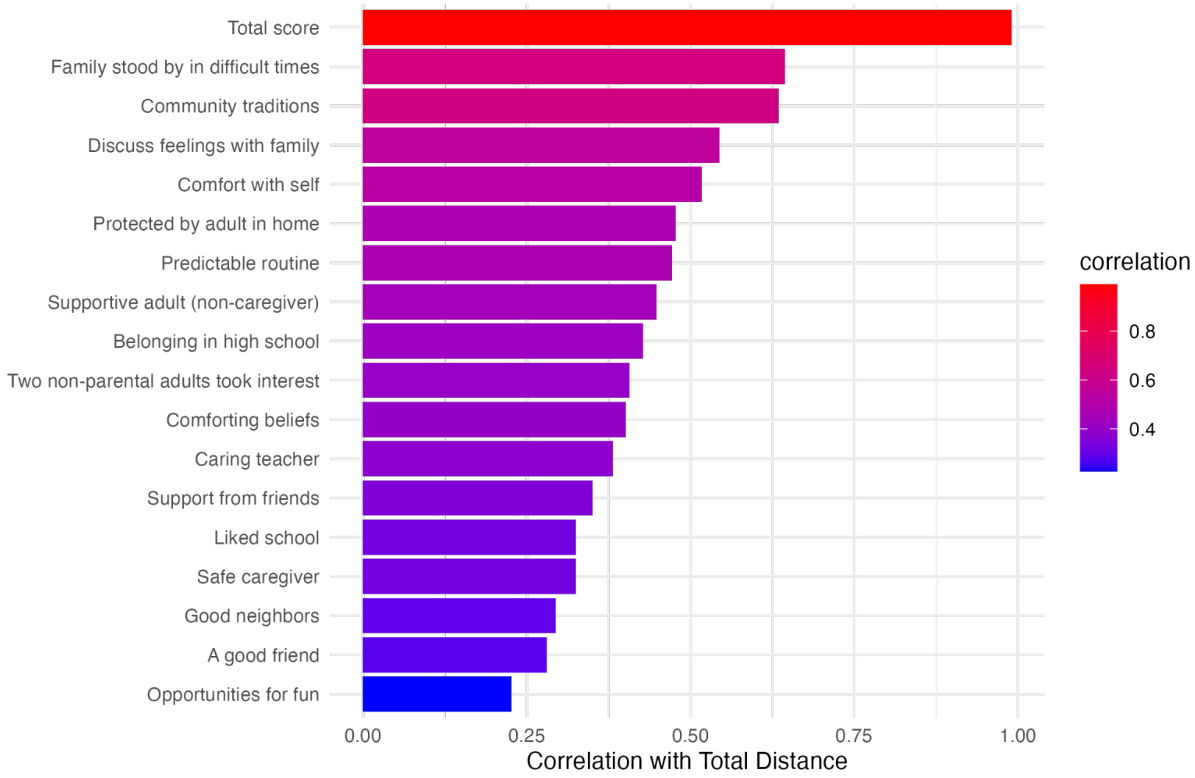
3.1. Drivers of Dissimilarity in Benevolent Childhood Experiences

Figure 28 displays the contribution of each item in the BCEs questionnaire to the full BCEs IS-RDM. To calculate those contributions, I computed an IS-RDM for each individual variable separately (i.e., one-dimensional Euclidean distances) and calculated the Pearson correlation between the vectorized lower triangle of the matrix of the individual IS-RDMs with the full BCEs IS-RDM. Higher correlations indicate greater alignment between variable-specific distances and the overall pattern of dissimilarity for all included items among all participants. This approach allows for the identification of which variables most strongly reflect the structure captured by the full RDM.

Distances according to the total score are almost completely correspondent with the full RDM. Individual items that contain the most distance among participants (i.e., are correlated over .5 with the full RDM) include feeling that one's family stood by oneself during difficult times, having enjoyed participating in community traditions, having been able to discuss feelings with family, and having liked or felt comfortable with oneself.

Figure 28

Contribution of Individual Variables to BCEs IS-RDM



Note. Distances in each positive experience variable correlating with the BCEs IS-RDM. PCE items are part of the positive experiences scale. BCE items are part of the BCEs scale.

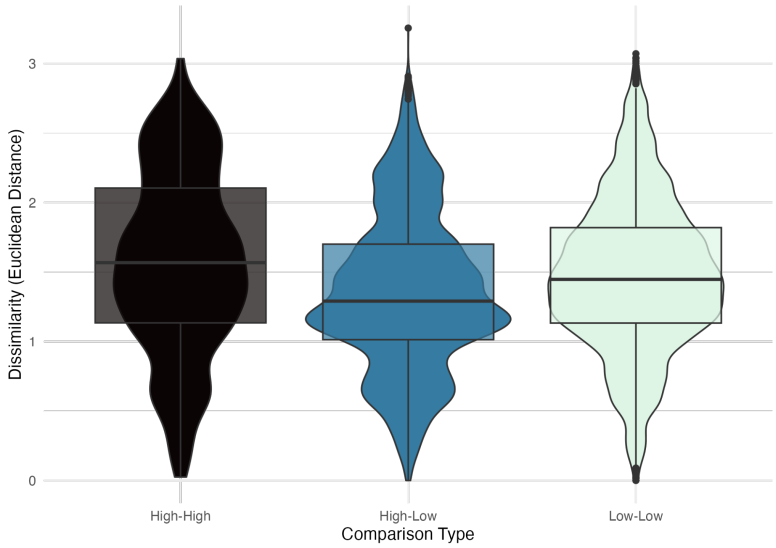
3.2. Drivers of Dissimilarity in Adverse Childhood Experiences

The adversity dissimilarity matrices were based on a relatively heterogeneous set of

measures, making it unclear which specific variables contributed most to the highest distances. It was also ambiguous whether greater dissimilarity tended to occur between individuals with overall high versus low levels of adversity, or between individuals who both had high adversity scores but experienced qualitatively different types of adversity. To contextualize these dissimilarity patterns, I created a total adversity score by calculating the mean across all adversity-related variables and divided participants into high and low adversity groups. Those in the top 25% of adversity scores were classified as the high adversity group, while the remaining 75% comprised the low adversity. Each pairwise comparison was thus categorized as high-high, high-low, or low-low. Figure 29 displays the distribution of dissimilarity scores for each of these comparison types. I also examined the 5% of interpersonal pairs with the highest dissimilarity to identify which groupings contributed most to these extreme values. The majority were high-low comparisons (n = 236), followed by some high-high comparisons (n = 70); none were low-low.

Figure 29

Dissimilarity by Comparison Type



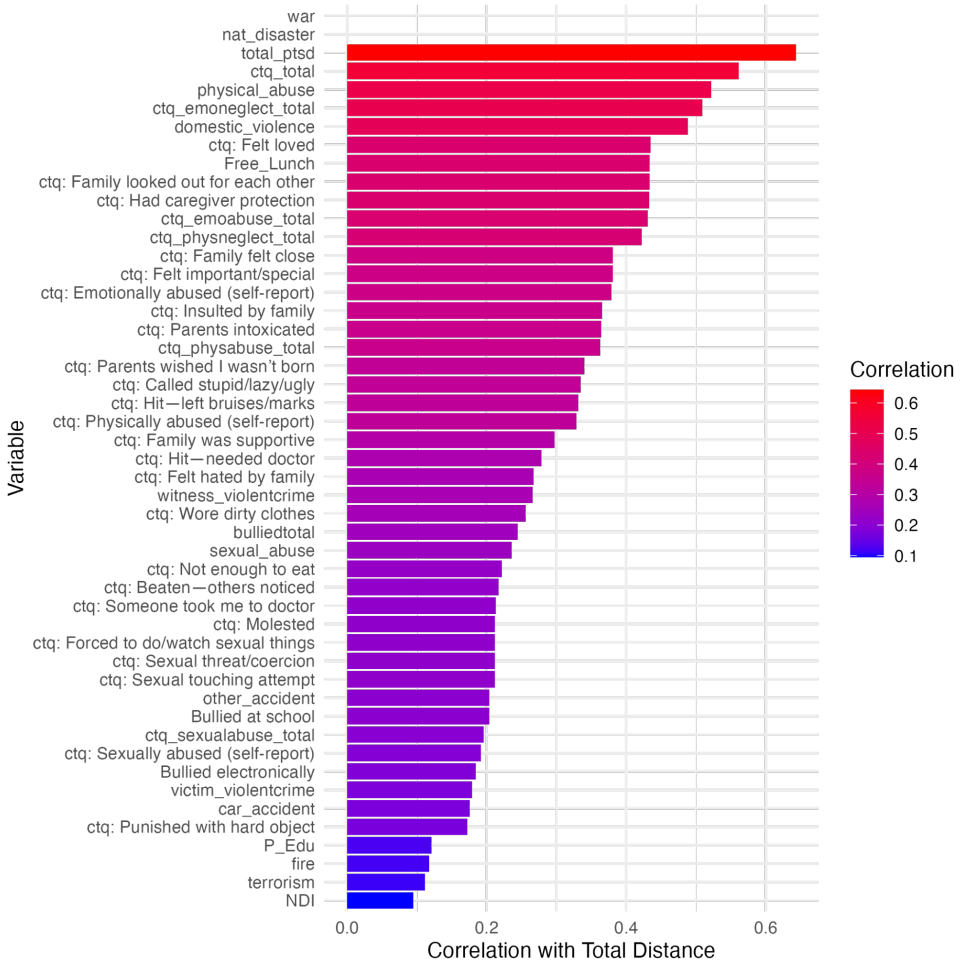
Note. Boxplots show that dissimilarity mean was largely similar across adversity comparison

types, though the high-high comparisons contained most of the highest distance scores.

I also investigated how each variable individually contributed to the Overall Adversity (OA) IS-RDM. To calculate those contributions, I computed an IS-RDM for each individual variable separately (i.e., one-dimensional Euclidean distances) and calculated the Pearson correlation between the vectorized lower triangle of the matrix of the individual IS-RDMs with the full OA IS-RDM. Higher correlations indicate greater alignment between variable-specific distances and the overall pattern of dissimilarity for all included items among all participants. This approach allows for the identification of which variables most strongly reflect the structure captured by the full IS-RDM, as shown in Figure 30. Top variable contributors to the OA IS-RDM include total exposure to trauma (as endorsed in the K-SADS interview PTSD module), CTQ total and the physical abuse and emotional neglect subscale scores, and exposure to domestic violence. Variables that were least represented by the OA IS-RDM included caregiver education, experiencing trauma related to fire, terrorism, and NDI. Two items (experiencing war and natural disaster) had no variance, and consequently did not correlate with the OA IS-RDM.

Figure 30

Contribution of Individual Variables to Overall Adversity (OA) IS-RDM



Note. Distances in each adversity variable correlating with the overall adversity (OA) IS-RDM.

3.3. Benevolent Childhood Experiences and Brain Dissimilarity

Full model results are presented in Table 19. The BCEs IS-RDM showed a direct positive relationship with the dissimilarity in brain development (visualized in Figure 31), even when accounting for all representations of adversity. None of the adversity constructs significantly moderated the relationship between BCE similarity and brain similarity in the linear models according to null hypothesis significance testing, though the interaction between the BCEs IS-RDM and the Deprivation IS-RDM did meet significance criteria with the permutation test. Though this interaction was a small effect, a D1 model comparison supported adding that

interaction to that model (F[2, 9.7, 5990] = 7.42, p = .011).

Table 20

BCEs & Brain Change Dissimilarity: Pooled Model Results

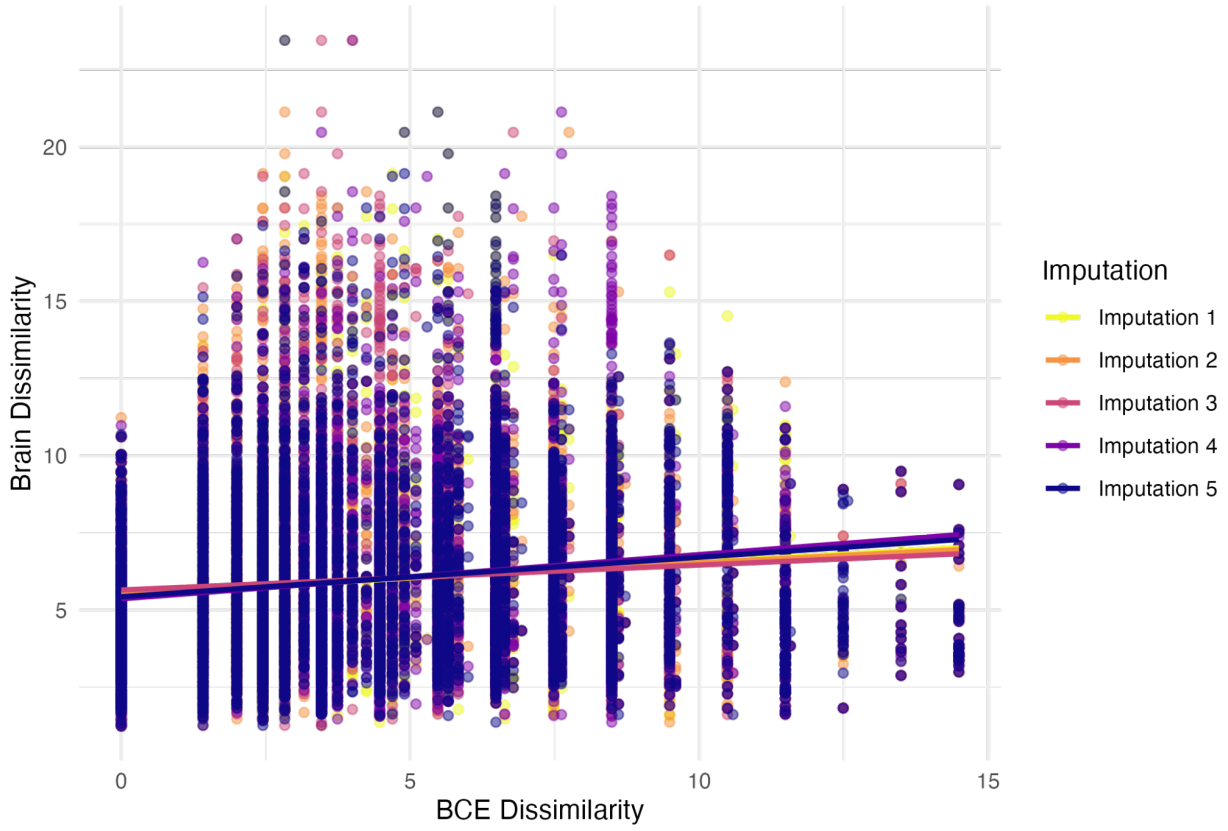
	Main Effect	OA	BCEs x OA	Dimensions	Threat x Dep	BCEs x Threat	BCEs x Dep
Intercept	0.000 (0.013)	0.000 (0.013)	0.001 (0.014)	0.000 (0.013)	-0.029* (0.013)	-0.002 (0.013)	0.009 (0.014)
	p = 1.000	p = 1.000	p = 0.964	p = 1.000	p = 0.028	p = 0.881	p = 0.538
BCEs	0.117* (0.033)	0.092* (0.032)	0.092* (0.031)	0.098* (0.031)	0.090* (0.032)	0.099* (0.032)	0.103* (0.029)
	p = 0.013	p = 0.031	p = 0.028	p = 0.020	p = 0.031	p = 0.023	p = 0.012
Overall Adversity (OA)		0.149*** (0.014)	0.149*** (0.015)				
		p = <0.001	p = <0.001				
Threat				0.162*** (0.015)	0.145*** (0.015)	0.160*** (0.015)	0.166*** (0.015)
				p = <0.001	p = <0.001	p = <0.001	p = <0.001
Deprivation (Dep)				0.020 (0.015)	0.021 (0.015)	0.019 (0.015)	0.018 (0.015)
				p = 0.182	p = 0.152	p = 0.230	p = 0.241
Interaction			-0.004 (0.031)		0.099*** (0.012)	0.019 (0.025)	-0.043 (0.025)
			p = 0.914		p = <0.001	p = 0.474	p = 0.128
R2	0.014	0.036	0.036	0.043	0.053	0.043	0.045
R2 Adj.	0.014	0.035	0.036	0.042	0.052	0.043	0.044
Num.Obs.	5995	5995	5995	5995	5995	5995	5995
Permuted Term	BCEs	OA*BCEs	BCEs	BCEs	Threat x Dep	BCEs x Threat	BCEs x Dep
Permuted p-value	<.001***	<.001***	0.166	<.001***	<.001***	0.144	<.001***
Adjusted p-value	<.001***	<.001***	0.166	<.001***	<.001***	0.166	<.001***

Note. Standardized estimates for regression models where outcome is structural brain change dissimilarity are shown in each column. Estimates are standardized. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation

information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. BCEs = retrospective positive childhood experiences. Dep = deprivation.

Figure 31

Association between BCEs and Brain Dissimilarity across Multiple Imputations



Note. Dissimilarity in retrospectively-reported positive experiences demonstrate a positive association with dissimilarity in structural brain development across imputed datasets.

Note: Scatterplot showing the relationship between behavioral childhood experiences (BCE) dissimilarity and brain dissimilarity across five multiple imputations. Trend lines represent linear fits for each imputed dataset.

3.4. Adverse Childhood Experiences and Brain Dissimilarity

The ACEs and BCEs IS-RDMs were moderately negatively correlated (r = –.51). The ACEs IS-RDM was positively correlated with the Overall Adversity, Threat, and Deprivation IS-RDMs (r = .33, .25, and .31, respectively). Full model results are presented in Table 20. The ACEs IS-RDM showed no significant relationship with the dissimilarity in brain development, either alone, or when controlling for BCEs or the earlier-measured Overall Adversity IS-RDM.

Table 21

ACEs & Brain Change Dissimilarity: Pooled Model Results

	Main Effect	BCEs	OA	ACEs x BCEs
Intercept	0.000 (0.013)	0.000 (0.013)	0.000 (0.013)	0.035 (0.018)
	p = 1.000	p = 1.000	p = 1.000	p = 0.079
ACEs	0.081 (0.034)	0.048 (0.037)	0.030 (0.039)	0.063 (0.035)
	p = 0.061	p = 0.253	p = 0.474	p = 0.129
BCEs		0.101* (0.039)		0.112* (0.038)
		p = 0.046		p = 0.031
Overall Adversity (OA)			0.155*** (0.018)	
			p = <0.001	
BCEs x ACEs				-0.104 (0.041)
				p = 0.052
R2	0.007	0.016	0.029	0.029
R2 Adj.	0.006	0.016	0.028	0.028
Num.Obs.	5995	5995	5995	5995
Permuted Term	ACEs	ACEs	ACEs	ACEs*BCEs
Permuted p-value	<.001***	<.001***	0.017*	<.001***
Adjusted p-value	<.001***	<.001***	0.017*	<.001***

Note. Standardized estimates for regression models where outcome is structural brain change dissimilarity are shown in each column. Estimates are standardized. Standard errors are shown in parentheses. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. BCEs = retrospective positive childhood experiences. ACEs = retrospective adverse childhood experiences.

3.4. Wave 1 Positive Experiences and Brain Dissimilarity

W1 BCEs and later-wave BCEs IS-RDMs were positively correlated (r = .24). Full model results are presented in Table 21. The W1 BCEs IS-RDM showed a direct positive relationship with the dissimilarity in brain development, even when accounting for all representations of adversity. Moreover, the effect of the W1 BCEs IS-RDM on the Brain IS-RDM was moderated by both the Overall Adversity (OA) IS-RDM and the Threat IS-RDM, though not by the Deprivation IS-RDM. D1 model comparison supported the addition of significant interactions to the models between W1 BCEs IS-RDM and the OA IS-RDM (F[1, 4, 5991] = 12.24, p = .025), as well as the Threat IS-RDM (F[1, 4, 5990] = 14.64, p = .019).

Table 22

Wave 1 Positive Experiences & Brain Change Dissimilarity: Pooled Model Results

	Main Effect	OA	W1 BCEs x OA	Dimensions	Threat x Dep	W1 BCEs x Threat	W1 BCEs x Dep
Intercept	0.000 (0.013)	0.000 (0.013)	-0.003 (0.013)	0.000 (0.013)	-0.030* (0.013)	-0.001 (0.013)	-0.001 (0.013)
	p = 1.000	p = 1.000	p = 0.834	p = 1.000	p = 0.021	p = 0.937	p = 0.910
W1 BCEs	0.048** (0.015)	0.039** (0.014)	0.044** (0.015)	0.043** (0.014)	0.038** (0.014)	0.049*** (0.014)	0.044** (0.014)
	p = 0.002	p = 0.008	p = 0.003	p = 0.003	p = 0.008	p = <0.001	p = 0.003
Overall Adversity (OA)		0.163*** (0.013)	0.158*** (0.013)				
		p = <0.001	p = <0.001				
Threat				0.167*** (0.013)	0.149*** (0.013)	0.163*** (0.013)	0.166*** (0.013)
				p = <0.001	p = <0.001	p = <0.001	p = <0.001
Deprivation (Dep)				0.034* (0.013)	0.034** (0.013)	0.032* (0.013)	0.034* (0.013)
				p = 0.010	p = 0.010	p = 0.015	p = 0.010
Interaction			0.047*** (0.013)		0.104*** (0.012)	0.050*** (0.013)	0.020 (0.013)
			p = <0.001		p = <0.001	p = <0.001	p = 0.125
R2	0.002	0.029	0.031	0.035	0.046	0.037	0.035
R2 Adj.	0.002	0.028	0.031	0.034	0.046	0.037	0.034
Num.Obs.	5995	5995	5995	5995	5995	5995	5995

Note. Standardized estimates for regression models where outcome is structural brain change dissimilarity are shown in each column. Estimates are standardized. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. W1 BCEs = positive childhood experiences measured largely at Wave 1. Dep = deprivation.

3.5. Direct model relationships

Follow-up analyses with direct estimates examined whether total scores on either the

BCEs and PCEs or ACEs questionnaire predicted annualized percent change in each of the twelve brain regions of interest. Neither the totals for BCEs and PCEs nor ACEs were associated with structural brain change.

3.6. Alternative BCEs IS-RDM construction

As the positive experiences (BCEs) IS-RDM was almost completely driven by differences in total scores, follow-up analyses were conducted that transformed the total score by dividing by the number of items to constrain values to a 0–1 range. After this transformation, differences along the total score were still the top correlated item with the full, alternative BCEs IS-RDM, though their correlation with the full IS-RDM was about .8 (rather than an almost-perfect correlation). This alternative representation yielded stronger associations with brain development and identified interactions with the OA and Threat IS-RDMs (see Table 22).

Table 23

Alternative BCEs IS-RDM construction results

	Main Effect	OA	BCEs x OA	Dimensions	Threat x Dep	BCEs x Threat	BCEs x Dep
Intercept	0.000 (0.013)	0.000 (0.013)	-0.018 (0.014)	0.000 (0.012)	-0.028* (0.013)	-0.014 (0.014)	-0.005 (0.013)
	p = 1.000	p = 1.000	p = 0.193	p = 1.000	p = 0.031	p = 0.308	p = 0.694
BCEs	0.219*** (0.026)	0.194*** (0.027)	0.204*** (0.029)	0.198*** (0.026)	0.193*** (0.026)	0.212*** (0.027)	0.201*** (0.027)
	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001	p = <0.001
Overall Adversity		0.125*** (0.015)	0.119*** (0.017)				
		p = <0.001	p = <0.001				
Threat				0.150*** (0.018)	0.134*** (0.018)	0.146*** (0.020)	0.147*** (0.019)
				p = <0.001	p = <0.001	p = <0.001	p = <0.001
Deprivation				0.001 (0.016)	0.001 (0.015)	-0.009 (0.016)	0.002 (0.016)
				p = 0.971	p = 0.931	p = 0.568	p = 0.901
Interaction			0.090* (0.028)		0.095*** (0.012)	0.101*** (0.018)	0.026 (0.026)
			p = 0.017		p = <0.001	p = <0.001	p = 0.351
R2	0.048	0.063	0.072	0.070	0.080	0.082	0.071
R2 Adj.	0.048	0.063	0.072	0.070	0.080	0.081	0.071
Num.Obs.	5995	5995	5995	5995	5995	5995	5995

Note. Standardized estimates for regression models where outcome is structural brain change dissimilarity are shown in each column. Estimates are standardized. Standard errors are shown in parentheses. The interaction term is described in the column heading. Model and permutation information (the permuted term, its p-value compared to null distribution, and BH adjusted p-value) are at the bottom of each column. OA = overall adversity. BCEs = retrospective positive childhood experiences. Dep = deprivation.

4. Discussion

4.1. Positive Experiences and Brain Development

This study provides initial evidence that interindividual differences in positive childhood experiences are associated with divergent patterns of structural brain development in the front-amygdala and hippocampal circuit and frontoparietal network during adolescence. Specifically, greater dissimilarity in positive experiences, assessed in late adolescence, was significantly associated with intersubject dissimilarity in structural brain changes through early- to mid-adolescence. This association persisted when controlling for dissimilarity in adverse childhood experiences, whether assessed early or retrospectively, indicating that positive experiences confer unique neurodevelopmental relevance beyond the well-documented effects of adversity. These results highlight the limits of deficit-focused models and support developmental frameworks that recognize the neurobiological pathways through which supportive environments and positive experiences shape brain development.

The model incorporating dissimilarity in retrospective positive experiences (BCEs IS-RDM) alongside the earlier measurements of threat, deprivation, and their interaction explained the most variance in dissimilar brain changes along the regions of interests. This model outperformed one using BCEs and overall adversity dissimilarity alone, aligning with evidence for modeling adversity along theoretically distinct dimensions to better understand outcome heterogeneity. The effect size of the model including dissimilarity in positive experiences and the dimensional interaction (adjusted $R^2 = .05$) represents a modest but meaningful effect of both positive and negative experiences on brain development, which is larger than reliable effects found in traditional brain-wide association studies (Marek et al., 2022) and more in line with other multivariate brain-behavior effects (Spisak et al., 2023).

4.2. Differences in Timing and Measurement

Retrospective measures, where (typically) adults recall events that have happened in the past, are the primary way that both cumulative adverse and positive childhood experiences have been measured (Felitti et al., 1998; Han et al., 2023; Hardt & Rutter, 2004). Yet, they present challenges to temporal interpretation, as they capture a broader range of exposure time, without gathering specificity in when those exposures may have happened. Retrospective reporting may also introduce recall biases and can be greatly influenced by individual perception (Hardt & Rutter, 2004). Such reports have been shown to have low concordance with prospective reporting for adversity and are not recommended to be considered as capturing interchangeable constructs (Baldwin et al., 2019; Danese, 2020). Here, I capitalized on available study measures in follow-up analyses that were captured either later (for adversity) or earlier (for positive experiences). However, the earlier and later measurements differed in breadth for each construct (e.g., ACEs questionnaire at later waves is less comprehensive than the cluster of adversity information at early waves, and the BCEs and PCEs items are more comprehensive than the social support information at early waves). This complicates direct interpretations about the influence of prospective and retrospective reporting. However, other work investigating the influence of timing and measurement provides broader context that can inform understanding of the changes observed in these results.

Retrospective reporting of adverse experiences (collected at ages 15-20), failed to identify the associations between dissimilarity in adversity brain development observed using earlier measurements (collected at ages 10-13). There are several reasons why this may be. It may reflect the limit of retrospective measurement, which, as noted above, may be biased to reflect later perception of events over quantifiable exposure. It also might be because, as many

have noted (Finkelhor, 2018; Lacey & Minnis, 2020a; McLennan et al., 2020), the ACEs questionnaire itself is a relatively limited instrument, and does not identify as many kinds of adversity as were represented in the early measurement IS-RDM. Finally, this difference could reflect the fact that this measure encompassed any adverse exposures that occurred later in adolescence, perhaps even after scanning time points, as this measure does not allow for responders to indicate when an event occurred. However, as it is more likely for children with adverse exposures to experience future adversity (McLaughlin et al., 2012), I find it less likely that this difference was solely driven by individuals who had low adverse exposures prior to early adolescence and high adverse exposures by later adolescence, obscuring effects. It is likely that advantages of measurement accuracy, breadth, and timing sensitivity together resulted in the relative predictive strength of adverse exposures measured in early adolescence. Such measurement has more potential to capture adverse exposures that occured during heightened periods of neural plasticity when environmental inputs can have stronger influence on structural brain development (Tottenham & Sheridan, 2010).

For positive experiences, later measures were instead more comprehensive than earlier measures. Perhaps due to this difference of breadth, the opposite effect emerged compared to adverse measurements, where later measures that captured the cumulative amount of lifetime positive experiences had *stronger* associations than the early measures that captured the domain of social support and belonging. However, the early measures did indicate some association with structural brain developmental differences, *and* the early measurement IS-RDM did more clearly interact with the early adversity IS-RDMs. This tension could be reflected by both the early measurement's disadvantage of limited coverage of the construct and advantage of timing, which likely captured a stronger temporal coincidence with the early measurements of adversity. In this

line, other research has suggested that coordinated timing of both risk and protective factors critically determine their neurobiological impact (Yoder et al., 2020). Given the number of changing variables in these timing and measurement-related differences, further research is needed to substantiate these interpretations.

4.3. Methodological Considerations

Univariate linear models examining direct relationships found no significant associations between annualized percent brain change in individual ROIs and either retrospectively-assessed positive experiences or ACEs total scores. This disparity between univariate and multivariate results highlight how multivariate analytic methods may capture distributed effects in individual differences that are otherwise lost in region-by-region analyses. This method in particular may be particularly relevant for constructs like positive or adverse experiences that may influence brain development through distributed rather than localized effects (Gehred et al., 2021b; Haidl et al., 2023). Intersubject representational similarity analyses may be sensitive to identifying patterns in how environmental factors become embedded in neurobiological development, which could occur through heterogenous pathways. This result aligns with other brain-behavior studies that have found improvements by using individual differences (e.g., deviations from normal development) or multivariate approaches compared to standard analytic techniques (Parkes et al., 2021; Tamman et al., 2024).

Interestingly, in follow-up analyses that reduced the strength of the total score of positive experiences within the BCEs IS-RDM, allowing individual items to be more prominent in their relationship to the overall pairwise distances, the effect of the BCEs IS-RDM increased. Additionally, this alternative construction of the BCEs IS-RDM showed an interaction with adversity IS-RDMs. This may indicate that allowing the distribution of individual differences to

vary more according to specific exposures rather than simply reflecting cumulative experiences through the total score, as more befitting an IS-RSA framework, is advantageous for delineating individual differences in brain neurodevelopmental outcomes. In other words, the multivariate pattern of individual differences in positive experiences may be more relevant to these outcomes than individual differences in total number of positive experiences. Given the exploratory nature of this study, I had no explicit hypotheses for either of these constructions, and this presents an exciting testable question for future work.

4.4. Theoretical Implications

The finding that interindividual differences in positive childhood experiences relate to distinct neurodevelopmental pathways has important translational implications for understanding resilience in the context of neural plasticity (Blair & Raver, 2012; McEwen & Morrison, 2013; Nelson & Gabard-Durnam, 2020b). Though the individual differences in brain development identified here do not necessarily indicate that longitudinal pathways are "good" or "bad," the results support theories that the brain adaptively fine-tunes itself to a range of environmental inputs in somewhat similar ways across individuals (Blair & Raver, 2012), a notion consistent with experience-dependent and experience-expectant models of brain development (Greenough et al., 1987; Marshall & Kenney, 2009; Shonkoff et al., 2012). This highlights the potential utility of interventions designed to enhance a variety of positive experiences during childhood and adolescence as a means of promoting healthy brain development. These effects may operate through mechanisms that are at least partially distinct from those targeted by interventions aimed at mitigating adversity-related effects (Owens & and Waters, 2020; Shonkoff, 2011). Further research grounded in translational and developmental neuroscience frameworks (Horn et al., 2020; Suleiman & Dahl, 2017) can inform whether and how positive experiences shape

developmental and psychological processes. Key questions include whether promoting certain dimensions of experiences have broader or longer-lasting effects by engaging neurodevelopmental mechanisms, how intervention timing influences psychological and neural outcomes, and whether some individuals are especially responsive to interventions based on their neurodevelopmental profiles.

4.5. Limitations

Several limitations should be considered when interpreting these findings. First, there are inherent challenges related to the timing and nature of experience measurement across study waves. In earlier waves, adversity and positive experiences were approximated using a constellation of questionnaire items, rather than a unified, validated scale. While this approach allowed us to capture a broad range of experiences, it introduces variability in construct validity. Future studies would benefit from employing standardized instruments that comprehensively assess both adverse and protective experiences from early in development (Berman et al., 2022). Furthermore, our operationalization of deprivation was constrained by available data. While we attempted to capture this dimension through family and neighborhood sociodemographic indicators, in addition to measurements of neglect, our measures likely do not fully reflect the true nature of deprivation. We were also unable to include the dimension of unpredictability, which has gained increasing recognition as a critical aspect of early experience affecting neural and behavioral outcomes (Ellis et al., 2009b; Ellis, Sheridan, et al., 2022).

Also, this study was conducted with a community sample in Oregon, which may limit generalizability. Although this sample exhibited a range of adverse experiences, and it is important to characterize both typical and atypical development (Fuligni et al., 2018), it is unclear whether the observed associations between positive experiences and brain development

would generalize to populations experiencing higher levels of adversity.

Finally, the early-wave measures of positive experiences available in this study were more narrow in scope compared to the later measurements, primarily focused on domains of social support and belonging. This may explain why dissimilarity along early measurements showed smaller associations with brain development in comparison to the later measures, but would need to be further investigated with future study.

5. Conclusion

These findings provide evidence that individual differences in neurobiological development may be influenced by positive childhood experiences, such that individuals with greater differences in their total amount of positive experiences diverge in their structural brain development within regions in the fronto-amygdala and hippocampal circuit and frontoparietal network. These results suggest that mechanisms of resilience may influence neurobiological development independently of adversity exposure. They underscore the value of considering protective factors alongside risk when characterizing individual differences in brain development. Future research should consider developmental timing and comprehensive measurement when examining positive experiences, as our findings suggest that concurrent measurement in early adolescence of the environment may better capture the influence of experiences on neurodevelopmental trajectories.

CHAPTER V: GENERAL DISCUSSION

1. Overview of Research and Goals

This dissertation investigated how adverse and positive childhood experiences relate to adolescent brain development and mental health. Specifically, it (1) disentangled the independent and interactive relationships of individual differences in adverse and positive childhood experiences to internalizing and externalizing symptom trajectories, (2) tested how threat and deprivation interact to shape both behavioral and neural outcomes, and (3) identified how differences in distributed patterns of brain development reflect individual differences in adverse and positive experiences.

A novel methodological contribution of this dissertation is the application of intersubject representational similarity analysis (IS-RSA), a multivariate approach that captures how dissimilarity in experiential profiles relates to dissimilarity in development. Unlike traditional variable-centered methods, IS-RSA can flexibly model the multidimensional structure of various experiences, offering a more holistic and ecologically valid understanding of how real-world patterns of individual differences in adversity and support relate to adolescent mental health and brain development.

Chapter I identified an opportunity to synthesize and extend emerging areas of interest among neurodevelopmental, resilience, and clinical researchers. There, I summarized various areas of research and called for continued work to incorporate updated measurements of positive experiences with the substantial investigations into the effects of childhood adversity. Chapter II examined links between life experiences and adolescent mental health trajectories. Chapter III examined dimensions of adversity in relation to brain development. Finally, Chapter IV sought to respond to the call in Chapter I by investigating how positive experiences relate to brain

development.

2. Summary of Results and Implications

Chapter II findings revealed that dissimilarity in adversity was more consistently linked to externalizing symptoms than internalizing ones, with deprivation and threat dimensions interacting to shape these associations. Positive experiences showed broader associations with mental health trajectories, and individual differences in positive experiences were related to both internalizing and externalizing symptoms across both parent and child reporters. Differences in positive experiences also interacted with adversity differences in predicting externalizing differences.

The analytic plan for Chapter III was refined through the registered report two-stage process, where external reviewers provided suggestions to improve our ability to evaluate the research question. As expected, we found that differences in threat and deprivation, and their interaction, were positively associated with differences in structural brain development. These findings met the pre-registered threshold established in stage one of the process, and provided support for a dimensional model of adversity that recognizes the unique contributions and their potential interactions. Unexpectedly, however, we did not find that these dimensions had better congruence with individual differences in specific regions. Rather, threat showed the strongest relationship with both the front-amygdala and hippocampus representation (as hypothesized), and also with frontoparietal network representation (not as hypothesized). An additional exploratory test examining regions identified from a systematic review (McLaughlin et al., 2019) found that adversity configurations did not relate to these regions anymore than any selection of regions from the whole brain, indicating that a multivariate representation of individual differences in adversity likely reflect distributed individual differences across the whole brain.

Another exploratory test found that latent class modeling of adversity did not yield similar results to modeling individual differences.

Chapter IV provided early evidence that individual differences in positive experiences, independent of adverse exposures, are also associated with divergent patterns of adolescent structural brain development along the same regions as identified in Chapter III. Differences in experiences were initially characterized as fully representative of differences in the total sum of measured experiences, and restriction of the total to exert less weight in the distance calculations resulted in a stronger relationship with structural brain developmental differences. Additionally, the timing, and likely kind, of experience measurement mattered. For adverse experiences, earlier and more comprehensive measures were more informative for identifying individual variation in brain development.

3. Integrative Discussion

The findings across these studies collectively underscore the importance of incorporating thorough representations of positive experiences into developmental research frameworks and suggest that variation in both adverse and positive experiences meaningfully contribute to variation in mental health and neurodevelopmental trajectories during adolescence. Although this point has been emphasized throughout these dissertation chapters, the combined findings highlight the potential to explain a comparable degree of outcome heterogeneity to that typically attributed to adversity by incorporating a relatively simple measure of positive experiences that extends beyond traditional representations like social support or individual protective factors. This opens exciting directions for future research that can draw on a range of methodological tools (e,g., refined measures of positive experiences, idiographic analytic approaches), target diverse populations (e.g., those affected by intersecting forms of social disadvantage or

oppression), and inform a variety of theoretical frameworks (e.g., ICARE model of development, theories of unpredictability, differential susceptibility theory).

This integrative discussion further synthesizes key insights gained across these chapters, highlighting advances in understanding dimensional approaches to adversity, the interpretation of effect sizes, methodological innovations, and clinical implications. Together, these themes illustrate the value of a multidimensional, developmentally informed lens for studying how childhood experiences shape adolescent mental health and neurodevelopment.

3.1. Dimensions of Adversity: Insights and Limitations

The dimensional approach to adversity employed in this dissertation yielded several important findings. First, examining threat and deprivation as distinct dimensions proved especially valuable for understanding developmental change, particularly when investigating neurodevelopmental differences. Notably, their interaction emerged as a significant predictor, suggesting that specific combinations of dissimilarity across these dimensions can meaningfully alter the magnitude of neurodevelopmental dissimilarity. Furthermore, the threat dimension, along with its interaction with deprivation, remained a significant predictor even when controlling for the conceptualizations of positive experience dissimilarity (i.e., early-measured positive social experiences and later, broader measures of positive experiences) in analyses using the subsample. In the subsample analyses, deprivation did not show a direct effect, which is perhaps unsurprising given that its effect size was substantially smaller than that of threat in the full sample ($\beta = 0.02$ vs. $\beta = 0.15$, respectively). These findings align with theoretical models suggesting that different dimensions of adversity independently influence development and interact with each other in affecting mechanistic pathways (McLaughlin et al., 2021).

However, our findings did not support a clear theoretical correspondence between

specific dimensions of adversity and theoretically relevant regions of interest (ROIs). While threat-related dissimilarity showed stronger associations with inter-subject dissimilarity in the development of the fronto-amygdala and hippocampus circuit—as hypothesized—it was also more strongly related to the frontoparietal network, which we had predicted to be more closely associated with deprivation. Additionally, these dimensions were not more strongly related with literature-based ROIs than with randomly generated combinations of brain regions. This pattern may indicate a more general, distributed effect of adversity on brain development, which may be difficult to detect using univariate approaches. This interpretation aligns with other research (Gehred et al., 2021a; Haidl et al., 2021) and underscores the complexity of biological embedding, suggesting that the effects of early experiences may manifest as coordinated, system-level changes rather than localized alterations in specific structures.

Several other interpretations may also explain these findings. Although adverse experiences likely influence distributed brain networks, the mechanisms through which they contribute to psychopathology may still operate through specific circuits, an idea not directly tested in this dissertation. Additionally, if individuals experienced threat or deprivation early in development, the associated effects on specific brain regions may have already occurred prior to early adolescence and thus would not have been detectable at the time of measurement. Finally, it is possible that such effects might be more readily observed in a high-adversity sample, where greater variation and intensity of exposure could amplify detectable brain-behavior associations.

Interestingly, although dimensional model theory does not explicitly suggest that dimensions should relate differently to mental health outcomes, but rather suggest that they both confer transdiagnostic risk that can accumulate over time (Stein et al., 2022b), our findings revealed some nuanced patterns in these relationships. Both threat and deprivation dissimilarity,

along with their interaction, predicted differences in externalizing symptoms. However, in the subsample of participants who had also completed the later-wave positive experiences measure, threat dissimilarity was no longer a significant predictor, while deprivation dissimilarity maintained its predictive effect. Nevertheless, both dimensions continued to show significant interactions with positive experiences.

This raises an important question about why the direct association between threat dissimilarity and externalizing dissimilarity did not replicate in the subsample (110 participants of the original 174) given the relatively larger association (r = .21). Although the subsample of included participants did not differ from those excluded in adversity exposure, as assessed through measures of abuse, traumatic event exposure, and bullying, it remains possible that differences in nature or distribution of threat experiences after exclusion contributed to the altered association. Curiously, the threat association did not substantially change with any other outcome in this study when doing subsample analyses, making this finding more puzzling. One study on both children and adolescents found that while both experiences of threat and deprivation were related to higher levels of psychopathology cross-sectionally, only deprivation related to psychopathology longitudinally (Schäfer et al., 2023), which may represent a weaker relationship for threat generally. Additional work in other samples will be necessary to determine the robustness of the link between threat-related adversity and externalizing symptoms. Altogether, these mental health findings, along with prior work (Miller et al., 2018b; Schäfer et al., 2023; Stein et al., 2022b), suggest that the relationship between adversity dimensions and mental health outcomes may vary in their strength over time, highlighting the importance of continued investigations of dimensions to uncover the correlates between adversity and psychopathology.

The measurement of deprivation remains particularly challenging for researchers (Berman et al., 2022), and the studies' operationalization of deprivation which attempted to capture aspects of socio-emotional, material, and neighborhood-level deprivation, may not have adequately captured the theoretical construct. Neglect experiences in early development may be particularly difficult to self-report retrospectively, and measures of deprivation are generally not as comprehensive as measures of threat. Although neglect is more common than abuse (Brown et al., 2025), which is reflected in higher average endorsement and greater variability in our sample, it is possible that we did not capture it thoroughly through adolescent self-report. Additionally, concerns persist regarding the inclusion of socioeconomic status (SES) measures as imperfect proxies for deprivation. While some SES indicators were used in this work and by prior studies (Sheridan et al., 2017), recent recommendations advise against relying on SES as a stand-in for deprivation (McLaughlin et al., 2021). Indeed, other work has indicated that deprivation and threat exposures partially account for the impact of low SES on developmental outcomes and may therefore offer a more precise characterization of environmental risk (Berman et al., 2022).

It is worth noting that in our sample, differences in free lunch status emerged as the top correlate to variation in the deprivation IS-RDM (r = .67). Differences in emotional neglect (r = .54) and physical neglect subscales were also highly correlated (r = .51), as well as the CTQ item assessing if an adolescent had someone to care and protect them (r = .54). Differences in neighborhood disadvantage index scores (r = .14) and parent education status (r = .22) were some of the weakest variables associated with the deprivation IS-RDM, suggesting that these variables are less reflective of overall differences across the broader set of deprivation indicators. This pattern indicates that effects observed in the deprivation analyses were likely not driven by SES proxies alone, given the high relevance of neglect interpersonal differences. Prior research

has shown that adjusting for SES covariates can reduce effect sizes, particularly for the strongest associations (Marek et al., 2022). Thus, it would be worthwhile to co-vary for socioeconomic indicators in further analyses.

In their measurement recommendations, Berman and colleagues (2022) recommended creating composite scores across multiple items to capture severity along each dimension, given that clearly-defined and precise measurements of adverse dimensions remain elusive. This IS-RSA approach extended this recommendation by representing multiple items capturing severity along each dimension, enabling the modeling of multidimensionality in heterogeneous or homogeneous exposure profiles. However, this use of IS-RSA also has limitations for representing dimensions, particularly because individual adverse experiences rarely map neatly into discrete assigned categories.

Future research should also incorporate other emerging adversity dimensions, such as unpredictability, which has been thought to play a significant role in both brain development and psychopathology (Brieant et al., 2022; Ellis et al., 2009a; Ellis, Sheridan, et al., 2022; Fisher & Lombardi, 2025).

3.2. Effect Sizes and Their Meaning

The observed effect sizes in the neuroimaging results were substantially larger than typical replicable effect sizes of bivariate brain-behavior associations. As Marek et al. (Marek et al., 2022) noted, in their rigorous analyses using the ABCD sample (n = 3,928), the median univariate effect size ($|r|$) was merely 0.01, with the top 1% of brain-wide associations showing $|r|$ values greater than 0.06. Our multivariate approach yielded effect sizes more in line with other multivariate brain-behavior associations (Spisak et al., 2023), with $|r|$ ranging from 0.08 to 0.18 for all experience IS-RDMs examined in both Chapter III and Chapter IV. Spisak and colleagues

demonstrated that medium sample sizes (n = 75-500) can produce reliable effect sizes using multivariate techniques, though they emphasize that multivariate studies incorporating cross-validation are most reliable. Incorporating cross-validation would therefore be a valuable extension of the current work. The observed effects for the mental health results demonstrated a similar magnitude, specifically for externalizing problems, to the effect sizes in other work exploring the influence of childhood maltreatment on mental health (Humphreys et al., 2020; M. Li et al., 2023).

It is important to note that the effects in IS-RSA refer to a fundamentally different operation of these constructs from most analytic approaches and can be challenging to interpret. In the context of IS-RSA, variance in dissimilarity reflects how much individuals differ from one another in their outcome trajectories, rather than how much individuals differ in outcome magnitude. This second-order form of variance captures the diversity of trajectory patterns across the sample, making it conceptually distinct from the variance typically modeled in regression-based or growth curve analyses. Consequently, interpreting effects in IS-RSA requires a shift from thinking about individual outcomes to thinking about inter-individual pattern similarity, which is a fundamentally relational construct. It will be important to explore this method in other and larger samples to see if effects and their size demonstrate reliability.

3.4. Utility of the IS-RSA Approach

The application of IS-RSA approach in this dissertation posed a fundamentally different question than is typical in developmental research: not whether exposures result in a quantifiable change in development, but rather whether individuals with dissimilar experiences show correspondingly dissimilar interpersonal patterns of development. Our findings suggest that IS-RSA may be particularly valuable for examining outcomes that are distributed across systems,

rather than localized to individual measures or regions.

IS-RSA differs from person-centered approaches such as latent profile analyses or growth mixture modeling which also capture similarity among individuals, in that it does not group individuals into discrete categories or trajectories. Instead, IS-RSA represents a continuous landscape of dissimilarity resulting from pairwise comparisons. In our sample, latent profile analysis of adverse exposures yielded groupings characterized by low adversity, lower SES, and high adversity profiles. However, these profiles did not meaningfully relate to brain development in any of the examined regions of interest. In contrast, IS-RSA likely offered greater sensitivity to individual variation, enhanced power to detect effects, and reduced the number of statistical comparisons—without requiring a priori assumptions about which brain regions would be affected or how.

Similar analytic differences emerged when comparing the multivariate, IS-RSA approach and the univariate neurodevelopmental analyses. While brain dissimilarity effects emerged using the multivariate IS-RSA approach, no significant associations were observed between experiences and individual ROIs when using univariate methods. Interestingly, the reverse pattern was observed for mental health outcomes, where associations were detected using univariate trajectory estimates. This divergence highlights the differential sensitivity of multivariate and univariate analytic approaches. It is possible that different analytical approaches may be optimal for different outcome domains. Alternatively, this discrepancy may reflect how outcomes were operationalized: brain development was modeled using multivariate patterns across regions, while mental health trajectories were summarized into single indices, potentially limiting their complexity and sensitivity.

As Spisak et al. (2023) noted, "There is a necessary trade-off between the innovativeness

of a task, measure or method, and the extent to which it has been validated." While some aspects of this work were rigorously pre-registered, much of its nature was inherently exploratory, given that IS-RSA remains a novel methodological tool with limited application in longitudinal developmental research. Although these studies were not explicitly designed to validate this method, they offer preliminary evidence that IS-RSA may provide an advantageous option for smaller samples investigating individual differences in normative neurodevelopmental trajectories, where univariate approaches would likely be underpowered.

This dissertation brings together empirical evidence that would ideally be replicated in larger cohorts. Future research will need to address boundaries of these findings' generalizability across diverse contexts. Moreover, it will be valuable to complement models with high neuroscientific validity and reliability with ones that also have high interpretability, an area where IS-RSA has limitations.

3.5. Clinical Implications for Adolescents

Though the findings of this dissertation are based on observational data, they illuminate further avenues for future research and generate testable hypotheses that could meaningfully refine prevention and intervention work with adolescents. Intervention designs for this age group can be greatly improved by grounding in developmental science (Giovanelli et al., 2020). However, a substantial gap persists between developmental theory and the design of many empirically supported intervention programs for children and adolescents, where even well-supported interventions overlook relevant developmental differences in their targets (Malti et al., 2017). Many interventions fail to account for the unique individual developmental contexts, general priorities of the relevant (i.e., adolescent) period, and characteristic strengths of that period. This may consequently limit effectiveness and impact.

Much research has elucidated the considerable inter-individual variability in adolescent brain development (K. L. Mills et al., 2021b), and the findings of this dissertation contribute to evidence that suggests environmental influences are related to these individual differences. Adolescent neural plasticity, which also enhances specialized learning in identity and social knowledge, creates substantial opportunities for interventions that recognize such learning-ready states and align with developmental priorities in promoting the development of well-being (Immordino-Yang et al., 2019; Suleiman & Dahl, 2017).

Integrating positive experiences in the next steps of intervention research offers a promising avenue for establishing effective initial treatment targets. Based on the findings in this dissertation, it is plausible that interventions aimed *intentionally amplifying* diverse positive experiences may yield greater benefits for mental health outcomes in some adolescents. Such a focus aligns well with strengths-based approaches (Bryant et al., 2021; Pulla, 2017) and may operate through mechanisms that only partially overlap with those targeted by adversity-mitigating programs, underscoring the need for complementary strategies (Zimmerman, 2013). Future research should investigate whether there are specific dimensions, types, or combinations of positive experiences that are particularly salient during adolescence, thereby improving the precision and effectiveness of clinical interventions. This dissertation highlights that family- and community-based experiences represent the greatest differences in general positive experiences among adolescents, which in turn relate to greater differences in their mental health trajectories. These domains may therefore represent particularly fruitful targets for future intervention efforts.

By advancing our understanding of how both adverse and positive experiences shape developmental trajectories, this dissertation contributes to an emerging framework that can inform more developmentally sensitive and effective approaches to supporting adolescent

wellbeing.

APPENDIX

SUPPLEMENTAL INFORMATION

Table S1

Schaefer 400 Atlas Selected Parcellations

Threat	Right Hemisphere	368	17Networks_DefaultA_PFCm_1
		371	17Networks_DefaultA_PFCm_4
		370	17Networks_DefaultA_PFCm_3
		392	17Networks_TempPar_2
		310	17Networks_SalVentAttnB_Ins_2
		383	17Networks_DefaultB_PFCv_2
		318	17Networks_LimbicB_OFC_6
	Left Hemisphere	161	17Networks_DefaultA_PFCm_1
		162	17Networks_DefaultA_PFCm_2
		163	17Networks_DefaultA_PFCm_3
		94	17Networks_SalVentAttnA_FrOper_2

		177	17Networks_DefaultB_PFCd_3
Deprivation	Right Hemisphere	360	17Networks_DefaultA_IPL_2
		384	17Networks_DefaultB_PFCv_3
		341	17Networks_ContB_IPL_4
		372	17Networks_DefaultA_PFCm_5
		362	17Networks_DefaultA_PFCd_2
		260	17Networks_DorsAttnA_TempOcc_2
		277	17Networks_DorsAttnB_PostC_4
		300	17Networks_SalVentAttnA_ParMed_3
		271	17Networks_DorsAttnA_SPL_7
		318	17Networks_LimbicB_OFC_6
	Left Hemisphere	141	17Networks_Cont_PFCl_7
		185	17Networks_DefaultB_PFCv_3
		44	17Networks_SomMotB_Aud_1
		154	17Networks_DefaultA_pCunPCC_1
		145	17Networks_ContC_pCun_2
		71	17Networks_DorsAttnA_SPL_6

		80	17Networks_DorsAttnB_PostC_8
		176	17Networks_DefaultB_PFCd_2
		148	17Networks_ContC_Cingp_2

Note. Parcellations are grouped by associated adversity dimension and hemisphere. Parcel 318 is represented twice since it has been identified in both a threat- and deprivation-focused study.

Figure S1.

Animation of observed deprivation parcellations

Figure S2.

Animation of observed threat parcellations

Sensitivity Analyses

Removing physical neglect from IS-RDMs

Each analysis was rerun without the incorporation of the physical neglect scale when calculating inter-subject distances for deprivation and overall adversity. The results of the analyses for each of the three hypotheses are included below. In summary, all results remained the same except for hypothesis one, where the deprivation IS-RDM no longer exerted a main effect on the brain development IS-RDM.

Hypothesis 1:

Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-1.479e-17	0.013	0.000	1.000
Threat IS-RDM	0.174	0.013	13.309	**<0.001**
Deprivation IS-RDM	0.013	0.013	0.982	0.326

Brain Development IS-RDM ~ Overall Adversity IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-5.639e-17	0.013	0.000	1.000
Overall adversity IS-RDM	0.159	0.013	12.55	**<0.001**

Hypothesis 2:

Comparisons of dimensional and overall adversity models

Model	*Adjusted R^2*	*P-Value*	*AIC*	*BIC*
Dimensional	0.033	**<0.001**	17130	17164
Overall adversity	0.025	**<0.001**	17175	17195

Note. The dimensional model includes the interaction term.

*Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM + Threat IS-RDM*Deprivation IS-RDM*

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-0.008	0.013	-0.658	0.510
Threat IS-RDM	0.169	0.013	12.790	**<0.001**
Deprivation IS-RDM	0.012	0.013	0.927	0.354
Interaction	0.031	0.011	2.640	**0.008**

Hypothesis 3:

Comparison of two overlapping correlations based on dependent groups.

Correlation 1	*Correlation 2*	*Difference between correlations*	*Related correlation*	*Z*	*P-Value*
Threat adversity & "threat" regions	Deprivation adversity & "threat" regions	0.376	0.27	30.017	**<0.001**

Threat adversity & "threat" regions	Overall adversity & "threat" regions	0.067	0.844	12.362	**<0.001**
Deprivation adversity & "deprivation" regions	Threat adversity & "deprivation" regions	-0.315	0.272	-23.451	**<0.001**
Deprivation adversity & "deprivation" regions	Overall adversity & "deprivation" regions	-0.279	0.719	-31.593	**<0.001**

Note. A negative difference between correlations indicates that correlation 2 is larger than correlation 1.

Removing emotional neglect from deprivation IS-RDM

Each analysis was rerun without the incorporation of the emotional neglect scale when calculating inter-subject distances for deprivation and overall adversity. The results of the analyses for each of the three hypotheses are included below. In summary, all results remained the same except for hypothesis three, where the threat IS-RDM had a weaker association with the structural brain development IS-RDM compared to the overall adversity IS-RDM.

Hypothesis 1:

Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-1.288e-17	0.013	0.000	1.000
Threat IS-RDM	0.172	0.013	13.557	**<0.001**
Deprivation IS-RDM	0.044	0.013	3.482	**<0.001**

Brain Development IS-RDM ~ Overall Adversity IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-4.167e-17	0.013	0.000	1.000
Overall adversity IS-RDM	0.172	0.013	13.65	**<0.001**

Hypothesis 2:

Comparisons of dimensional and overall adversity models

Model	*Adjusted R^2*	*P-Value*	*AIC*	*BIC*
Dimensional	0.051	**<0.001**	17017	17051
Overall adversity	0.030	**<0.001**	17147	17167

Note. The dimensional model includes the interaction term.

*Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM + Threat IS-RDM*Deprivation IS-RDM*

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-0.017	0.013	-1.356	0.175
Threat IS-RDM	0.163	0.013	12.943	**<0.001**

Deprivation IS-RDM	0.048	0.013	3.875	**<0.001**
Interaction	0.133	0.013	10.473	**<0.001**

Hypothesis 3:

Comparison of two overlapping correlations based on dependent groups.

Correlation 1	*Correlation 2*	*Difference between correlations*	*Related correlation*	*Z*	*P-Value*
Threat adversity & "threat" regions	Deprivation adversity & "threat" regions	0.245	0.128	19.366	**<0.001**
Threat adversity & "threat" regions	Overall adversity & "threat" regions	-0.051	0.840	-10.144	**<0.001**

Deprivation adversity & "deprivation" regions	Threat adversity & "deprivation" regions	-0.411	0.128	-27.944	**<0.001**
Deprivation adversity & "deprivation" regions	Overall adversity & "deprivation" regions	-0.416	0.608	-40.452	**<0.001**

Note. A negative difference between correlations indicates that correlation 2 is larger than correlation 1.

Imputing all missing data (no wave substitutions)

Hypothesis 1:

Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-7.827e-17	0.013	0.000	1.000

Threat IS-RDM	0.106	0.014	7.707	**<0.001**
Deprivation IS-RDM	-0.038	0.014	-2.785	**0.005**

Brain Development IS-RDM ~ Overall Adversity IS-RDM

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-5.058e-17	0.013	0.000	1.000
Overall adversity IS-RDM	0.062	0.013	4.889	**<0.001**

Hypothesis 2:

Comparisons of dimensional and overall adversity models

Model	*Adjusted R^2*	*P-Value*	*AIC*	*BIC*

Dimensional	0.051	**<0.001**	17017	17051
Overall adversity	0.009	**<0.001**	17274	17307

Note. The dimensional model includes the interaction term.

*Brain Development IS-RDM ~ Threat IS-RDM + Deprivation IS-RDM + Threat IS-RDM*Deprivation IS-RDM*

Predictors	*Estimate*	*SE*	*T-Value*	*P-Value*
(Intercept)	-0.005	0.013	-0.373	0.709
Threat IS-RDM	0.105	0.014	7.638	**<0.001**
Deprivation IS-RDM	-0.040	0.014	-2.891	**0.004**
Interaction	0.014	0.012	1.154	0.248

Hypothesis 3:

Comparison of two overlapping correlations based on dependent groups.

Correlation 1	*Correlation 2*	*Difference between correlations*	*Related correlation*	*Z*	*P-Value*
Threat adversity & "threat" regions	Deprivation adversity & "threat" regions	0.283	0.370	20.684	**<0.001**
Threat adversity & "threat" regions	Overall adversity & "threat" regions	0.081	0.850	12.141	**<0.001**
Deprivation adversity & "deprivation" regions	Threat adversity & "deprivation" regions	-0.401	0.370	-29.626	**<0.001**
Deprivation adversity &	Overall adversity &	-0.255	0.780	-30.736	**<0.001**

“deprivation”	“deprivation”
regions	regions

Note. A negative difference between correlations indicates that correlation 2 is larger than correlation 1.

Permutation Tests (Main Analyses)

The beta coefficient for each hypothesis was compared to a null distribution created by permuting participant IDs 10,000 times in each of the similarity matrices separately. These p-values represent the likelihood of obtaining the beta coefficient values (or a more extreme value) in the main analyses compared to these permuted comparisons.

Hypothesis	*Predictor*	*P-Value*
1	Threat IS-RDM	**<0.001**
1	Deprivation IS-RDM	**0.006**
1	Overall adversity IS-RDM	**<0.001**
2	Interaction	**<0.001**
3	Threat IS-RDM	**<0.001**
3	Deprivation IS-RDM	**<0.001**

Exploratory Analysis 3 (Latent Classes)

T-Test Results from 2-Class Model

ROI	*Hemisphere*	*Class 1 average*	*Class 2 average*	*T-Value*	*P-Value*
Amygdala	Right	0.282	-0.117	0.966	0.339
Amygdala	Left	0.077	0.111	-0.081	0.936
Hippocampus	Right	0.244	0.127	0.712	0.479
Hippocampus	Left	0.192	-0.126	1.356	0.182
Superior Parietal	Right	-1.272	-1.134	-0.423	0.673
Superior Parietal	Left	-1.243	-1.385	0.513	0.610
Rostral Middle Frontal	Right	-1.629	-1.581	-0.163	0.872
Rostral Middle Frontal	Left	-1.419	-1.622	0.710	0.482
Rostral Anterior Cingulate	Right	-0.811	-1.310	1.373	0.177

Rostral Anterior Cingulate	Left	-0.743	-0.863	0.381	0.704
Medial Orbitofrontal	Right	-1.381	-1.513	0.405	0.687
Medial Orbitofrontal	Left	-0.977	-1.216	0.865	0.3914

Anova Results from 2-Class Model

ROI	*Hemisphere*	*F-Value*	*P-Value*
Amygdala	Right	0.217	0.805
Amygdala	Left	1.309	0.274
Hippocampus	Right	0.573	0.566
Hippocampus	Left	1.551	0.217
Superior Parietal	Right	0.666	0.516
Superior Parietal	Left	0.033	0.967

Rostral Middle Frontal	Right	0.234	0.792
Rostral Middle Frontal	Left	0.457	0.634
Rostral Anterior Cingulate	Right	1.008	0.368
Rostral Anterior Cingulate	Left	0.026	0.974
Medial Orbitofrontal	Right	1.446	0.240
Medial Orbitofrontal	Left	0.287	0.751

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