Dimensional Views of Childhood Adversity and Psychopathology: Insights from Growing Up in New Zealand

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A thesis submitted in partial fulfilment of the requirements for

the degree of Master of Science in Psychology

the University of Auckland, 2024

Abstract

Background: The dose-response relationship demonstrated by the cumulative risk approach has helped identify which children are in need for intervention and is univocal in the transdiagnostic risk that childhood adversity poses for psychopathology. However, the homogenization of different types of experiences and reliance on stress-based explanations, are key limitations of this approach. As such, different frameworks such as the Dimensional Model of Adversity and Psychopathology (DMAP) and the Harshness-Unpredictability framework, have focused on categorising distinct forms of adverse experiences into core dimensions based on their neurobiological and evolutionary consequences on development. Similarly, many have argued for dimensional views of psychopathology through the use of: a) symmetrical bi-factor models, where each symptom loads onto its specific factor and a general factor; and b) bi-factor S-1 models, consisting of specific factors and a general factor that is defined a priori by a reference domain. Further investigation is required to integrate these dimensional approaches to better understand the relationship between childhood adversity and psychopathology.

Aims: The aim of this study was to investigate the factor structure and reliability of common models of psychopathology using the Total Difficulties Score (TDS) of the Strengths and Difficulties Questionnaire (SDQ). Another aim was to construct different adversity dimensions and test their association with the SDQ and the mediating role of executive control in this relationship.

Methods: Using Growing Up in New Zealand data, the structure of the TDS was assessed for 4632 children as well as its association with sociodemographic and behavioural correlates. Adversity dimensions (threat, deprivation, and unpredictability) were measured using various mother-reported data from the child's first 4.5 years of life. Direct and indirect effects of

these adversity dimensions were examined with the SDQ at age 8 years and through the Luria Handclap Task (executive control) measured at age 4.5 years.

Results: Confirmatory factor analyses and ancillary statistics revealed anomalous results across most models tested. The bi-factor S-1 model emerged as the most suitable structure for the TDS and showed correlations with lower prosocial behaviour using the SDQ. All adversity dimensions were associated with greater TDS; however, only deprivation showed significant indirect effects through poorer executive control in a combined mediation model.

Conclusions: Bi-factor S-1 models represent a theoretically superior alternative to symmetrical bi-factor models, and present an opportunity to leverage the SDQ in transdiagnostic research. Further, this is the first study to highlight the utility in integrating multiple adversity dimensions from different frameworks, and show the transdiagnostic role of executive control as a target for intervention and prevention.

Acknowledgements

This Masters would not have been possible without the Growing Up in New Zealand participants, families, and researchers, for which I am truly grateful.

I would like to thank my primary supervisor Professor Karen Waldie for her invaluable support and mentorship. Karen, I really cannot thank you enough for all the guidance you have provided me in the time we have worked together. I would also like to thank my secondary supervisor Dr Rebecca Slykerman for her valuable input, encouragement, and constructive feedback.

To my mom and dad, who were the first to encourage my career change and who are always proud of my achievements. Thank you for your unfaltering support and for showing me what selflessness and perseverance look like.

I am also grateful to friends, both near and far, who have been instrumental in helping me get through this year. I am beyond lucky to have you all in my life.

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List of Abbreviations

ACE	=	Adverse Childhood Experiences
ALM	=	Allostatic Load Model
APRB	=	Absolute Relative Parameter Bias
BSCT	=	Biological Sensitivity to Context
CBCL	=	Child Behaviour Checklist
CES-DC	=	Center for Epidemiological Studies Depression Scale for Children
CFA	=	Confirmatory Factor Analysis
CFI	=	Comparative Fit Index
CI	=	Confidence Interval
CR	=	Cumulative Risk
dACC	=	Dorsal Anterior Cingulate Cortex
DCW	=	Data Collection Wave
DMAP	=	Dimensional Model of Adversity and Psychopathology
DSM	=	Diagnostic and Statistical Manual of Mental Disorders
DSIS-C	=	Domain-Specific Impulsivity Scale for Children
EC	=	Executive Control
ECV	=	Explained Common Variance
EPDS	=	Edinburgh Postnatal Depression Scale
FD	=	Factor Determinacy
GUiNZ	=	Growing Up in New Zealand
HPA	=	Hypothalamic-pituitary-adrenal axis
mPFC	=	Medial Prefrontal Cortex
NIH	=	National Institutes of Health
NZDI	=	New Zealand Index of Deprivation

NZiDep	=	New Zealand Index of Deprivation for Individuals
PHQ	=	Patient Health Questionnaire
PUC	=	Percentage Uncontaminated Correlations
PROMIS	=	Patient Reported Outcomes Measurement Information System
RMSEA	=	Root Mean Square Error of Approximation
SDQ	=	Strengths and Difficulties Questionnaire
SE	=	Standard Error
SEM	=	Structural Equation Modelling
SRMR	=	Standardised Root Mean-Square Residual
TDS	=	Total Difficulties Score
TLI	=	Tucker-Lewis Index
WASI	=	Women's Abuse Screening Tool
WLSMV	=	Weighted Least Square Mean and Variance Adjusted Estimation

1 General Background

It is widely established that adverse childhood experiences (ACEs), such as exposure to interparental violence, child sexual abuse, and neglect, have substantial effects on health and development (Kalmakis & Chandler, 2015). This abundant literature stems from early observations of adverse developmental trajectories for children exposed to important individual risk factors, such as poverty or sexual abuse, which is what is now deemed a specificity approach to childhood adversity (Rutter, 1981). As researchers in the field began to recognise that adversities frequently co-occur (Finkelhor et al., 2007; Dong et al., 2004), researchers transitioned to a cumulative risk (CR) approach that examines associations between the number of unique adversities experienced by a child and developmental sequelae, demonstrating a graded relationship between the two (as first reported in the foundational ACEs study by Felitti et al., 1998). Due to the simplicity in implementation and replication of these approaches, many studies have reported valuable insights regarding the increased risk childhood adversity poses for poor outcomes across the lifespan, such as asthma (Wing et al., 2015), cardiovascular disease (Godoy et al., 2021), and experiences of intimate partner violence (Fergusson et al., 2008). Further, this approach has revealed strong associations between childhood adversity and psychopathology (Herzog & Schmahl, 2018; O'Hare et al., 2022), which is broadly defined as the study of abnormal mental health states (Heckers, 2014), and is the primary focus of this thesis. Importantly, this early research helped identify which children are most at risk for developmental disadvantage and, as such, require intervention.

Research into ACEs has burgeoned over the past decade (Struck et al., 2021), and this literature has established several patterns related to childhood adversity and psychopathology. Firstly, childhood adversity is suggested to pose transdiagnostic risk for psychopathology, as studies show elevated risk for almost all types of mental health disorders, such as mood

disorders (Angst et al., 2011), personality disorders (Raposo et al., 2014), and psychotic disorders (Morgan et al., 2020; Rosenfield et al., 2022). Evidence from US population-based studies shows that the elevated risk is largely non-specific across categories of psychiatric disorders (McLaughlin et al., 2010; Kessler et al., 1997). In addition, this elevated risk is consistent across the lifespan, showing associations in childhood, adolescence, and adulthood (McLaughlin et al., 2010; Kessler et al., 2010). Lastly, a World Mental Health Survey using data from 21 countries showed that 29.8% of global mental health problems can be accounted for by childhood adversities and reported strong associations between childhood adversities and all types of disorders regardless of income level (Kessler et al., 2010).

Despite the foundational knowledge that specificity and cumulative risk approaches have offered developmental psychopathology, there are several limitations to this conceptualization of adversity. Firstly, childhood adversities frequently co-occur (Dong et al., 2004; Finkelhor et al., 2007), making it impossible to determine whether an outcome of interest is attributed to a specific adversity or a cluster. Furthermore, a CR framework sums the total number of unique adversities experienced by a child to obtain a risk score whereby a child experiencing a risk profile characterised by sexual abuse and community violence would be treated homogenously to a child whose risk profile was characterized by poverty and neglect. The homogenisation of different types of environmental experiences and, in return, their effects on development, is a key limitation of this common approach. It is evident that there is scientific consensus on the potent influence of adversity on psychopathology. However, there exists much debate on the theoretical, methodological, and mechanistic underpinnings of both adversity and psychopathology. As researchers began to establish the graded relationship between adversity and developmental deficits, their attention shifted towards studying the mechanisms underlying this relationship, which has

predominantly focused on the stress-response system comprising the autonomic nervous system and hypothalamic-pituitary-adrenal (HPA) axis.

In this thesis, I first provide an overview of several theoretical frameworks that delineate the effects of childhood adversity on development and psychopathology by a focus on stress pathways, while highlighting its drawbacks as a universal mechanism. I then discuss major paradigm shifts in psychology and psychiatry research that argue for: a) Dimensional views of psychopathology; and b) Dimensional models of childhood adversity, namely the harshness-unpredictability and threat-deprivation frameworks. Through this overview, I delineate the theoretical underpinnings of mental health as it pertains to childhood adversity rather than an exhaustive review of studies related to psychopathology. Lastly, I provide a rationale for the integration of the two dimensional models and review the extant literature on childhood adversity and mental health in the Growing Up in New Zealand (GUiNZ) study.

1.1 Theoretical perspectives of childhood adversity

1.1.1 Stress as the underlying mechanism

Much research has been done on how early adversity becomes biologically embedded or 'gets under the skin' (Berens et al., 2017). The most widely studied mechanism mediating the effects of adversity on psychopathology concerns disruptions to stress response systems, specifically alterations in the hypo-thalamic-pituitary-adrenal (HPA) axis (Raymond et al., 2018). This neuroendocrine system produces cortisol, a steroid hormone otherwise known as glucocorticoid, that is produced to respond to and regulate physiological responses to stress (Silverman & Sternberg, 2012). An abundant literature has shown that adversity exposure results in disruptions to the HPA axis (Gunnar & Quevedo, 2007). Impaired functioning of the HPA axis due to adversity exposure has been strongly linked with several physical developmental outcomes (Heim et al., 2000), but the evidence of its association with psychopathology is less clear. Here I provide an overview of various theoretical models that

focus on the dysregulation of the stress-response system and corresponding evidence for its mediating role between adversity and psychopathology.

Proponents of the CR approach support disruptions to the stress response system as the primary mechanism underlying the relationship between adversity and development. Arguably, the Allostatic Load Model (ALM) is the prevailing conceptual model linking adversity, stress dysregulation and development. Briefly, the model puts forth the concept of *allostasis*, which refers to the body's ability to achieve equilibrium in its stress-response system (McEwen, 1998). Failure to effectively regulate these systems as a result of increasing environmental stressors leads to a short-term adaptation deemed the *allostatic state*, and in return, the chronic effects of remaining in this state are referred to as *allostatic load*, a measure of long-term 'wear and tear' on the body that increases the risk for disease and poorer development (McEwen, 2003). Evidence for increased allostatic load and its association with ACEs and poorer development has been seen for children (Bush et al., 2011; Danese & McEwen, 2012), adolescents (Doan et al., 2011), and adults (Finlay et al., 2022). These associations demonstrate a strong link between allostatic load and physical health outcomes, but do not entirely capture the relationship between adversity exposure and psychopathology. To delineate this inconsistency, researchers found that increased allostatic load was seen in individuals with psychological resilience as opposed to poor mental health (Brody et al., 2013). Another study showed differential associations between cortisol responses based on gender and neural circuitry but only within individuals with high externalising behaviours in the non-clinical range, suggesting a complex relationship between HPA axis regulation and neural activity and important considerations regarding the dimensionality of mental health (Konzok et al., 2021). This underscores many issues in the interpretation of stress system dysregulation as a universal mechanism for all types of developmental outcomes.

Other models grounded in evolutionary psychology, such as the biological sensitivity to context (BSCT), adaptive calibration (ACM), differential susceptibility, and diathesis-stress models, implicate both the stress-response system and individual variations as mediators between adversity and later psychopathology. Diathesis-stress models argue that vulnerability to psychopathology in poor environments varies according to an individual's susceptibility to different factors, or diatheses, which can be genetic, biological, or psychosocial (e.g., polymorphisms or temperament) (Monroe & Simons, 1991). Despite its popularity, the model is limited by its inability to explain differential effects that vary according to the environment. As such, BSCT and differential susceptibility theory extend the diathesis-stress model by suggesting that individual differences in stress responsivity can be adaptive or maladaptive in either poor or supportive environmental contexts (Belsky & Pluess, 2009), and some studies have shown support for this (Bolten et al., 2013). Recently, the ACM attempts to unify these concepts by adding an implicit developmental focus that emphasises different responses that improve inclusive fitness according to one's developmental stage (Del Giudice, Ellis, & Shirtcliff et al., 2011; Del Giudice, Hinnant, & Ellis et al., 2011). Importantly, the ACM is one of the few stress-based physiological models that accounts for sex differences, depicting different stress responses and adaptations for males and females in response to environmental adversity (Del Giudice, Ellis, Shortcliff et al., 2011). However, some research has shown that the effects of gender within the ACM did not entirely predict stress-response signatures when adversity was pervasive and severe (Doom et al., 2013).

Despite these theoretical advances, all of which have received varying support (see review, Doom & Gunnar, 2018), there are limitations in using stress-response system dysregulation as a universal mechanism underlying adversity and psychopathology. First, differential stress signatures have been identified for various psychiatric disorders (see

review, Koss & Gunnar, 2018) and, as such, there is a lack of understanding of when and how each type of dysregulation leads to pathology (Fries et al., 2008; Gunnar & Vasquez, 2001; Hellhamer et al., 1997). Second, as outlined by McLaughlin and Sheridan (2016), a stress perspective cannot universally explain the psychosocial and cognitive impacts of adversity and its relation to psychopathology, such as the lack of a stress-based explanation for language deficits as a result of adversity exposure (Farah et al., 2006). Further, one review highlighted no clear pattern between different stress-response signatures, type of adversity (deprivation or abuse), and associated psychosocial impairments (e.g., self-regulation) within the first five years of life (Wesarg et al., 2020). This highlights theoretical deficits in the stress perspective in accounting for age-appropriate associations and the influences of distinct types of environmental experiences. As such, stress models are not explicitly developmental and provide little understanding of the proximal mechanisms that explain the inconsistent relationships between adversity, stress-response system dysregulation, and psychopathology. Lastly, even if further research fills these gaps of knowledge, how efficient is a stress-based theoretical perspective in providing intervention targets? By targeting the stress-response system as a universal mechanism, researchers are limited in either developing interventions that regulate cortisol levels, which have demonstrated inconsistent efficacy (Slopen et al., 2014), or preventing adversity exposure entirely, which is a difficult target to achieve (McLaughlin, 2016).

Given the limitations of the stress response system as a universal mechanism, an exploration of other perspectives of adversity is worthwhile. Gabard-Durnam and Mclaughlin (2019) discuss the different features of adversity and underlying neurobiological assumptions that are emphasised by different theoretical frameworks. Based on their discussion, the models discussed above largely assert experience-dependent mechanisms as the underlying neurobiological mechanism of environmental experience, which reflects a non-

developmentally specific view of learning that occurs across the lifespan in response to environmental experience (Gabard-Durnam & McLaughlin, 2019). By focusing on the stress-response system, these models make the implicit assumption that all types of adversity trigger a developmental cascade through the same underlying mechanism and exert similar effects on the brain and other physiological systems. In contrast, other models of adversity emphasise different features of adversity, such as its timing or duration, and reflect experience-expectant mechanisms, which I elaborate on in the following section with respect to the stress-response system.

1.1.2 Programming the HPA axis: Sensitive versus critical period models

One feature of adversity that has been thoroughly investigated in the literature is the timing of adversity exposure. Given the established literature on heightened brain plasticity in early life, many have utilised sensitive or critical period models to investigate how different adversity exposures have differential effects based on their timing (Ismail et al., 2017). Although used interchangeably, critical period models refer to developmental windows where adversity exposure leads to irreversible or permanent effects that are unlikely to be modified by later environmental experiences; whereas a sensitive period model refers to a period where adversity exposure will have a stronger effect but can be modified by later experience (Ben-Shlomo & Kuh, 2002). From a neurodevelopmental perspective, these models rely on experience-expectant plasticity, whereby neural circuitry develops within specific developmental timeframes as a result of environmental experiences (Gabard-Durnam & McLaughlin, 2019).

The foetal origins of adult disease hypothesis, otherwise known as the Barker Hypothesis, perhaps best exemplifies a “critical period” model (see review, Barker, 2007). Based on geographical observations of areas with high infant mortality rates having much higher rates of cardiovascular disease decades later, Barker and colleagues demonstrated that

poor nutrition in utero and its corresponding effects on birthweight increase susceptibility to heart disease in adulthood, even when adjusting for lifestyle factors (Barker, 1986). Since then, many chronic diseases in adulthood have been shown to be biologically programmed during foetal development (Barker, 2007). Gluckman and Hanson (2006) have expanded the developmental time frame of this foundational work to a sensitive period model, most popularised by the 'First 1000 Days' campaign (www.thousanddays.org). Known as the Developmental Origins of Health and Disease framework, this evolutionary-developmental paradigm stresses the interplay of epigenetics, plasticity, and the mismatch between pre and postnatal environments in determining a predictive adaptive response, which is a survival strategy made by the developing organism (Gluckman et al., 2007).

While this body of work has been largely metabolic and nutrition-focused, the paradigms referred to above have also been applied to the study of mental health and neurodevelopment. For example, exposure to maternal psychopathology in utero has been strongly linked to mental health difficulties in offspring (D'Souza et al., 2016; Glover, 2015; O'Connor et al., 2003; Slykerman et al., 2015). In support, one meta-analysis reports programming of the HPA axis as a mediator between maternal prenatal stress and later mental health difficulties (Pearson et al., 2015). However, the stress-response signature associated with prenatal programming varies widely and has often been examined with psychiatric disorders in a categorical fashion (Koss & Gunnar, 2018), despite high comorbidity being the rule rather than the exception in mental illness (Kotov et al., 2017). Other research has applied a sensitive period model of adversity and psychopathology by focusing on epigenetic mechanisms (Conradt et al., 2018). There has been some evidence for epigenetic pathways between adversity and psychopathology, such as post-traumatic stress disorder (Parade et al., 2016), however, this field is still lacking an integrated approach that

translates epigenetic tissue modifications to neurobiological changes (Koss & Gunnar, 2018).

In two innovative studies, Dunn and colleagues tested the degree to which different life course models explain the association between adversity with DNA methylation (Dunn et al., 2019) and psychopathology at age 8 (Dunn et al., 2018). Results from both of these studies showed that a sensitive period model explained more variability in DNA methylation than accumulation or recency exposures (Dunn et al., 2019), however, an opposite pattern of results emerged when examining adversity-psychopathology relations, such that accumulation and recency models best explained this relationship instead of a sensitive period model. These findings highlight the complexity of applying a model that emphasises developmental timing to all types of adversity exposure, where the effects of poverty or neglect may be better accounted for by a sensitive period model rather than experiences of trauma, as it is difficult to identify periods where a brain expects to encounter harm (Gabard-Durham & McLaughlin, 2019). Furthermore, applying a sensitive period to adversity-psychopathology relations is difficult given that adversity can alter the developmental window itself, either accelerating or slowing development. Together, these results suggest that no single theoretical framework for adversity can best account for the elevated risk it poses for psychopathology.

Collectively, these models highlight the need for a more explicit developmental focus, as stress-based frameworks mentioned in the preceding section (e.g., ALM) are insufficient in explaining the relationship between adversity and elevated risk for psychopathology. While accounting for the timing of exposure provides additional information on the relationship between adversity and psychopathology, it does not consistently explain how these effects are translated into neurodevelopmental deficits that increase transdiagnostic risk for psychopathology. This is also compounded by the fact that HPA axis alterations do not

clearly map onto discrete psychological disorders. Given this challenge, in the following sections, expansions within psychopathology research are introduced that can aid our understanding of how adversity gets biologically embedded and poses transdiagnostic risk for psychopathology.

1.2 Dimensional models of psychopathology

1.2.1 Structure and statistical modelling of psychopathology

A key limitation of the existing literature on adversity and its effects on psychopathology is the examination of psychiatric disorders categorically, despite internalising and externalising disorders being highly comorbid (Caspi et al., 2020). While US population-based studies have shown that childhood adversity poses an elevated risk for virtually all common mental health disorders (Kessler et al., 2010), there have been some cross-national differences in this relationship. For example, research in Japan has suggested that physical abuse may be a specific risk for mood disorders as opposed to anxiety disorders (Fujiwara & Kawalkami, 2011). Adding to this, substance abuse disorders were most associated with sexual abuse and parental substance abuse in South Africa (Slopen et al., 2010), but with family violence and neglect in Nigeria (Oladeji et al., 2010). While these diverging findings may represent important cultural differences, they also represent a traditional psychiatric nosological lens that interprets the antecedents of psychiatric disorders according to the Diagnostic and Statistical Manual of Mental Disorders (DSM), which classifies mental disorders as distinct categories. This begs the question, would the effects of childhood adversity on psychopathology benefit from a transdiagnostic lens?

Recently, there has been a paradigm shift within psychology and psychiatry research that argues for a dimensional view of psychopathology, which favours a continuous spectrum rather than discrete diagnostic categories when examining mental illness (Lahey et al., 2021). Arguments for this paradigm shift have been discussed extensively (see Kotov et al., 2017).

In brief, advocates of a dimensional view argue that an abundant literature has shown that: a) Psychopathology exists along a spectrum (Wright et al., 2013); and b) Traditional diagnostic categories have low reliability (Kotov et al., 2017) and high comorbidity with other diagnoses (Widiger & Trull, 2007). One study reported that 36.8% of symptoms in the DSM-5 repeat 1,022 times across multiple diagnoses, with the most repetitive symptoms representing symptoms of major depressive disorder (Forbes et al., 2023). As such, there can be vast differences in the symptoms exhibited by individuals with a given psychiatric disorder, and also a substantial number of individuals who exhibit dysfunction but do not meet these diagnostic criteria (Kotov et al., 2017). The substantial overlap in symptomatology across psychiatric disorders and the use of strict cut-offs greatly impact intervention and treatment. As such, a quantitative movement has aimed to estimate the overall structure of psychopathology to shift away from these limitations.

Early research within this movement was conducted by Achenbach (1966) on a sample of adolescents, where he noted substantial covariance among internalising (primarily defined as anxiety and depression) and externalising (primarily defined as aggressive and delinquent behaviour) symptoms. Many studies have replicated this finding, showing that internalising and externalising symptoms are strongly correlated in samples of adults (Krueger, 1999) and even more so for child and adolescent samples (Cosgrove et al., 2011; Lahey et al., 2004). This formed the foundation for researchers to investigate a general dimension that accounts for the high correlation between these two spectra of disorders. Dubbed the "p-factor" and conceptually similar to the g-factor of intelligence, this general factor of psychopathology was first investigated in adult samples using different models of hierarchically structured constructs (Lahey et al., 2012; Caspi et al., 2014).

Figure 1 shows different hierarchical models used within psychopathology research. Several approaches guide the construction of these models, where some researchers use

confirmatory factor analysis to test a priori predictions about the structure of psychopathology (Patalay et al., 2015), and others use exploratory factor analysis to reveal a structure that best fits their data (Michelini et al., 2019). The correlated factors model (Model A) represents the traditional model used in psychopathology research, where specific factors typically represent internalising, externalising, and thought (i.e., psychotic) disorders, and are allowed to covary (Caspi et al., 2014). A bi-factor model (Model C) incorporates a second-order higher dimension (p-factor) that is orthogonal to the aforementioned specific factors, where each symptom loads onto a specific factor as well as a general higher factor. The p-factor within the bi-factor model represents the common variance shared by all symptoms being examined and conceptualises a transdiagnostic factor that is associated with all symptoms of psychiatric disorders (Lahey et al., 2021). In contrast, the higher-order factor model (Model B) allows symptoms to load onto each specific factor and in return, each specific factor loads onto the general factor. In this model, the p-factor represents the shared variance among different spectra disorders. Although the definitions of both general and specific factors are similar for interpretation purposes in bi-factor and higher-order models, bi-factor models have been used more frequently given the orthogonality of specific and general factors, which allows for tests of associations from specific and general factor scores with external variables, e.g., sociodemographic/clinical factors (Lahey et al., 2021)

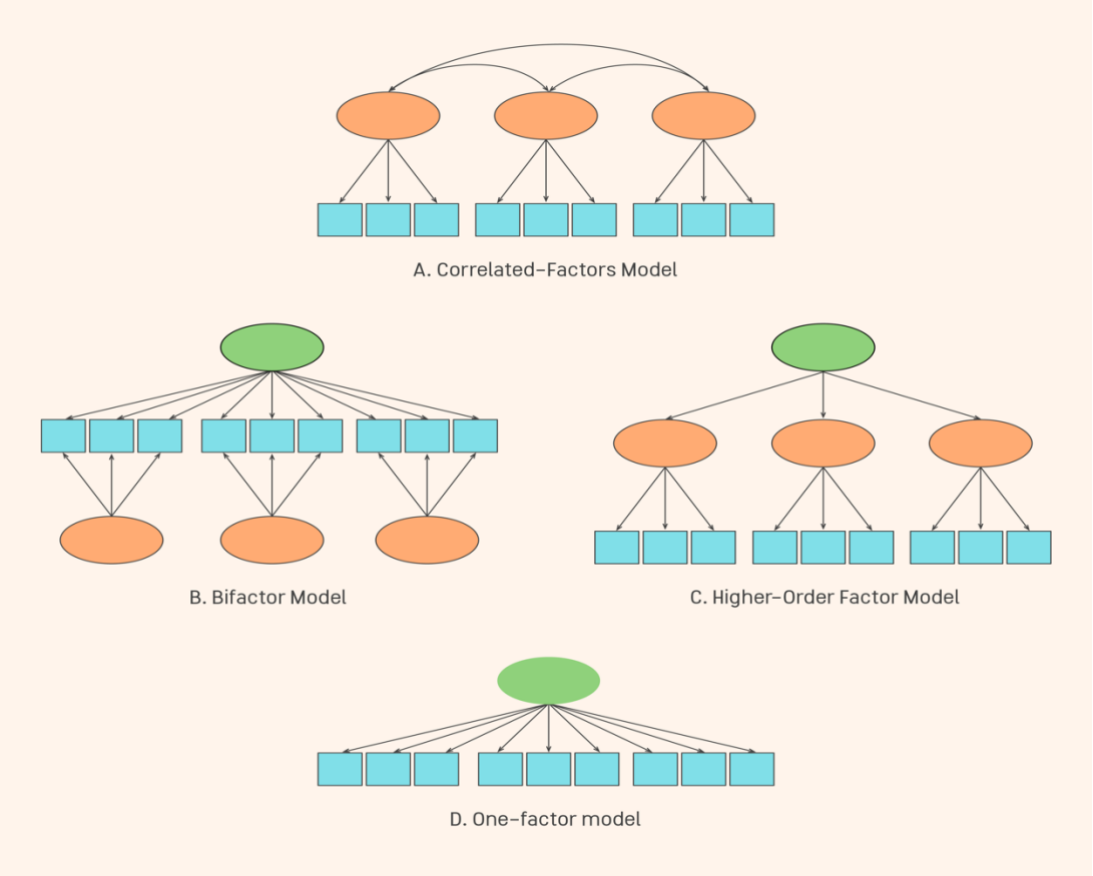

Figure 1. Hierarchically structured models of psychopathology. A) Correlated-Factors Model. B) Bifactor Model. C) Higher-Order Factor Model. D) One-Factor Model.

The bifactor models shown in Figure 1 represent symmetrical bifactor models, where each symptom or indicator loads onto a specific and general factor. Despite their popularity, some have pointed to anomalous results and inconsistencies in their application and interpretation (Forbes et al., 2021; Heinrich et al., 2023). As such, researchers have recommended an alternative modelling framework, the bi-factor (S-1) approach, which is argued to be a more theoretically sound approach to modelling the p-factor (Heinrich et al., 2023). In a bi-factor (S-1) model, researchers define the general factor a priori by setting a reference domain that only loads onto the general factor without loading onto a specific factor. For example, Figure 2 shows a bi-factor (S-1) model where externalising disorders are

set as a reference domain, and as such are only allowed to load onto the general factor (Heinrich et al., 2021).

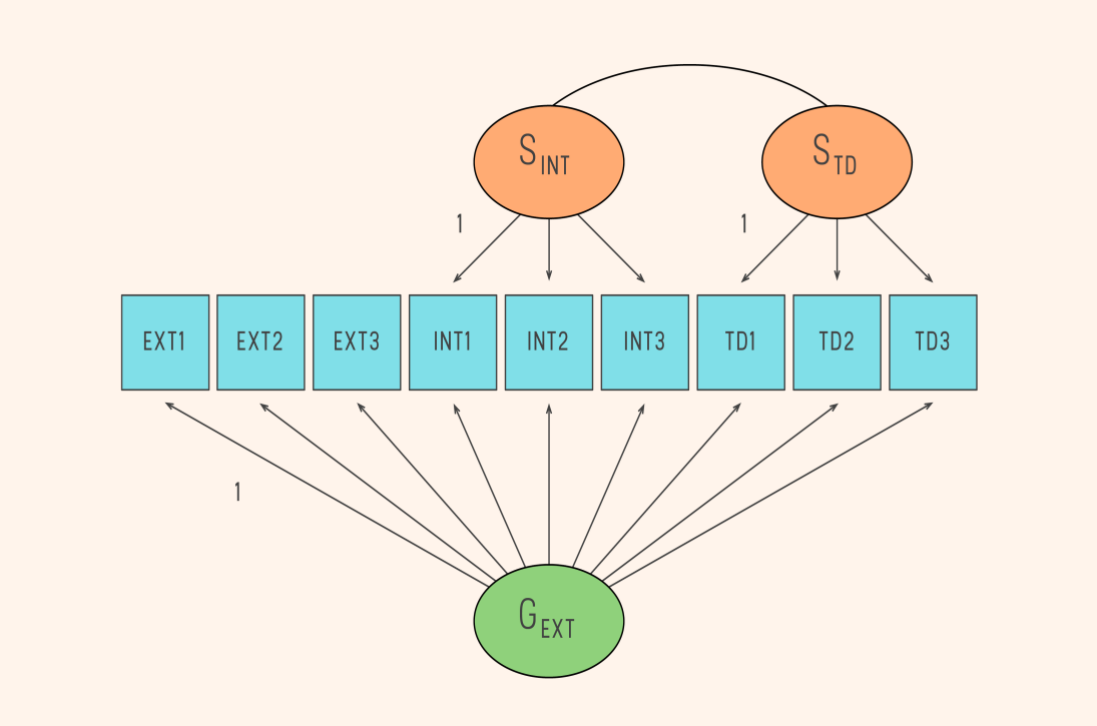

Figure 2. Bi factor S-1 Model with Externalising Disorders set as the reference domain. EXT = Externalizing; INT = Internalizing; TD = Thought Disorder; S_{INT} = Internalizing Specific Factor; S_{TD} = Thought Disorder Specific Factor; G_{EXT} = General Factor with Externalizing as reference.

The definition and operationalisation of the p-factor has been more consistent in samples of adults rather than in children and adolescents. Inconsistencies in the modelling of a p-factor across these samples are due to the wide range of content assessed for youth as opposed to a more cohesive assessment of psychopathology for adults. For example, the p-factor in adult populations when sampled using diagnostic interviews or self-report questionnaires, shows that it is primarily characterised by thought and internalising disorders (Caspi et al., 2014; Hyland et al., 2018; Kim & Eaton, 2015; Oltmanns et al., 2018). In contrast, the p-factor in samples of children and adolescents shows greater variability. In

middle childhood, the p-factor has been defined as primarily consisting of both internalising disorders (Lahey et al., 2015; Olino et al., 2018), and externalising and autistic disorders in other samples (Hankin et al., 2017; McElroy et al., 2018). This variability is also evident in adolescent samples, such that some find the p-factor to be marked more strongly by internalising disorders (Hamlat et al., 2019), externalising disorders (Murray et al., 2016), or thought disorders (Laceulle et al., 2015). This highlights the need for further research and integration of the p-factor across developmental periods.

1.2.2 Evidence for the p-factor

Many have highlighted issues with the p-factor, with critiques of this research arguing its nature as a statistical artefact, inappropriate use of bi-factor models, and lack of consistency across studies (Bonifay et al., 2017; Bornovalova et al., 2020; van Bork et al., 2017). Despite this, a large body of research has demonstrated the utility of this transdiagnostic measure of psychopathology in terms of the construct's structural and criterion validity, heritability, and stability over time (Smith et al., 2020). With these key strengths in mind, I discuss evidence for the p-factor in samples of children and adolescents, given the early developmental focus of this thesis.

The bi-factor model has been repeatedly shown to perform best in samples with children and adolescents, exhibiting better model fit than a correlated-factors model (Lahey et al., 2015; Patalay et al., 2015). Many different iterations of bi-factor models have exhibited good model fit and vary in the factors specified, with some measuring eating pathology (Forbush et al., 2010), and in fewer instances thought disorder/psychosis (Stochl et al., 2015). When included in models, symptoms of thought disorder tend to load the highest onto the p-factor (Caspi et al., 2014; Oltmanns et al., 2018). Bi-factor models have also been successfully constructed using both self-reported and proxy-reported data, and with categorical or continuous measures (Smith et al., 2020). In one study, researchers found that a

parent-reported p-factor predicted teacher-reported school functioning in adolescence (Lahey et al., 2015), showing the robustness of this construct. The p-factor has also been shown to predict life outcomes, such as future mental health difficulties (e.g., suicide attempts, psychiatric hospitalisations), criminal behaviour, and poor physical health (Laceulle et al., 2020; Pettersson et al., 2018; Sallis et al., 2020), over and above associations with specific factors and with no major differences in the strength of associations when using different informants of psychopathology. Lastly, further support for the criterion validity of the p-factor comes from evidence regarding its personality and cognitive correlates, such as high neuroticism and low IQ (Castellanos-Ryan et al., 2016; Olino et al., 2014).

Many theories exist to explain the p-factor's aetiology, such as those that are personality or temperament-based, which argue the p-factor reflects impulsive responsivity to emotion or dispositional negative emotionality (Carver et al., 2013; Tackett et al., 2013). Other interpretations rely on cognitive mechanisms and suggest the p-factor is a result of low intellectual functioning or thought dysfunction (Caspi & Moffitt, 2018). There is preliminary evidence for each of these interpretations (see review, Smith et al., 2020); however, it is difficult to reconcile any of these interpretations as the core underlying mechanism for *all* symptoms reflected in the p-factor. Recently, Southward et al. (2023) tested several leading theories regarding the interpretation of the p-factor, and reported findings that suggest a tripartite definition of the construct including neuroticism, impulsivity, and impairment. In parallel, a growing research has shown strong evidence for the genetic basis of the p-factor (Allegrini et al., 2020; Lahey et al., 2011; Pettersson et al., 2013). For example, one study reported moderate heritability of the p-factor in an adolescent sample ($H^2 = 0.43$), demonstrating the construct's validity (Lahey et al., 2017). Therefore, while there is debate and ongoing research on the causal mechanisms for the p-factor, its heritability suggests that it is a construct worth investigating.

Other evidence for the validity and robustness of the p-factor is its substantial stability across the lifespan. Despite differences in the symptoms measured across models and the use of symptom or disorder-level data, studies consistently report high stability of the p-factor across childhood (McElroy et al., 2018), adolescence (Castellanos-Ryan et al., 2016; Riley et al., 2019), and adulthood (Greene & Eaton, 2017). Furthermore, one study showed strong homotypic continuity for specific and general factors, such that general, internalising, and externalising factors respectively predicted general, internalising, and externalising factors 1.5 years later (Snyder et al., 2017). While these results showed strong homotypic continuity, there are other competing hypotheses regarding the developmental trajectory of the p-factor, namely p-differentiation and dynamic mutualism. P-differentiation argues that the p-factor represents a general disposition to psychopathology that becomes more specific over time, whereas dynamic mutualism argues that the p-factor is the manifestation of different symptoms reinforcing each other to establish long-term comorbidity (Murray et al., 2016; McElroy et al., 2018). Both hypotheses have received preliminary support (McElroy et al., 2018), yet further research is needed to understand the specific dynamics of how this transdiagnostic measure unfolds over time.

There have been some attempts to synthesise research on bi-factor models, both across different iterations of the models and across samples. Levin-Aspenson et al. (2021) were able to establish bi-factor models with good fit across several large psychiatric epidemiological studies, but found differences in the interpretation of the p-factor and reliability of specific factors across samples. In another study, researchers tested different variations of bi-factor models using the Child Behaviour Checklist (CBCL), and showed similarity across these models in terms of good model fit and factor loadings, but differences in the reliability of general and specific factors (Hoffman et al., 2022). This highlights the

need for cautious interpretation of the p-factor across studies and theoretically sound external validation analyses.

As with the CBCL, many studies have tested different hierarchical structural models using the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 2001). Using data from a large-scale study based in Britain, Goodman et al. (2010) conducted a structural analysis of the SDQ and found that using the prosocial scale along broader internalising and externalising dimensions, assessed via the peer/emotion and conduct/hyperactivity subscales respectively is more appropriate in low-risk samples, whereas using all five subscales adds additional value in clinical samples. Fernandez De la Cruz et al. (2018) lend support to these findings using two large paediatric clinical samples from England and Norway, showing that a 5-factor solution, where all specific subscales of the SDQ are correlated, provide the best model fit and discriminant validity between psychiatric disorders. In another study, researchers used an Item Response Theory Analysis of the four 'difficulties' subscales in the SDQ (peer, emotional, conduct, and hyperactivity problems), and showed that there was evidence for a strong general factor in bi-factor models (Keller & Langmeyer, 2019).

Many studies have shown support for different iterations of bi-factor models using the SDQ, such as support for both a bifactor model with one general factor and 5 correlated specific factors in a sample of 8-13 year olds using parent and teacher versions of the SDQ (Kobor et al., 2013). Similarly, a bifactor model with two correlated general factors and 4 specific factors proved a good fit for a sample of 4-17 year olds (Caci et al., 2015). In contrast, some studies supplemented the SDQ with other measures and dichotomised items to increase statistical power, and subsequently showed that bi-factor models with one general factor and three specific factors (internalising, externalising, and thought disorder) showed good fit (Afzali et al., 2018; Carragher et al., 2016). Lastly, Chen et al. (2022) showed support for a bi-factor model using parent and adolescent self-reported SDQ with one general

factor and four specific factors, however relied solely on model fit indices. Given the range of models tested and the inconsistent reporting of reliability indices, comparison across different studies is difficult. Further, the inclusion of the strength-based prosocial scale in some bi-factor models presents issues in interpretation given reported problems with reverse-coded items (van de Looij-Jansen et al., 2011). Collectively, these findings highlight the need for further research regarding the utility of bi-factor models using the SDQ total difficulties subscale, which are informed by recent structural analyses (Goodman et al., 2010; van de Looij-Jansen et al., 2011), and appropriate reporting of relevant statistical indices for bi-factor models (Rodriguez et al., 2016).

Given that other studies have successfully tested different structures of psychopathology using behavioural and mental health screening questionnaires (Carragher et al., 2016; Patalay et al., 2016), GUiNZ data provides an opportunity to test this dimensional approach using a rich dataset which allows for sound external validation analyses. It is important to note from the outset that the present study uses the term psychopathology to refer to mental health and behavioural difficulties for the GUiNZ cohort, which is not meant to insinuate that the present study is measuring diagnosed or severe psychopathology, but is in reference to the statistical techniques used within this line of research. Also worth noting is potential issues with the use of one scale such as the SDQ to represent a transdiagnostic measure of psychopathology, which may reflect issues within the psychometric properties of the instrument itself. However, the current literature supports the use of broader internalising and externalising scales of the SDQ in low risk and/or community samples (Goodman et al., 2010), and might shed light on the gaps in integrating p-factor research within community and epidemiological samples for earlier developmental periods.

Next, I overview dimensional models of childhood adversity, beginning with a definition of childhood adversity, and highlighting how a transdiagnostic measure of

psychopathology has been already employed in childhood adversity research, and the ways in which it benefits the field.

1.3 Dimensional models of adversity

1.3.1 Defining childhood adversity

In their foundational paper, Felitti et al. (1998) defined ACEs as "childhood abuse and household dysfunction", which has been argued to be both vague and constraining to explicit maltreatment, parenting practices, and associated household chaos (Hill, 2019). Since then, many have called for a consensus on the definition of childhood adversity, which would allow comparisons across studies and also provide the ability to distinguish between concepts like trauma, stress, and toxic stress (Mclaughlin, 2016). One definition that is gaining popularity in the literature defines childhood adversity as experiences that represent a deviation from an expected environment and which would likely require a significant adaptation by an average child (Mclaughlin, 2016). The 'expectable' environment in this sense refers to the necessary inputs required for healthy development, which can be disrupted due to the presence of unexpected inputs (e.g., sexual abuse) or the absence of expected inputs (e.g., cognitive stimulation). However, the disruption to the expectable environment is only referred to as adversity if it is likely that an average child would have to significantly adapt to it, presumably in terms of either neurobiological or psychosocial adaptation (Mclaughlin, 2016). Therefore, this definition establishes the type of environmental experience that would qualify as adversity, but makes no assumptions about the necessary response of the individual exposed to such an experience or its temporal specificity. In contrast, stress and toxic stress respectively address the response of the individual and the duration of this response, where stress refers to the adaptation of an individual to circumstances over time and toxic stress refers to the duration and cumulative effect of this response (Humphreys & Zeanah, 2015; Monroe, 2008; Shonkoff & Garner, 2012). Lastly,

trauma can be distinguished as a specific type of environmental experience, one where an individual is exposed to or witnesses threat or actual death, injury and violence (American Psychiatric Association, 2013).

With this definition of childhood adversity in mind, in the following section two dimensional models of adversity are explored that distil myriad adverse experiences into their active ingredients, beginning with the Dimensional Model of Adversity and Psychopathology (DMAP).

1.3.2 Dimensional Model of Adversity and Psychopathology (Threat-Deprivation framework, DMAP)

The DMAP provides a mechanistic framework to understand the effects of adversity on neurodevelopment and psychopathology (Mclaughlin et al., 2014). This is achieved by a neurobiologically based analysis centring key cognitive neuroscience principles, such as the role of experience-driven plasticity and its influence on brain development (Mclaughlin & Sheridan, 2014). Decades of neuroscientific research have shown that early life is a period with heightened brain plasticity, characterised by the interplay of genetic and environmental factors that shape brain development (Kolb & Gibb, 2011). As illustrated in previous sections, experience-driven plasticity can be experience-expectant, whereby neural circuitry develops as a result of specific environmental stimuli within specific developmental windows, or experience-dependent, which reflects learning across the lifespan (Gabard-Durham & McLaughlin, 2019). Using these principles, the model re-conceptualises previous perspectives by emphasising the importance of learning in response to environmental cues and its effects on the development of neural circuitry.

Whereas stress perspectives provide a blanket explanation for the ways in which adversity would influence neurodevelopment and later psychopathology, this model mechanistically outlines how ACEs can be categorised along distinct core underlying

dimensions that exert unique influences on learning and corresponding brain development (Mclaughlin et al., 2014). The framework proposes that multiple types of adversity can be distilled along the core dimensions of *threat* and *deprivation*, while also highlighting the possibility of other plausible dimensions and the likelihood that they co-occur (Mclaughlin & Sheridan, 2016). Many studies have provided support for the DMAP by directly comparing it to specificity and cumulative approaches, highlighting how the latter methodologies may obscure specific associations between distinct environmental experiences and psychopathology (Henry et al., 2021; Stein et al., 2022). Distinctions between the core dimensions of threat and deprivation will be elaborated on below, relying on an accumulating literature on the downstream effects of learning disruptions on development and how they relate to the onset of psychopathology.

Threat refers to environmental experiences that expose a child to actual or threat of harm to their survival and includes either being the target of or witnessing said experience (Mclaughlin et al., 2014). Consistent with the definition of trauma (APA, 2013), threatening experiences are hypothesised to alter neurobiological development, most importantly in terms of social information processing, such as threat detection and emotion regulation (Mclaughlin & Sheridan, 2014; Trautmann et al., 2022). Many studies have supported alterations in socioemotional development as an underlying mechanism linking threat and later psychopathology. In a series of foundational studies by Pollak and colleagues, children exposed to maltreatment exhibited distinct emotion recognition patterns in ways that facilitated the rapid identification of threat, such as increased accuracy in identifying angry faces (Pollak & Sinha, 2002; Pollak et al., 2009). In return, these distinct socioemotional processing patterns have shown associations with many psychiatric disorders (Briggs-Gowan et al., 2016; Dodge et al., 1990). Previous research has also connected threat-related attention bias to emotion regulation (Cisler & Koster, 2010), an established transdiagnostic mechanism

that conveys overall risk for psychopathology (Aldao et al., 2016). Further, one study reported that emotion regulation mediated the relationship between child maltreatment and the p-factor longitudinally (Weissman et al., 2019). Another study showed that threat, but not deprivation, was associated with greater externalising problems in adolescence through differences in social information processing, such as affective theory of mind (Heleniak & McLaughlin, 2020).

Neuroscientific evidence provides substantial support for the threat dimension in the DMAP, where one systematic review highlighted the associated neural systems disrupted due to threat. These include the: 1) fronto-amygdala circuit, which comprises the amygdala and medial prefrontal cortex (mPFC); 2) hippocampus; and 3) salience network, which comprises the anterior insula, dorsal anterior cingulate cortex (dACC), and supplementary motor area (Mclaughlin, Weissman, & Bertran et al., 2019). The review found strong evidence for the DMAP and its predictions of neurodevelopmental consequences following experiences of threat. Children exposed to threat were consistently shown to have reductions in amygdala, mPFC, and hippocampal volume (Edmiston et al., 2011; Hanson et al., 2010; Hanson et al., 2015; Saxbe et al., 2018), increased amygdala reactivity to threatening stimuli (McCrory et al., 2013), and differences in the salience network evidenced by greater activation of the anterior insula (Cisler et al., 2019; Marusak et al., 2015) and reduced dACC response (Weissman et al., 2019). Importantly, these patterns follow the DMAP's predictions and were not consistently seen as a result of deprivation exposure, suggesting a distinct influence of threatening experiences on the brain.

In the DMAP, *deprivation* encompasses limited age-specific environmental complexity in terms of cognitive and social environmental inputs, which can range from being entirely absent to insufficient (Mclaughlin & Sheridan, 2014). As such, deprivation can be represented by a lack of psychosocial or cognitive enrichment, and on the more severe end

of the spectrum, the absence of a primary caregiver due to institutional rearing (Mclaughlin et al., 2014). The DMAP predicts that experiences characterised by deprivation will alter neural circuitry associated with cognitive ability. The role of the environment in shaping neural circuitry is especially critical within the first few years of life when brain development is at its most malleable state. Early brain development is characterised by periods of proliferation and pruning, where the former is characterised by rapid overproduction of synaptic connections between neurons to lay the foundation for future learning, and the latter being the refinement of these connections through selective elimination guided by both genetic and environmental factors (Changeux & Danchin, 1976; Petanjek et al., 2011). As such, development is both cumulative and hierarchical, and heightened periods of plasticity may be both harmful and beneficial to a child. In the case of deprivation, the absence of required cognitive/social inputs for healthy development can lead to fewer learning opportunities that, in turn, might alter cognitive processes which are used to sculpt even more complex cognitive ability later (Ellis et al., 2022). Similarly, experience-expectant mechanisms may be at play that eliminate synaptic connections that are underutilised due to reduced opportunities of learning (Ellis et al., 2022).

There has been consistent evidence that deprivation exerts an overall risk for future psychopathology and that this relationship is specifically mediated by alterations in cognitive ability. Firstly, experiences of deprivation, ranging from more severe such as institutionalisation to less severe ones like low socioeconomic status (SES), have been associated with increased risk for many forms of psychopathology (Humphreys et al., 2020; Peverill et al., 2021). The DMAP's prediction that deprivation, but not threat, is associated with heightened risk for psychopathology through deficits in cognitive functioning has been well supported. In two studies, Miller and colleagues showed that when examining threat and deprivation simultaneously, only deprivation exerts risk on future psychopathology through

language ability (Miller et al., 2018; Miller et al., 2021). This pattern of results is also seen in preschool children when using SES as a proxy for deprivation rather than measures that reflect direct cognitive stimulation in the immediate rearing environment (Wade et al., 2021). Studies that examined both deprivation and threat simultaneously have shown that only deprivation, measured using proxies such as low SES and parental education, is associated with deficits in executive functioning (Sheridan et al., 2020; Vogel et al., 2021) and response inhibition (Machlin et al., 2019). Research has also shown that assessment methods are important in unveiling these associations, where one study reports associations between both threat and deprivation with teacher-reported inhibitory control (Wolf & Suntheimer, 2019), whereas one meta-analysis reports a stronger association between deprivation and objectively assessed executive function (Johnson et al., 2021).

A systematic review has shown that experiences characterised by deprivation are associated with alterations in neural circuitry implicated in cognitive development, namely in the frontoparietal and striatal circuits (McLaughlin et al., 2019). Children exposed to deprivation, but not threat, consistently showed significant differences in the structure of the frontoparietal network, namely in terms of reduced thickness and volume of the dorsolateral prefrontal cortex (Edmiston et al., 2011; Herzberg et al., 2018; Hodel et al., 2015; McLaughlin et al., 2014). Additionally, children who experienced extreme kinds of deprivation, such as institutionalisation, consistently displayed reduced total grey and white matter volume (Mehta et al., 2009; Sheridan et al., 2009). Lastly, in studies investigating striatal activation in tasks involving reward processing, children exposed to deprivation consistently showed reduced dorsal and ventral striatum recruitment (Goff et al., 2013; Hanson et al., 2015; Mehta et al., 2010), with an opposite pattern for children exposed to threat (Dennison et al., 2016). Importantly, these results align with the predictions of the

DMAP even though the studies were not designed to directly test for the differential impacts of different types of adversity.

There is a substantial evidence base for the DMAP which supports its neurobiological analysis of the differential effects of threat and deprivation and its integration with transdiagnostic mechanisms. Using principles of experience-driven plasticity, this framework provides a mechanistic understanding of the relationship between adversity and psychopathology, highlighting how distinct neurodevelopmental adaptations as a result of different environmental experiences contribute towards poorer development (McLaughlin & Sheridan, 2014).

1.3.3 Harshness-Unpredictability Model

Similar to the DMAP, the Harshness-Unpredictability framework uses evolutionary psychology principles to categorise ACEs into their core dimensions. This framework is rooted in life history theory, which looks at how organisms allocate their time and energy towards life cycle goals, such as physical/cognitive growth and reproduction (Ellis et al., 2009). The model is fundamentally concerned with the 'why' of adverse development, more specifically why development operates the way it does in response to adversity and how this ties into our history of natural selection (Ellis et al., 2022). A key strength of this model is that it challenges the traditional deficit-based approach to childhood adversity, and argues that 'maladaptive' development occurs to improve evolutionary functioning rather than impair it, given its adaptiveness under conditions of early life stress (Belsky et al., 1991; Ellis et al., 2022). While the Harshness-Unpredictability model focuses on predicting an individual's behaviour and health in relation to their reproductive fitness, it also offers a unique evolutionary view into vulnerability for psychopathology.

Enhancing one's reproductive fitness across the lifespan involves distinct trade-offs between life cycle goals, such as those between bodily maintenance and growth, current and

future reproduction, or those between offspring quality and quantity (Ellis et al., 2009). These trade-offs occur due to different developmental and environmental experiences and are understood via variations in life history traits, which relate to one's reproductive fitness and are phenotypically reflected by mediators such as biological ageing and parenting practices (Ellis et al., 2022). A life history strategy represents the collection of different life history traits and is typically represented on a slow-fast continuum, which has been well-supported in human research (Ellis et al., 2009). There are key differences between slow and fast life history strategies in terms of reproductive timing/effort and other phenotypic mediators, whereby those with a slow strategy have delayed reproduction and sexuality, invest more heavily in offspring, low impulsivity, and are focused on maximising long-term survival (Belsky et al., 1991). An opposite pattern emerges for those with a fast strategy, which is typically characterised by early reproduction, high number of sexual partners, and risky and aggressive behaviour (Del Giudice et al., 2015).

Ellis and colleagues propose that environmental harshness and unpredictability are distinct dimensions of early life that regulate individuals' life history strategies (Ellis et al., 2009). Their model argues that evolutionary history favoured different life history strategies in response to these frequently occurring and adverse environmental experiences. In addition to the dimensions of harshness and unpredictability, the model views development as hierarchically based on energy availability, given the role that energy expenditure and caloric intake have on development (Ellis et al., 2022). As such, variations in life history traits are contingent first on stressors directly related to bioenergetic resources, and when health conditions are satisfied, rely on psychosocial circumstances (Ellis et al., 2022).

In the Harshness-Unpredictability framework, environmental harshness refers to extrinsic sources of morbidity-mortality, such as poverty, family violence, and famine, which collectively correspond to a greater risk of injury, illness, or death (Ellis et al., 2022). A key

feature of this dimension of adversity is that the risk of morbidity-mortality from these environmental experiences either cannot be prevented or that there is a low return on investment in attenuating such risk (Andre & Rousettt, 2020). For example, greater investment in offspring in environments characterised by high levels of harshness (e.g., war) will have little impact on increasing survival or reducing the risk of death or injury. The model makes the prediction that environments with high levels of harshness will be associated with faster life history strategies, exhibited by life history traits such as greater number of sexual partners and high impulsive and aggressive behaviour (Ellis et al., 2009). Importantly, this dimension also diverges from the threat-deprivation framework by using distal ecological cues that our evolutionary history has sensitised us to detect (Ellis et al., 2022). For example, research has often operationalised harshness as socioeconomic status (Chen et al., 2002), but also with more direct indicators of morbidity-mortality such as family death or neighbourhood danger (Chang et al., 2019). This literature has largely supported that cues of harshness are associated with faster life history strategies (Chang et al., 2019; Gettler et al., 2015; Nettle, 2010).

There has been some preliminary support for the consequences of harshness on psychopathology. In one study, researchers found that those with traits corresponding to that of a faster life history strategy have higher levels of general psychopathology, especially in terms of paranoid ideation, interpersonal sensitivity, and depression (Kahl et al., 2022). Another study found support for the mediating role of life history traits, such that childhood trauma was associated with greater overall psychopathology through accelerated pubertal development (Colich et al., 2020). Adding to this, researchers also found support for the role of parenting practices as a mechanism underlying the relationship between income harshness and externalising behaviours (Li & Belsky, 2022). One study found that threat, but not deprivation-based forms of harshness, was associated with greater DNA methylation age and

advanced pubertal stage, and that DNA methylation age mediated the relationship between threat-harshness and depressive symptoms (Sumner et al., 2019). Lastly, broader ecological indicators of harshness in early life, such as neighbourhood poverty, are shown to be predictive of neural architecture in adolescence, with specific associations with the fronto-parietal, cortico-limbic, and salience systems (Michael et al., 2023), which have been linked to internalising symptoms (Brieant et al., 2021).

Unpredictability is best defined as the random variation in harshness, such that levels of harshness in an environment can be predictable or unpredictable (Ellis et al., 2009). Unpredictable environmental conditions are characterised by low autocorrelation and cue reliability, meaning that the unpredictable environmental condition must not correlate with itself over time and provide little predictive utility about future environmental conditions (Ellis et al., 2022). The central tenet behind unpredictability as a dimension of adversity is that it is associated with future-discounting of costs and benefits, and as such is associated with faster life history strategies (Hill et al., 1999). There are two ways to classify and measure unpredictability, namely a statistical learning or ancestral cue perspective. The former involves directly measuring environmental parameters to detect the underlying statistical structure of one's environment (Li et al., 2018), whereas the latter involves using privileged sources of information from the environment which individuals are sensitised to given our evolutionary past (Young et al., 2020). Within a Western context, the ancestral cue perspective is often operationalised variations in parental transitions and household/neighbourhood chaos, using indicators like the number of parental relationships or residential moves, or changes in neighbourhood safety and family stress (Young et al., 2020). Many of these different ways of measuring unpredictability have been associated with indicators of a faster life history strategy, such as greater aggressiveness (Simpson et al., 2012) and sociosexuality (Szepsenwol et al., 2017).

Despite the differences in operationalizing and measuring unpredictability, research consistently shows associations between this dimension and risk for psychopathology. For example, some studies measure unpredictability as variations in constructs related to chaos or instability on a household, socioeconomic, or neighbourhood level. When operationalized this way, unpredictability measured by variations in caregiver consistency and childhood SES was associated with greater socioemotional problems (Evans et al., 2005) and overall mental health difficulties (Dwairy et al., 2008; Hurst & Kavanagh, 2017). Moreover, one study showed that early life unpredictability measured by changes in parental residence/relationships was associated with externalising and criminal behaviours in adulthood via externalising behaviours at age 16 years (Doom et al., 2016). Notably, when multiple proxies of unpredictability are used, such as household disorganisation and instability, factors related to household chaos tend to be more associated with greater externalising behaviours (Deater-Deckard et al., 2009; Mills-Koonce et al., 2016). Another important consideration is the inclusion of maternal depression as a marker of unpredictability. While not typically used to index unpredictability, many studies have shown that maternal depression poses elevated risk for developing multiple forms of psychopathology across the lifespan and cognitive disadvantage via unpredictable maternal signals (Halligan et al., 2004; Glynn & Baram, 2019; Verbeek et al., 2012). Further, maternal depression has shown strong associations with household chaos (Andrews et al., 2022; Hur et al., 2015; Marsh et al., 2020) and executive functioning (Gueron-Sela et al., 2018). Collectively, these findings highlight the transdiagnostic risk that multiple forms of unpredictability pose for psychopathology; and the ongoing efforts in appropriately capturing and measuring this dimension.

There is mixed evidence for the underlying mechanisms of unpredictability and its influence on psychopathology. This research emerges within the context of the Hidden

Talents approach, which is derived from life history theory, and attempts to characterise alterations to cognitive abilities that are adaptive under harsh and unpredictable early environments but are linked to greater psychopathology (Ellis, Abrams, & Masten., 2022). A recent meta-analysis highlighted small but significant reductions in child executive functioning in relation to greater household instability and disorganisation, with stronger associations when using household instability and informant-reported executive functioning (Andrews et al., 2021). Whereas deprivation, and in some instances threat, are typically associated with reduced cognitive ability (Johnson et al., 2021), there is also evidence for cognitive enhancements due to unpredictability (see Wade et al., 2022). Some of these adaptations include associations between greater unpredictability and enhanced attention shifting (Fields et al., 2021; Mittal et al., 2015) and working memory (Nweze et al., 2021; Young et al., 2018). Notably, many of these studies recruited participants from severely deprived environments and point to the possible mitigating effect of unpredictability on cognitive ability when deprivation is pervasive and severe. In another study, effortful control was negatively predicted by harshness (neighbourhood and parenting) but positively predicted by unpredictability (parental transitions) (Warren & Barnett, 2020). Despite this, some of these studies are limited by biased measurements of cognitive ability and retrospective assessment of early adversity.

There has also been some research on the neural correlates of unpredictability that correspond with the cognitive adaptations discussed above. Much of this work has been informed by animal models that simulate unpredictability by experimentally manipulating rodent maternal signals, showing neurobiological adaptations in regions that underlie cognitive and emotional processes, such as the amygdala, hippocampus, and medial prefrontal cortex (Bolton et al., 2017; Gee, 2021; Liu & Fisher, 2022). Analogous to animal models, human studies have used unpredictability measures coded from mother-child dyadic

interactions to study neurobiological adaptations. In one of these studies, greater unpredictability via maternal signals was associated with imbalances in the medial temporal lobe prefrontal cortex (Granger et al., 2021). On the other hand, unpredictability indexed by household chaos and financial instability shows a more complex relationship. In a sample of adolescents, household chaos moderated the relationship between parental monitoring practices and greater insular cortex activation (i.e., brain regions attributed to salience processing and cognitive flexibility, see Uddin, 2021) only in low chaos households (Lauharatanahirun et al., 2018). The findings illustrate how the effects of parental control on adolescent risky behaviour are mitigated in environments characterised by high levels of unpredictability. Similarly, Kim-Spoon et al. (2017) showed that parental control was associated with enhanced cognitive control (measured via neural activation of regions of the frontoparietal network during a task) for adolescents but only in low chaos environments, whereas only those in high chaos environments showed associations between poor cognitive control and social and emotional problems.

The Harshness-Unpredictability framework provides a dimensional view of adversity and its influences on psychopathology and development from an evolutionary perspective. Many studies have adopted this framework and perspective when examining adversity, highlighting how environmental experiences can influence life traits towards that of a faster life history strategy. However, further research is needed to understand the complex neurobiological adaptations associated with unpredictability in particular, and the methodological implications that come with measuring this dimension. Next, I turn towards a rationale for the integration of the aforementioned dimensional models discussed, using key integrative concepts as conceptualised by Ellis et al. (2022) and empirical articles that have looked at dimensions from either dimensional model in tandem.

1.4 Thesis Rationale: Integrated dimensional model

Although developed separately, Ellis et al. (2022) discuss a rationale for the integration of the DMAP and Harshness-Unpredictability frameworks. Their rationale for this integration lies in the complementarity between their frameworks, such that an evolutionary analysis of the DMAP and a neurobiological analysis of the Harshness-Unpredictability framework, provide a cohesive argument for *which* dimensions of early adversity are of importance, and *why* and *how* these dimensions confer risk for adverse development. A key strength of this integrated model is its developmental significance as it pertains to psychopathology, given that it can explain concepts of equifinality, where different early experiences lead to similar outcomes, and multifinality, where similar early experiences lead to different outcomes (Gee, 2021). Similarly, a dimensional view of psychopathology will aid our understanding of how childhood adversity poses transdiagnostic risk through mediators that convey risk for all spectra of disorders. To illustrate the benefits of this integrated model and dimensional view of psychopathology, here below I first review GUiNZ research that has investigated these two topics, and I discuss how a dimensional view will circumvent some of these limitations. I also address the limitations of a dimensional view in terms of measurement and its focus on the environment rather than biology.

An integrated model of adversity leverages conceptual differences between two different models in a theoretically compatible manner. Firstly, the harshness dimension can be categorised into greater extrinsic morbidity-mortality due to the presence or absence of specific environmental inputs, thus can be theoretically mapped onto the threat and deprivation dimensions in the DMAP. Second, the DMAP and Harshness-Unpredictability frameworks address different levels of the environment, with the former focusing on adversity within the immediate caregiving environment and the latter within the broader ecological context. This also allows the examination of multi-level adaptations to the

environment, such as concurrent adaptations (e.g., social information processing biases), and proximal (e.g., inflammatory processes that increase impulsivity, Gassen et al., 2018) and distal (e.g., accelerated pubertal development) ecological adaptations (Ellis et al., 2022). Lastly, the integration of these two models relies on a strong theoretical foundation that grounds neurodevelopmental principles like experience-driven plasticity within an evolutionary-developmental perspective (McLaughlin et al., 2021). This allows for testable hypotheses on the links between different dimensions of adversity, transdiagnostic mediators, and psychopathology, using an explicitly developmental approach that incorporates multiple theoretical perspectives.

There has been limited research on the neurobiological effects of threat, deprivation, and unpredictability measured simultaneously. Chahal et al. (2022) investigated the effects of these dimensions on the functional architecture and connectivity of brain networks in a longitudinal study of adolescent participants. Results showed differential effects of each adversity dimension, such that deprivation showed widespread differences in network development across cognitive and emotional processing circuits, and more focal differences for threat (frontoparietal and default mode connectivity) and unpredictability (salience network connectivity) (Chahal et al., 2022). Further, they reported small increases in internalising symptoms associated with changes in network connectivity for youth who had high levels of adversity of any dimension. Despite this, others report conflicting findings in the relationship between adversity dimensions, psychopathology, and the mediating role of functional connectivity of different neural circuits (Goetschius et al., 2020; Rakesh et al., 2021).

Few studies have examined threat, deprivation, and unpredictability simultaneously, which limits the ability to disentangle the unique effects of each adversity dimension to their hypothesised underlying mechanism and link to psychopathology. In addition, not all of these

studies have investigated the simultaneous effects of these adversity dimensions with respect to psychopathology. Rosen et al. (2019) highlighted distinct associations between each dimension of adversity and various cognitive abilities, reporting significant associations between violence exposure and household instability with associative memory and memory-guided attention accuracy, respectively. Notably, their findings show that early cognitive stimulation (i.e., deprivation) and household instability (i.e., unpredictability) are no longer significantly associated with cued attention accuracy when adjusting for all adversity dimensions simultaneously (Rosen et al., 2019). Similarly, Phillips et al. (2023) examined the simultaneous effect of deprivation and unpredictability, on preschool executive control and its association with later psychopathology. When examining these two dimensions simultaneously, only deprivation continued to be significantly predictive of adolescent psychopathology via preschool executive control. While these results add to the literature, they are limited given the inability to simultaneously control for threatening experiences. Another study tested the developmental correlates of threat, deprivation, and unpredictability, showing links with aggressive behaviour, health problems and cognitive ability, and substance use and risky sexual behaviour, respectively (Usacheva et al., 2022). In contrast, Ning et al. (2023) demonstrated mediating effects of cognitive ability and emotion regulation on psychological distress via exposure to adversity but with no distinct effects of threat or deprivation, potentially reflecting reporting bias or developmental considerations of psychopathology. Lastly, findings from a two-decade longitudinal study lend support for the proximal mechanisms suggested by the DMAP and Harshness-Unpredictability framework, showing that: a) only deprivation was associated with lower IQ in early childhood and anxiety and cannabis use in adulthood; b) threat was associated with anxiety and depression in adulthood; and c) unpredictability with number of sexual partners at 16 and depression and cannabis use in adulthood (McGinnis et al., 2022). Together, these findings highlight the

need for further research that examines adversity dimensions simultaneously to disentangle their unique neurodevelopmental effects.

As briefly noted earlier, the GUiNZ study has contributed a wealth of information on childhood adversity for New Zealand's most vulnerable children. The dominant approach within this body of research has been cumulative risk employed within a life-course framework, which has yielded important insights about the cluster of risk factors that are important for guiding national policy. In a review of the study's first decade's worth of findings, Morton et al. (2022) discuss the levels of inequality facing NZ youth in terms of well-being and development outcomes. They report inequalities in the home environment, such that approximately 10% of children have regularly witnessed parental conflict (i.e., psychological, physical, and emotional) and that a substantial proportion of the cohort were living in houses that did not adhere to recommended safety guidelines (e.g., damp and cold environments, no working smoke alarms) (Morton et al., 2022). Parallel to this, approximately 13% of children experienced significant levels of deprivation within their first 1000 days, which was associated with multiple co-morbidities and poorer wellbeing outcomes at age 4 years (Russell et al., 2020) and were further exacerbated by the time children were 8 years of age (Morton et al., 2022). Furthermore, macro-level deprivation was examined in the GUiNZ study, showing that The GUiNZ cohort has also experienced substantial unpredictability, such that by 8 years of age, 3 out of 4 children were exposed to some residential mobility (Morton et al., 2022).

GUiNZ researchers have also examined childhood adversities and their association with school readiness outcomes and behavioural and emotional problems. Walsh et al. (2019) mapped standard ACE definitions (Felitti et al., 1998) onto GUiNZ data and examined their associations with a range of standardised measures of preschool skills. Half of the cohort experienced at least one ACE up to preschool age, which is likely to be underestimated due to

missing data. The study replicated Felitti et al. (1998) seminal finding on the dose-response relationship between ACEs and developmental sequelae, showing that greater ACEs lead to poorer school readiness outcomes, such as writing, counting, affective knowledge, and inhibitory control (Walsh et al., 2019). Adding to this, in a series of studies, researchers showed associations between CR patterns and developmental disadvantage in multiple domains, demonstrating that consistently high exposure was associated with abnormal levels of problem behaviours at age 4.5 years, but that both medium and high levels of adversity exposure were associated with problems in health, behaviour and education (D'Souza et al., 2019; Wallander et al., 2021). This work expands on ACEs research given its explicit developmental focus on early life exposure, specifically related to maternal sociodemographic and health-related indicators. However, these studies rely on a stress perspective and as such may benefit from a dimensional approach to elucidate other transdiagnostic pathways between adversity and later mental health difficulties.

Given the limitations present in the extant literature and the demonstrated benefits of a dimensional approach to adversity and psychopathology, the present study has several aims. In Study 1, the aim is to examine the structure of psychopathology at age 8 years using the total difficulties score (TDS) of the SDQ employed in the GUiNZ study to determine whether separate internalising, externalising, and general factors of psychopathology exist in this cohort and using this instrument. Next, I tested the reliability and external validity of the best-fitting model by examining associations with known correlates of the measured factors, which include sociodemographic, personality, and behavioural variables.

In Study 2, the aim was to examine the unique effects of early life threat, deprivation, and unpredictability on TDS at age 8 years. Core predictions of the DMAP and Harshness-Unpredictability frameworks were tested by examining the association between adversity dimensions and their linked proximal mechanisms proposed by these two models.

While the present study is largely exploratory, and given the limited research utilising all three dimensions, I hypothesised that:

- Threat, deprivation, and unpredictability would be related to higher TDS in middle childhood, controlling for socio-demographic covariates.
- The effects of unpredictability and threat on TDS would be mediated by executive control when examined individually, controlling for the effects of other dimensions and covariates. However, these indirect effects would not remain significant when controlling for other adversity dimensions simultaneously.
- The effect of deprivation on TDS would be mediated by executive control, controlling for the effects of other dimensions, sociodemographic covariates, and previous levels of TDS.

2 Methods

2.1 Background

Data utilised in this thesis was obtained from GUiNZ, a prospective longitudinal cohort study based in New Zealand that is reflective of the ethnic and sociodemographic makeup of the country (Morton et al., 2015). The overarching aim of the GUiNZ study is to investigate multiple dimensions of child development, namely: health and wellbeing; family; education; psychological development; neighbourhood and environment; and culture and identity (Morton et al., 2013). A major strength of the study includes the wealth of data collection occurring before birth and in the child's early life, which has substantially influenced policy-making in New Zealand (Bird et al., 2016). To date, there have been six major data collection waves (DCW): antenatal; 9 months; and at 2; 4.5; 8; and 12 years of age (Morton et al., 2013). Data across major phases of the study were collected via computer-assisted face-to-face and telephone interviews as well as online questionnaires (Morton et al., 2020). The study children and their families are expected to be regularly assessed until age 21 years.

2.2 Participants and General Procedure

Details of the GUiNZ design, recruitment, and sample characteristics at each major DCW have been described in comprehensive reports previously (Morton et al., 2012; Morton et al., 2014). Eligibility criteria for the study included an expected due date between April 2009 and March 2010 as well as living within the selected recruitment region, which encompassed areas covered by Auckland, Counties Manukau, and Waikato District Health Boards (Morton et al., 2013). These regions accounted for approximately one third of New Zealand's live births and 29% of its population, and were selected to maximise sociodemographic diversity to be appropriately reflective of New Zealand (Morton et al., 2013). As such, the wider GUiNZ cohort consists of 6,853 children (52% male) born to 6,822

mothers, and is nationally representative in terms of its ethnic makeup allowing for comparisons across the country's four major ethnic groups: European; Māori; Pacific; and Asian.

Data for the present thesis were collected from the 8 Year DCW, where 6571 children were eligible to participate, representing 96% of the baseline cohort. A total of 5556 children (81% of eligible cohort) had available data in the Eight Year DCW across at least one component of the study (i.e., mother or child-reported data) (Morton et al., 2020). Children were included in Analysis One if they had complete data on all items of the SDQ (N = 4625) and, in Analysis Two, if they had sufficient data across SDQ items to compute total difficulties scores (TDS) (N = 4632). I utilised TDS in Analysis Two, given the need to control for previous levels of behavioural difficulties in mediation analysis and documented issues with SDQ data collection at age 4.5 years (Walker et al., 2023). Those that had missing data for the SDQ were more likely to be Māori or Pacific, born to mothers with lower levels of education, and born to mothers living in a high deprivation area during pregnancy (all *p*'s < .001).

Ethical approval for the GUiNZ study was obtained from the Ministry of Health Northern Y Regional Ethics Committee (NTY/08/06/055). Mothers provided their written informed consent for participation on behalf of both themselves and their children.

2.3 Measures

2.3.1 Strengths and Difficulties Questionnaire

At age 8 years, mothers reported on children's emotional and behavioural problems using the SDQ. The SDQ is a widely used scale consisting of 25 items (see Table 1), which measures multiple aspects of child behaviour and is divided into 5 subscales: prosocial; peer; emotional; conduct; and hyperactivity (Goodman, 2001). Each item is scored on a 3-point Likert scale ('0 = Not true', '1 = Somewhat true', '2 = Certainly true'). The scale also offers a

total behavioural difficulties score (range 0-40) which sums all subscales with the exception of the strength-based prosocial subscale. The total behavioural difficulties score is categorised as borderline and abnormal at a cut-off score of greater than or equal to 14 and 20, respectively (Goodman, 1997).

Table 1

Strengths and Difficulties Questionnaire (SDQ) items sorted by each subscale (Prosocial score and Conduct, Hyperactivity/Inattention, Peer, and Emotional Problems).

SDQ Subscales and Items
Prosocial Scale
Considerate of other people's feelings
Shares readily with other children, for example toys, treats, pencils
Helpful if someone is hurt, upset, or feeling ill
Kind to younger children
Often volunteers to help others (parents, teachers, other children)
Conduct Problems
Often loses temper
Generally well behaved, usually does what adults request
Often fights with other children or bullies them
Often lies or cheats
Steals from home, school or elsewhere
Hyperactivity/Inattention Problems
Restless, overactive, cannot stay still for long
Constantly fidgeting or squirming
Easily distracted, concentration wanders
Thinks things out before acting
Good attention span, sees work through to the end
Peer Problems
Rather solitary, prefers to play alone
Has at least one good friend
Generally liked by other children
Picked on or bullied by other children
Gets along better with adults than with other children
Emotional Problems
Often complains of headaches, stomach-aches, or sickness
Many worries or often seems worried
Often unhappy, down-hearted or tearful
Nervous or clingy in new situations, easily loses confidence
Many fears, easily scared

The SDQ has shown good concurrent validity and reliability in community (Muris et al., 2003) and clinical samples (Becker et al., 2004), as well as satisfactory psychometric properties in prior GUiNZ research (D'Souza et al., 2017). Further, the SDQ has performed equally well compared to other widely used clinical and research tools used to screen psychopathology, such as the Child Behaviour Checklist or the Achenbach System of Empirically Based Assessment (Goodman & Scott, 1999; Janssens & Deboutte, 2009).

In a recent study, researchers performed a structural analysis of the SDQ and found that using broader internalising and externalising dimensions of the scale is more appropriate when screening in low-risk community samples (Goodman et al., 2010). As such, the present study used the hyperactivity/conduct and emotional/peer problems scales in Analysis 1 to assess externalising and internalising dimensions, respectively. In addition, the reverse-scored items comprising the strengths-based prosocial subscale were removed from the analysis which is in line with the literature (Afzali et al., 2017; Carragher et al., 2016; Goodman et al., 2010). Further supporting the decision for broader internalising and externalising dimensions, the average TDS in this sample (N = 4632) was 7.54, which is in line with mean TDS of epidemiological and community samples (Ortuno-Sierra et al., 2015; Peacock et al., 2011). In Analysis 2, I dichotomised total behavioural difficulties scores to perform logistic regression and used the measure in its continuous form for mediation analysis. Where total behavioural difficulties were dichotomized, children were defined as having either normal or abnormal scores, with the latter being defined as scores within the borderline and abnormal range (greater or equal than 14). Further, total behavioural difficulties scores at age 4.5 years were utilized in mediation analyses to control for previous levels of behavioural difficulties. The internal consistency of the SDQ (excluding the prosocial subscale) within the sample was high (Cronbach's $\alpha = 0.83$).

2.3.2 External Correlates

The following correlates were used to test the external validity of structural models of psychopathology assessed in this thesis: gender; age; deprivation at age 8 years; and child impulsivity, depression, anxiety, prosocial behaviour, and dysregulation. Deprivation was measured at age 8 years using the New Zealand Index of Deprivation (NZDI), which accounts for a variety of socioeconomic indicators from the New Zealand Census (range 1-10; greater scores reflect greater deprivation) (Atkinson et al., 2014). To measure child impulsivity, I used scores from the 8-item Domain-Specific Impulsivity Scale for Children (DSIS-C), which has been shown to have satisfactory psychometric properties (Tsukayama et al., 2013); and associations with externalising spectrum disorders (Beauchaine et al., 2017). Further, I measured child depression and anxiety using the 10-item Center for Epidemiological Studies Depression Scale for Children (CES-DC) and the 8-item Patient Reported Outcomes Measurement Information System (PROMIS) Anxiety short form scale, respectively, which have both shown good psychometric properties in previous research (Cha et al., 2022; Freitag et al., 2023). Lastly, I measured social competence using the prosocial subscale of the SDQ (range = 0 to 10) given documented relationships with psychopathology across childhood and adolescence (Memmott-Elison & Toseeb, 2023; Milledge et al., 2019).

2.3.3 Mediator Variable - Luria Handclap Task (Executive Control)

To assess executive control, children were administered the Luria Handclap task, which was adapted from the Luria Pencil Tapping task from the Luria-Nebraska Neuropsychological battery given that levels of exposure to using and holding pencils at age 4.5 years may differ across participants (Golden et al., 1979). The task involves reverse imitation, requiring children to remember and execute specific hand-clapping rules given by the researcher (e.g., clap once when the researcher claps twice, clap twice when the researcher claps once) (for further details, see Buckley et al., 2021).

The task is a developmentally sensitive measure of executive control (Diamond & Taylor, 1996; Putko et al., 2014), allowing the assessment of attention, working memory, and response inhibition/inhibitory control (Golden et al., 1979), especially in samples consisting of disadvantaged children (Rhoades et al., 2009). Researchers first demonstrated and explained the task to children and allowed a maximum of 6 teaching trials. Children received feedback during teaching trials and were allowed to move on to test trials if they correctly responded on three consecutive trials. In total, the task consists of 16 hand clap trials where children receive 1 point for each correct reverse imitation (out of a maximum of 16 points). Those with missing data were more likely to have higher levels of behavioural difficulties, be living in a high deprivation area, and be of Māori, Pacific, or Asian descent. Lastly, the Luria Handclap Task was utilised as a continuous variable in the present study (N = 4158) and is referred to hereafter as executive control (EC).

2.4 Predictor variables

Variables from ages 1 to 4.5 years were selected a priori and sorted into different dimensions of adversity, namely threat, deprivation, and unpredictability, based on concepts presented in the theoretical overview above. Each indicator of childhood adversity was dichotomized as absent (i.e., '0') or present (i.e., '1') using a predefined threshold by previous GUiNZ research. When using a set of items developed by the GUiNZ study with no suggested cut-off score, I coded variables as indicative of adversity if scores were at the 90th percentile to ensure higher levels of adversity. While categorising adversity as either absent or present introduces limitations to the interpretation of the data, it follows recommendations for modelling dimensional models of adversity due to the conceptual and methodological difficulties in modelling dimensions using standardised scales or factor analysis (Berman et al., 2022).

2.4.1 Threat

Threat was captured by a composite score consisting of the following: bullying; harsh physical punishment; harsh parenting; and exposure to intimate partner violence. These experiences were measured using the following indicators: a) bullying at ages 2 and 4.5 years; and b) harsh physical punishment, harsh parenting and experiences of intimate partner violence within the first 4.5 years. Higher scores were reflective of greater exposure to threatening experiences (out of a maximum possible score of 5). Due to low frequencies in the upper tail of the threat dimension, scores were winsorized at ≥ 3 (95th percentile).

Experiences of harsh physical punishment, harsh parenting, and intimate partner violence were coded following previous GUiNZ research (Walsh et al., 2019). Due to low frequencies and high missing data of maternal harsh physical punishment and harsh parenting, I used several indicators up until age 4.5 years to measure these forms of adversity. Harsh physical punishment was measured using items from the Parenting Practices Questionnaire (Robinson et al., 1995), which measure three global parenting styles, namely authoritative, authoritarian, and permissive parenting. Children were categorised as having experienced harsh physical punishment if their mothers reported that they smack their child 'often' at age 2 years, and if they smack, grab, or physically punish their child at least 'half the time' at age 4.5 years (Walsh et al., 2019). Children were coded as having experienced harsh parenting if mothers reported, within the child's first 4.5 years of life, that they, 'very often', criticise their child's ideas, and shout or explode with anger when their child misbehaves (Walsh et al., 2019). Children were also assessed for their exposure to intimate partner violence via the Women's Abuse Screening Tool (WASI) at age 9 months and by the WASI and World Health Organisation Violence questionnaire at age 4.5 years (Brown et al., 2006; Garcia-Moreno et al., 2006). Children were coded as having experienced intimate partner violence if mothers reported that arguments 'quite often' resulted in pushing, shoving,

throwing, breaking things, or hitting, kicking, pushing, or slapping (Pryor, 2004; Walsh et al., 2019).

Mothers also reported on whether their child experienced bullying. I used the same item from the SDQ at ages 2 and 4.5 years, which asked mothers if their children were picked on or bullied by other children based on their behaviour in the past 6 months (Goodman, 1997). Children received a score of 1 for each age their mothers reported that it is 'somewhat true' that their child is picked on or bullied by other children.

2.4.2 Deprivation

Deprivation was indexed using 8 indicators. These indicators reflected both distal and proximal risk factors for deprivation, and were categorised into the following subgroups: a) Neighbourhood deprivation; b) Household deprivation; c) Parental cognitive stimulation; and d) Stimulating home environment. Greater scores in this dimension reflect greater levels of material and cognitive deprivation (out of a maximum possible score of 8). Due to low frequencies in the upper tail of the deprivation dimension, scores were winsorized at ≥ 5 (95th percentile).

I assessed neighbourhood-level deprivation using the 2006 and 2013 New Zealand Index of Deprivation (NZDI) (Atkinson et al., 2014; Salmond & Crampton, 2002). The NZDI measures area-level deprivation using socioeconomic indicators from the New Zealand Census, such as: people aged 18-64 that are unemployed, home ownership rates, people aged 18-64 without any qualifications, and people with no access to a car (Atkinson et al., 2014). Using a total of 9 dimensions of deprivation from census data, each census area receives a score from 1-10, with greater scores reflecting greater neighbourhood-level deprivation. In this study, children received a score of 1 for each age if they were in the high deprivation category (deciles 8-10) at ages 1 and 4.5 years (maximum score of 2).

Household material deprivation was assessed using the New Zealand Index of Deprivation for Individuals (NZiDep) (Salmond et al., 2006; Dominick, 2018). Mothers were asked several questions to assess their exposure to poverty, which included endorsements of items such as: forced to buy cheaper food; couldn't afford replacements for shoes with holes; put up with feeling cold to save heating costs; received help from a community organisation in the form of food, clothes or money; and obtained a prescription for their child but did not collect it because they could not afford it (Salmond et al., 2006). These items were summed and children with scores in the 90th percentile received a score of 1 based on mothers' endorsement at age 1 and 4.5 years (maximum score of 2).

Parental cognitive stimulation was assessed using a range of items at ages 1 and 4.5 years (see Table 2). Items were assessed on a 5 point Likert scale ('1 = seldom or never', '2 = once a week', '3 = several times a week', '4 = once a day', '5 = several times a day'). Response scales were collapsed such that each measure of parental cognitive stimulation was dichotomised as present (at least several times a week; '0') or absent (seldom or never, once a week; '1'). These items were then summed at each age and children with scores at the 90th percentile were considered to have low levels of parental cognitive stimulation at ages 1 and/or 4.5 years (maximum score of 2).

Table 2

GUiNZ items used to create parental cognitive stimulation variables.

Variable	Items
Cognitive Stimulation - Age 1	Plays games with baby
	Talks to baby during everyday activities
	Plays with toys with baby
	Sings songs or tells stories to baby
	Reads books to baby
Cognitive Stimulation - Age 4.5	How often do you read books with your child?
	How often do you encourage your child to print letters, words, or numbers?
	How often do you encourage your child to read words?
	How often do you encourage your child to count?
	How often do you encourage your child to recognise numbers?

Next, I assessed whether a child's home environment was stimulating using two indicators: a) the number of hours a child watches television; and b) safety of the child's play area. Children received a score of 1 if mothers reported that the average number of hours a child watches television on a weekday across ages 2 years and 45 months, was 3 or more hours, which follows recommended guidelines for screen time by the American Academy of Paediatrics (Hill et al., 2016). Lastly, children received a score of 1 if mothers reported at age 2 years that the outside area where the child plays was not fully fenced and that the driveway was not fully fenced off or separate from where the child plays.

2.4.3 Unpredictability

To index unpredictability, I created a composite score consistent with the ancestral cues (Young et al., 2020) and household chaos (Andrews et al., 2020) perspectives. This composite score consists of four subgroups: parental transitions, unpredictable caregiving behaviour, and financial uncertainty. As in previous dimensions, greater scores are indicative of greater levels of unpredictability (out of a maximum score of 8). Due to low frequencies in the upper tail of the unpredictability dimension, scores were winsorized at ≥ 3 (95th percentile).

Parental transitions included mother-reported changes in residential living arrangements and relationship status. To assess changes in residential living arrangements, I summed the number of moves experienced by children and their families at ages 1, 2, 3, and 4.5 years (dichotomized as present if mothers reported moving 4+ times; 90th percentile). In addition, changes in relationship status were dichotomized as present or absent if mothers reported a change in partner status (current partner, no current partner) during the first 4.5 years of their child's life.

I also indexed unpredictability with measures of maternal mental health. When children were 9 months old, mothers completed the Edinburgh Postnatal Depression Scale (EPDS) (Cox et al., 1987). The EPDS consists of 10 self-report items (scores ranging 0-30) to screen for postnatal depression, where a score of greater than 12 is used to indicate clinically significant depressive symptoms (Waldie et al., 2015). Children received a score of 1 if their mothers reported clinically significant depressive symptoms. In addition, maternal depression was assessed at age 4.5 years using the Patient Health Questionnaire, which consists of 9 self-reported items and scores ranging from 0 to 27 (PHQ-9) (Kroenke et al., 2001). Consistent with previous research (D'Souza et al., 2019; Kroenke & Spitzer, 2002), a cut-off of greater than or equal to 10 was used to indicate clinically significant depressive symptoms. Mothers

also reported on their drug use since the birth of the baby. Children received a score of 1 if mothers endorsed the use of hard drugs or marijuana since the birth of their child. At age 45 months, mothers were asked to do a stocktake of the number of child care arrangements a child had since birth. Missing data was supplemented by mother reports of caregiving arrangements from ages 9 months, 2 years, and 31 months. Reported frequencies of caregiving arrangements were summed and dichotomized as unpredictable at 3 or more caregiver arrangements since the child's birth (90th percentile). Lastly, I used two indicators of financial stress at ages 9 months and 2 years: (i) stress about housing difficulties or (ii) money problems. If mothers reported that money problems or housing difficulties were "highly stressful", children received a score of 1 for each age (maximum score of 2).

2.4.4 Covariates

Given the numerous confounders that can affect the relationship between adversity, executive control, and mental health, I controlled for several socio-demographic variables. All regression models controlled for the effects of gender and maternal self-prioritised ethnicity. Many studies report gender differences in mental health outcomes across childhood and adolescence (Campbell et al., 2021; Eisenbarth, 2017), and this is also seen in studies that used the SDQ (Kawabe et al., 2021; Kunze et al., 2018; Yoon et al., 2023). I also controlled for ethnicity given its potential confounding effects on adversity and utilized mother-reported self-prioritised ethnicity during the antenatal DCW. In this study, maternal ethnicity was treated as a dichotomous variable (0 = New Zealand European; 1 = Other) and missing data was supplemented with mothers externally prioritised ethnicity following Statistics New Zealand guidelines (Statistics New Zealand, 2004). In addition to gender and maternal ethnicity, mediation analyses controlled for both total behavioural difficulties and child age at 4.5 years. I controlled for previous levels of total behavioural difficulties to test whether the predictors of interest showed a relationship with total behavioural difficulties in middle

childhood above and beyond total behavioural difficulties in early childhood. Further, children ranged in age from 4 to 5.7 years at the time they were assessed for executive control using the Luria Handclap Task. Due to the confounding effects this may have on task performance (Buckley et al., 2019), child age was controlled for in analyses that included the Luria Handclap Task.

2.5 Analytical Plan

2.5.1 Analysis 1A: Defining p; model fit; and reliability

Several structural models of psychopathology were tested at 8 years of age using item-level SDQ data and confirmatory factor analysis (CFA). Based on model specification, specific factors were either categorised into broader internalising and externalising dimensions or as the subscales of the SDQ. In addition, items worded as strengths within each problem subscale were recoded prior to their inclusion in the model. Following analytical and theoretical methods reported in previous studies (Caspi et al., 2014; Chen et al., 2022; Otuna-Sierra et al., 2015), I tested six different models: a) a one-factor model, where all SDQ items load onto one general factor (Model A); b) correlated factors model, with peer and emotion subscales loading onto an internalising factor and conduct and hyperactivity/inattention subscales loading onto an externalising factor (Model B); c) symmetrical bifactor model with one general factor and two uncorrelated specific factors (i.e., internalising and externalising), where all SDQ items load onto a general factor and their respective orthogonal specific factor (Model C); d) symmetrical bifactor model with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales) (Model D); e) symmetrical bifactor model with one general factor and four correlated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales) (Model E); and f) S-1 bifactor model including one general factor with conduct problems subscale set as the reference domain, and three correlated

specific factors (hyperactivity/inattention, peer, and emotion problems subscales) (Model F). I use the conduct problems scale as a reference domain in Model F (S-1 bifactor model) to allow comparability with previous studies (Chen et al., 2022). The decision to set conduct problems as a reference domain is also supported by previous findings of the developmental heterogeneity in conduct disorder and its comorbidity with other important psychopathological dimensions, which suggests some important transdiagnostic features (Dugré & Potvin, 2022). All CFA's were conducted in Mplus 8.10. Given that the SDQ uses ordered categorical variables, weighted least square mean and variance adjusted (WLSMV) estimation with delta parameterization was used to address the ordinal nature of variables and nonmultivariate normal data (Flora & Curran, 2004; Muthén et al., 1997). Previous simulation studies have shown that WLSMV outperforms robust maximum likelihood estimation in producing unbiased estimates when sample sizes are large, and data is nonmultivariate normal and ordinal in nature (Finney & DiStefano, 2013; Kline, 2005; Li, 2016).

In accordance with previous studies (Caspi et al., 2014; Hoffman et al., 2022), I report multiple indices to evaluate model fit: comparative fit index (CFI); Tucker-Lewis index (TLI); root mean square error of approximation (RMSEA); and standardised root mean-square residual (SRMR). Although I report chi-square statistics and corresponding p-values, these are not used to assess model fit due to their sensitivity to large sample sizes (Kline, 2005). While there are multiple guidelines for assessing model fit, this study follows Hu & Bentler's (1999) recommendations: a) CFI or TLI values of ≥ 0.90 and RMSEA and SRMR values ≤ 0.80 indicate acceptable model fit; and b) CFI and TLI values of ≥ 0.95 and RMSEA and SRMR values of ≤ 0.60 indicate good-to-excellent fit. Lastly, I report standardised estimates of factor loadings as a measure of effect size (salient loadings > 0.3) (Miočević et al., 2018).

Given that previous applications of bifactor models of psychopathology have often yielded anomalous results (Watts et al., 2019), several researchers have recommended the use of a range of statistical indices used to evaluate bifactor models and their use in structural equation modelling (SEM) and the derivation of factor score estimates (Constantinou & Fonaghy, 2019; Eid et al., 2017; Rodriguez et al., 2016). In the present study, I calculated the following indices using formulas provided by Rodriguez et al. (2016): 1) explained common variance (ECV); 2) Lucke's omega (ω); 3) Hierarchical omega (ωH) and subscale hierarchical omega (ωHs); 4) Relative omega (Relative ω); 5) H index (H); 6) Factor Determinacy (FD); 7) Percentage uncontaminated correlations (PUC); and 8) Absolute relative parameter bias (ARPB).

ECV is the proportion of total common variance explained by a given factor and is a measure of unidimensionality (whether a scale has a strong general factor; Reise & Haviland, 2005). For specific factors, I calculate two measures of ECV which measure the proportion of common variance explained by a specific factor relative to the total variance of all items (ECV-SG) and the proportion of common variance explained by a specific factor relative to the total variance of items loading on that specific factor (ECV-SS) (Rodriguez et al., 2016). For the general factor, these two measures are simply ECV. ECV values range from 0 to 1, and are typically expressed as a percentage, with higher values being indicative of greater unidimensionality (Stucky & Edelen, 2014).

Omega (ω) provides a model-based estimate of internal reliability that is analogous to Cronbach's alpha but suitable for a multidimensional composite. This statistical index is analogous to Cronbach's alpha, scores greater than or equal to 0.70 or 0.80 considered acceptable or good, respectively (Cronbach, 1951; Rodriguez et al., 2016). Hierarchical omega (ωH) and subscale hierarchical omega (ωHs) respectively measure the percentage of systematic variance in raw total scores due to general factors and subscale factors after

partitioning the variability due to the general factor (Reise et al., 2013; Rodriguez et al., 2016). High values for ωH (i.e., > 0.8) indicate a strong general factor and unidimensionality of total scores, but values of greater than 0.5 and 0.75 are considered acceptable or preferred, respectively (Reise et al., 2010).

I also report the *H* index, a measure of construct replicability where values of > .80 suggest a well-defined latent variable (Hancock & Mueller, 2001; Rodriguez et al., 2016). It is measured by dividing the proportion of variance explained by a construct and dividing it by variance that is unexplained, where values greater than 0.70 to 0.80 indicate likely replicability across studies (Hancock & Mueller, 2001).

Factor determinacy (FD) measures the correlation between a latent factor and factor scores (Rodriguez et al., 2016), and is typically used to determine whether extraction of factor scores from structural equation models are trustworthy when used in subsequent analyses (Caspi et al., 2023). Possible values for FD range from 0 to 1, with scores greater than 0.90 being recommended for factor score estimates to be used in subsequent analyses (Gorsuch, 2014). PUC represents the percentage of covariances among items that only reflect variance from the general factor (Rodriguez et al., 2016). Lastly, ARPB represents the difference between an indicator's loading in a one-factor/unidimensional model versus its loading in a bi-factor model (Rodriguez et al., 2016). Previous work suggests that bias greater than 15% may pose some concerns (Muthén et al., 1987; Rodriguez et al., 2016).

2.5.2 Analysis 1B: External correlates of p

I investigated the external validity of the general factor of psychopathology from the best fitting model in the preceding analysis by examining associations with variables that have established relationships with psychopathology in childhood. Bivariate correlations were examined between factor score estimates extracted from the best fitting model and the following variables: Gender; Age; Deprivation Index; DSIS-C; CES-DC; PROMIS Anxiety

short form scale; and the prosocial behaviour subscale of the SDQ dysregulated at age 8 years. External validity analyses were conducted in SPSS Version 29.0.

2.5.3 Analysis 2: Direct and indirect effects of adversity on total behavioural difficulties at age 8 years

Given the need to control for previous behavioural difficulties in analysis two and previously reported issues with GUiNZ data collection of the SDQ at age 4.5 years (Walker et al., 2023), I utilised total behavioural difficulties at age 8 years as the outcome of interest (N = 4,632). This was to obviate the need to impute for completely missing items from the SDQ (Item 12) (Walker et al., 2023), which would be required to conduct SEM and maintain consistency across models at 4.5 and 8 years of age.

Composite scores were constructed for each adversity dimension and sample descriptives are provided in Table 10. Further, I conducted bivariate correlation analyses (Table S1) and collinearity diagnostics for all adversity indicators, which warranted no further investigation (tolerance values above 0.1 and variance inflation factors below 2) (Glantz et al., 1990). Given potential confounding effects between covariates and predictors, I ran a series of hierarchical binary logistic regressions to test for interaction effects between each adversity dimension individually with both gender and maternal ethnicity on total behavioural difficulties. Adversity dimensions were centred in interaction terms to avoid any issues with multicollinearity and if found significant at a 5% level, were included in subsequent analyses. Next, I conducted hierarchical binary logistic regression to test the impact of adversity dimensions on parent-reported SDQ total behavioural difficulties at age 8 years, while controlling for the effects of socio-demographic covariates. Odds ratios and 95% confidence intervals (CI) are presented for logistic hierarchical regression analysis and statistical significance was set at a 5% level. The final hierarchical binary logistic regression

consisted of two blocks: maternal ethnicity and gender (Block 1); Threat, Unpredictability, and Deprivation (Block 2).

Next, I examined the mediating effect each adversity dimension on total behavioural difficulties at age 8 years through executive control. In total, I conducted four mediation models: a) the first model included EC as a mediator between deprivation and total behavioural difficulties (Model 1; Figure 3); b) the second model included EC as a mediator between threat and total behavioural difficulties (Model 2; Figure 3); c) the third model included EC as a mediator between unpredictability and behavioural difficulties (Model 3; Figure 3); and d) the fourth model included EC as a mediator between deprivation and total behavioural difficulties, while simultaneously including the dimensions of threat and unpredictability as covariates (Model 4; Figure 3). All mediation models included the following as socio-demographic covariates: gender; TDS at age 54 months (to control for previous levels of behavioural difficulties); maternal ethnicity; and child age at 54 months. I provide 95% bootstrap CI based on 10,000 bootstrapped samples for indirect effects across all models and utilise the Cribari-Neto heteroscedasticity sandwich estimator, which is robust to violations of non-normality and heteroscedasticity (Hayes, 2012). Multicollinearity was examined across all regression models using collinearity diagnosis indices and was not detected across any model (tolerance range = .277, .993; VIF range = 1.007, 2.337) (Glantz et al., 1990; O'Brien, 2007). List-wise deletion was used to deal with missing data across all regression models.

All regression analyses were conducted in SPSS Version 29.0 and all mediation analyses were conducted using SPSS Process Macro Version 4.2. We used the Benjamini-Hochberg procedure to account for multiple testing, given its superiority in minimizing false-negative rates compared to Bonferroni (Benjamini & Hochberg, 1995; White, 2019). All *p*'s < 0.5 remained significant after this correction was applied.

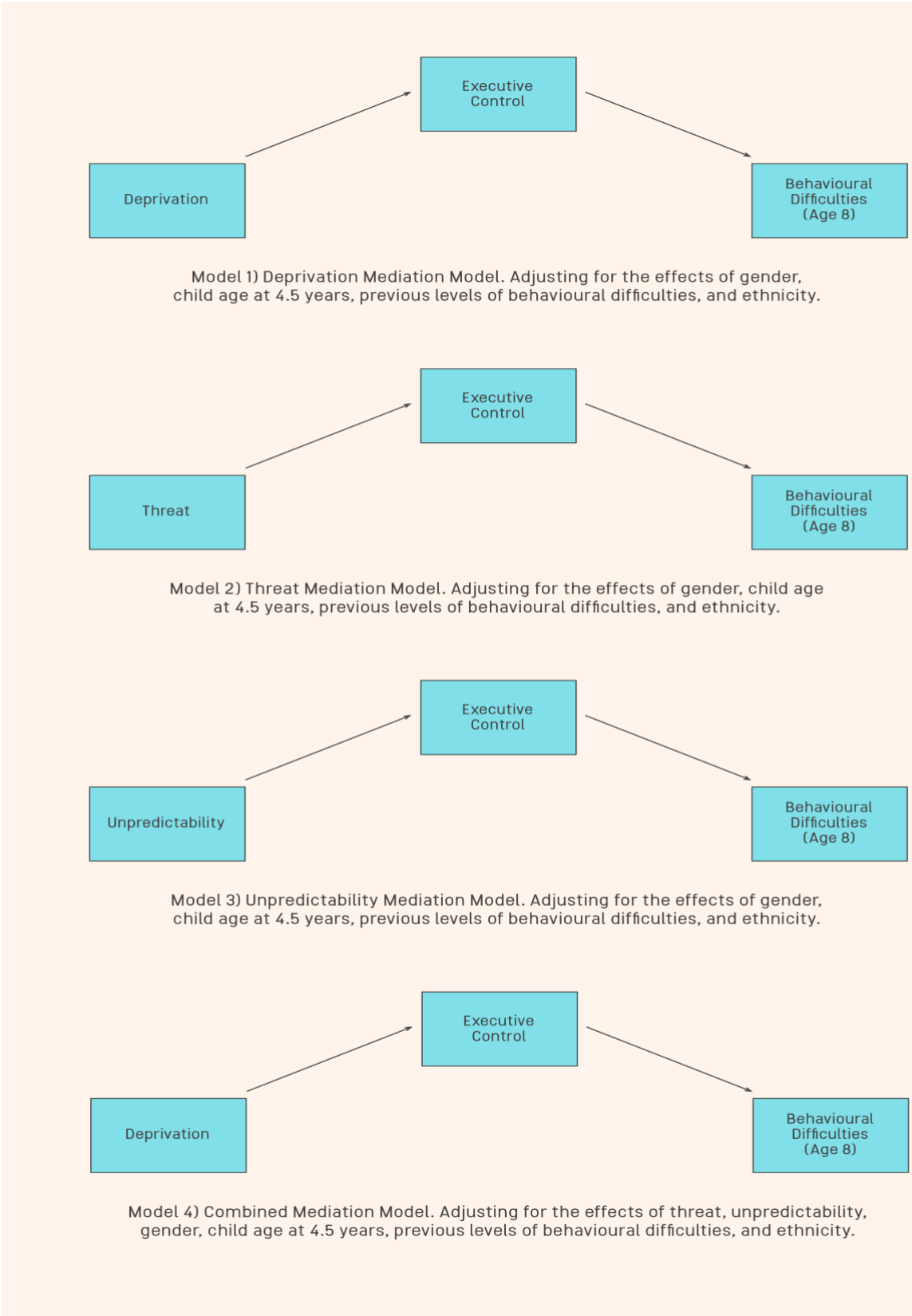

Figure 3. Mediation models testing the indirect effect of each adversity dimension on total difficulties scores at age 8 years through executive control, adjusting for covariates.

3 Results

3.1 Analysis 1

3.1.1 Model fit and factor loadings

A total of 6 structural models of psychopathology were tested using CFA, 4 of which were variations of the bi-factor model. Model fit statistics are presented in Table 3 and standardized factor loadings for all model are presented in Tables 4-7. Based on model fit statistics, all bi-factor models (Models C-F) fit the data well, with Model E fitting best.

Table 3

Model fit statistics for structural models of psychopathology using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years (Models A-F).

	$\chi 2$	df	CFI	TLI	RMSEA (90% CI)	SRMR
Model A	8342.381***	170	.767	.740	.102 (.100, .104)	.105
Model B	4635.631***	169	.873	.857	.076 (.074, .077)	.085
Model C	2808.405***	150	.924	.904	.062 (.060, .064)	.058
Model D	2667.730***	150	.928	.909	.060 (.058, .062)	.058
Model E	1683.987***	144	.956	.942	.048 (.046, .050)	.046
Model F	2265.910***	152	.940	.925	.055 (.053, .057)	.053

Note. CFI = comparative fit index, TLI = Tucker-Lewis index, RMSEA = Root Mean Square Error of Approximation, CI = confidence interval, SRMR = standardised root mean squared residual. * $p < .05$. ** $p < .01$. *** $p < .001$. Model A: one-factor model, where all SDQ

items load onto one general factor. Model B: correlated factors model, with peer and emotion subscales loading onto an internalising factor and conduct and hyperactivity/inattention subscales loading onto an externalising factor. Model C: symmetrical bifactor model with one general factor and two uncorrelated specific factors (i.e., internalising and externalising), where all SDQ items load onto a general factor and their respective orthogonal specific factor. Model D: symmetrical bifactor model with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). Model E: symmetrical bifactor model with one general factor and four correlated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). Model F: S-1 bifactor model including one general factor with conduct problems subscale set as the reference domain, and three correlated specific factors (hyperactivity/inattention, peer, and emotion problems subscales.

Factor loadings and *p* values for Model A and B are presented in Table 4. Model A, a one-factor model, showed poor fit on all fit indices (CFI, TLI, RMSEA, and SRMR), and factor loadings were moderately high (range = .345, .774; average λ = 0.57; all *p*'s < .001) and significant (all *p*'s < .001). Model B, a correlated factors model, showed acceptable fit for RMSEA, but had poor fit with respect to all other model fit indices. Additionally, factor loadings in this model were moderately high for both externalising (range = .567, .807; average λ = 0.679) and internalising (range = .442, .753; average λ = 0.599) factors and significant (all *p*'s < .001). Factor intercorrelation between internalising and externalising factors in Model B was significant and moderate (*r* = .57, *p* < .001).

Table 4

Standardised factor loadings for a one-factor model (Model A), correlated-factors model (Model B), and a symmetrical bi-factor model (Model C) using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years.

SDQ Items	Model A	Model B		Model C		
	P-factor	EXT	INT	P factor	EXT	INT
Conduct Scale						
Often loses temper (26)	**.583*** **	**.614*** **		**.542*** **	.271***	
Often lies or cheats (28)	**.534*** **	**.567*** **		**.512*** **	.230***	
Steals from home, school, or elsewhere (30)	**.544*** **	**.571*** **		**.569*** **	.142***	
Often fights with children or bullies them (12)	**.661*** **	**.688*** **		**.687*** **	.185***	
Generally well behaved (27)[+]	**.635*** **	**.674*** **		**.547*** **	**.375*** **	
Hyperactivity Scale						
Restless, overactive, cannot stay still for too long (2)	**.755*** **	**.791*** **		**.397*** **	**.733*** **	
Constantly fidgeting or squirming (10)	**.774*** **	**.807*** **		**.431*** **	**.708*** **	
Easily distracted, concentration wanders (15)	**.735*** **	**.775*** **		**.417*** **	**.687*** **	
Thinks things out before acting (29) [+]	**.531*** **	**.575*** **		**.356*** **	**.467*** **	
Good attention span (31) [+]	**.685*** **	**.727*** **		**.382*** **	**.666*** **	
Peer Scale						
Rather solitary, prefers to play alone (6)	**.345*** **		**.442*** **	**.385*** **		.200***
Has at least one good friend (11) [+]	**.515*** **		**.596*** **	**.637*** **		-.173***

Generally well liked by other children (14) [+]	**.645*** **	**.753*** **	**.821*** **	-.201*** **
Picked on or bullied by other children (19)	**.531*** **	**.625*** **	**.614*** **	0.100*** **
Gets along better with adults than with other children (23)	**.390*** **	**.474*** **	**.456*** **	.084*** **
Emotional Problems Scale				
Often complains of headaches, stomach-aches, or sickness (3)	**.372*** **	**.454*** **	**.368*** **	**.331*** **
Many worries or often seems worried (8)	**.548*** **	**.670*** **	**.483*** **	**.605*** **
Often unhappy, down-hearted, or tearful (13)	**.619*** **	**.738*** **	**.629*** **	**.418*** **
Nervous or clingy in new situations (16)	**.465*** **	**.571*** **	**.427*** **	**.483*** **
Many fears, easily scared (24)	**.536*** **	**.663*** **	**.452*** **	**.665*** **

Note. * $p < .05$. ** $p < .01$. *** $p < .001$. Salient loadings are bolded. [+] These SDQ items are positively worded as strengths but were reverse coded prior to entering them in the model. Model A: one-factor model, where all SDQ items load onto one general factor. Model B: correlated factors model, with peer and emotion subscales loading onto an internalising factor and conduct and hyperactivity/inattention subscales loading onto an externalising factor. Model C: symmetrical bifactor model with one general factor and two uncorrelated specific factors (i.e., internalising and externalising), where all SDQ items load onto a general factor and their respective orthogonal specific factor.

Model C, a symmetrical bi-factor model with one general factor and internalising and externalising specific factors which were constrained not to correlate, showed acceptable to good fit (see Table 4). Average factor loadings for the general, externalising and internalising factors were 0.506, 0.446, and 0.251, respectively. All items loading on the general factor were positive, significant (*p*'s < .001) and salient (range = .368, .821). For the externalising factor, all factor loadings were positive and significant (*p*'s < .001), however several factor loadings were close to zero (i.e.., ≤ .2). All items indicating hyperactivity/inattention problems had salient factor loadings onto the externalising factor (range = 0.467, 0.733), whereas items from the conduct problems scale weakly loaded onto the externalising specific factor (range = .142, .375). The internalising factor showed some anomalous results; whereas all items from the emotional problems subscale significantly loaded onto the internalising specific factor (range = .331, .665; all *p*'s < .001), items from the peer problems subscale included factor loadings that were negative or close to zero (range = -0.201, 0.2) (see Table 4).

The second bi-factor model (Model D) was specified with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotional problems), and showed acceptable to good fit (CFI and TLI > .90 and SRMR < .60). Average factors loadings were: 0.517 for the general factor; 0.316 for the conduct problems factor; 0.544 for the hyperactivity/inattention factor; 0.422 for the peer problems factor; and 0.536 for the emotional problems factor. All items loading on the general, hyperactivity/inattention, peer, and emotional factors were positive and significant (*p*'s < .001). The conduct problems specific factor partially collapsed, evidenced by some item factor loadings that were not significant or close to zero (range = 0.06, .955; see Table 5).

Table 5

Standardised factor loadings for a bi-factor model with one general factor and four uncorrelated specific factors (Model D) and a bi-factor model with one general factor and four correlated specific factors (Model E) using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years.

SDQ Items	Model D		Model E	
	P-factor Loading	Specific factor Loading	P-factor Loading	Specific factor Loading
Conduct Scale				
Often loses temper (26)	**.651*** **	.104*	.245***	**.657*** **
Often lies or cheats (28)	**.541*** **	**.955** **	.285***	**.564*** **
Steals from home, school, or elsewhere (30)	**.534*** **	**.356***	.212***	**.605*** **
Often fights with children or bullies them (12)	**.733*** **	.107*	**.320*** **	**.702*** **
Generally well behaved (27)$^{+}$	**.718*** **	.060	**.569*** **	**.544*** **
Hyperactivity Scale				
Restless, overactive, cannot stay still for too long (2)	**.544*** **	**.633*** **	.164***	**.821*** **
Constantly fidgeting or squirming (10)	**.562*** **	**.628*** **	.046	**.915*** **
Easily distracted, concentration wanders (15)	**.546*** **	**.611*** **	**.376*** **	**.718*** **
Thinks things out before acting (29) $^{+}$	**.494*** **	.287***	**.577*** **	**.382*** **
Good attention span (31) $^{+}$	**.521*** **	**.565*** **	**.523*** **	**.592*** **
Peer Scale				

Rather solitary, prefers to play alone (6)	**.318*** **	**.391*** **	-.107**	**.574*** **
Has at least one good friend (11) [+]	**.467*** **	**.542*** **	**.347*** **	**.543*** **
Generally well liked by other children (14) [+]	**.641*** **	**.495*** **	**.475*** **	**.678*** **
Picked on or bullied by other children (19)	**.552*** **	.263***	.173***	**.669*** **
Gets along better with adults than with other children (23)	**.363*** **	**.417*** **	-.014	**.577*** **
Emotional Problems Scale				
Often complains of headaches, stomach-aches, or sickness (3)	**.332*** **	**.365*** **	-.013	**.514*** **
Many worries or often seems worried (8)	**.439*** **	**.639*** **	-.078*	**.767*** **
Often unhappy, down-hearted, or tearful (13)	**.578*** **	**.470*** **	0.140***	**.767*** **
Nervous or clingy in new situations (16)	**.393*** **	**.496*** **	-.018	**.638*** **
Many fears, easily scared (24)	**.403*** **	**.711*** **	-.108**	**.767*** **

Note. * $p < .05$. ** $p < .01$. *** $p < .001$. Salient loadings are bolded. Model D: symmetrical bifactor model with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). Model E: symmetrical bifactor model with one general factor and four correlated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). [+] These SDQ items are positively worded as strengths but were reverse coded prior to entering them in the model

Model E was specified as a bi-factor model with one general factor and four correlated specific factors (conduct, hyperactivity/inattention, peer, and emotional problems) (Table 5). While this model showed the best fit to the data (CFI = .956, TLI = .942, RMSEA = .048, SRMR = .046), the general factor in this model collapsed, evidenced by factor loadings that were close to zero, negative, or not significant (range = -1.08, .577; average λ = .206). Factor loadings were all significant, positive, and moderately high for the conduct (range = .544, .702; average λ = .614), hyperactivity/inattention (range = .382, .915; average λ = .686), peer (range = .543, 669; average λ = .608), and emotional (range = .514, .767; average λ = .69) problems specific factors. As shown in Table 6, factor intercorrelations were all significant (p's < .001) and moderate in size (range = .422, .657), thus ruling out multicollinearity.

Table 6

Factor intercorrelations between Conduct, Hyperactivity/Inattention, Peer, and Emotional problems specific factors in Model D, using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years.

	Conduct	Hyper	Peer
Hyper	.610***		
Peer	.551***	.422***	
Emot	.624***	.424***	.657***

Note. * p < .05. ** p < .01. ***p < .001. Model D: symmetrical bifactor model with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales).

Model F was specified as an S-1 bi-factor model with conduct problems set as a reference domain (See Table 7) (Chen et al., 2022; Hoffman et al., 2021), and as such had one general factor and three correlated specific factors (hyperactivity/inattention, peer, and emotional problems). This model showed acceptable to good fit on all model fit indices (CFI and TLI > .90, RMSEA AND SRMR < .06). Average factor loadings were: 0.484 for the general factor (range = .167, .772); 0.55 for the hyperactivity/inattention problems factor (range = .247, .664); 0.51 for the peer problems factor (range = .462, .612); and 0.6 for the emotional problems factor (range = .411, .751). All factor loadings were positive and significant (p's < .001), and the majority were salient. Factor intercorrelations were small to moderate in size (range = .149, .489) and significant (all p's < .001), ruling out substantial multicollinearity.

Table 7

Standardised factor loadings for a S-1 bi-factor model using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years, with conduct problems set as the reference domain (Model F).

SDQ items	Model F	
Psychopathology Symptoms	P-factor Loading	Specific factor Loading
Conduct Scale		
Often loses temper (26)	**.693*** **	N.A
Often lies or cheats (28)	**.637*** **	N.A
Steals from home, school, or elsewhere (30)	**.634*** **	N.A
Often fights with children or bullies them (12)	**.772*** **	N.A
Generally well behaved (27)$^{+}$	**.762*** **	N.A
Hyperactivity Scale		
Restless, overactive, cannot stay still for too long (2)	**.525*** **	**.649*** **
Constantly fidgeting or squirming (10)	**.530*** **	**.664*** **
Easily distracted, concentration wanders (15)	**.526*** **	**.632*** **
Thinks things out before acting (29) $^{+}$	**.528*** **	.247***
Good attention span (31) $^{+}$	**.520*** **	**.558*** **
Peer Scale		
Rather solitary, prefers to play alone (6)	.167***	**.612*** **
Has at least one good friend (11) $^{+}$	**.421*** **	**.470*** **
Generally well liked by other children (14) $^{+}$	**.587*** **	**.478*** **
Picked on or bullied by other children (19)	**.451*** **	**.462*** **
Gets along better with adults than with other children (23)	.251***	**.528*** **
Emotional Problems Scale		
Often complains of headaches, stomach-aches, or sickness (3)	.271***	**.411*** **
Many worries or often seems worried (8)	**.332*** **	**.693*** **
Often unhappy, down-hearted, or tearful (13)	**.489*** **	**.573*** **
Nervous or clingy in new situations (16)	.298***	**.573*** **
Many fears, easily scared (24)	.288***	**.751*** **

Note. * $p < .05$. ** $p < .01$. ***$p < .001$. Salient loadings are bolded. Model F: S-1 bifactor model including one general factor with conduct problems subscale set as the reference domain, and three correlated specific factors (hyperactivity/inattention, peer, and emotion problems subscales). [+] These SDQ items are positively worded as strengths but were reverse coded prior to entering them in the model.

3.1.2 Model reliability

Table 8 compares factor-level statistical indices for all bi-factor models (Models C-F). These statistical indices are used to inform "(a) the quality of unit-weighted total and subscale score composites, as well as factor score estimates, and (b) the specification and quality of a measurement model in structural equation modelling" (Rodriguez et al., 2016, pp. 137). I report the following statistical indices: ECV-SG; ECV-SS; Omega (ω, ωs); Omega H (ωH)/Omega HS (ωHS); Relative ω; H index; Factor Determinacy (FD); Percentage Uncontaminated Correlations (PUC); and Average Relative Parameter Bias (ARPB).

Table 8

Model-based reliability statistics for bi-factor structural models of psychopathology using the total difficulties score of the parent-reported Strengths and Difficulties Questionnaire at age 8 years (Models C-F).

	ECV (SG)	ECV (SS)	ω	ωH/ ωHs	Relative Omega	H	FD	PUC	ARPB
Model C									
P factor	.578	.578	.923	.735	.796	.899	.94	0.526	.267
INT	.155	.329	.859	.159	.185	.682	.862		
EXT	.267	.505	.895	.412	.46	.818	.908		
Model D									
P factor	.524	.524	.932	.776	.832	.897	.919	.789	.156
CON	.100	.341	.870	.173	.199	.913	.975		
HYP	.147	.524	.879	.449	.511	.712	.859		
PEER	.087	.445	.774	.347	.448	.547	.757		
EMOT	.142	.613	.823	.502	.610	.712	.856		
Model E								.789	-3.571
P factor	.172	.172	.862	.246	.285	.703	.837		
CON	.181	.757	.830	.647	.780	.762	.862		
HYP	.239	.765	.896	.721	.805	.900	.945		
PEER	.177	.828	.785	.725	.924	.754	.875		

EMOT	.231	.985	.825	.825	1.00	.842	.919		
Model F								.842	.361
P factor	.522	.522	.922	.740	.802	.898	.935		
HYP	.162	.542	.879	.460	.522	.73	.878		
PEER	.131	.617	.778	.504	.649	.647	.816		
EMOT	.186	.759	.823	.627	.762	.773	.888		

Note. ECV (SG) = explained common variance of a specific factor relative to the total variance of all items; ECV (SS) = explained common variance of a specific factor relative to the total variance of items loading on that specific factor; ω = Lucke's omega reliability index; ωH = omega-hierarchical; ωHS = subscale omega-hierarchical; H = index of construct replicability; FD = factor determinacy; PUC = percentage uncontaminated correlations; ARPB = average relative parameter bias. INT = internalising; EXT = externalising; CON = conduct problems subscale; HYP = Hyperactivity/inattention problems subscale; PEER = peer problems subscale; EMOT = emotion problems subscale. Model C: symmetrical bifactor model with one general factor and two uncorrelated specific factors (i.e., internalising and externalising), where all SDQ items load onto a general factor and their respective orthogonal specific factor. Model D: symmetrical bifactor model with one general factor and four uncorrelated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). Model E: symmetrical bifactor model with one general factor and four correlated specific factors (conduct, hyperactivity/inattention, peer, and emotion problems subscales). Model F: S-1 bifactor model including one general factor with conduct problems subscale set as the reference domain, and three correlated specific factors (hyperactivity/inattention, peer, and emotion problems subscales)

In the present study, ECV values for general factors across all models explained 17.2% to 57.8% of the common variance extracted. With the exception of Model D, which had a collapsed general factor, all other bi-factor models had general factors that explained over half of the common variance extracted (Table 8). ECV-SG was low across all models, as specific factors showed low ECV with respect to the general factor (average ECV-SG = 17%, range 8.7% to 26.7%). The strength of specific factors with respect to themselves was generally high across all models, with average ECV-SS of 41.7% for Model C, 48.1% for Model D, 83.3% for Model E, and 63.9% for Model F. Across all models, ECV for general factors were lower than required thresholds (values ≥ 85%), indicating likely multidimensionality of the data.

Across all models, general factors showed good reliability (ω ranging from 86.2% to 93.2%) and specific factors showed acceptable to good reliability (ωs ranging from 77.4% to 89.6%). With the exception of the general factor in Model E which collapsed (ωH = 24.6%), general factors across all models showed high percentages of systematic variance while accounting for specific factors (ωH ranging from 73.5% to 77.6%). Compared to ωs coefficients (average ωs = 83.9%), ωHS estimates for specific factors were significantly reduced once controlling for the general factor (average ωHS = 50.3%). To illustrate this discrepancy further, I also measured relative ω, which compares ωH and ωHS to their coefficient omega counterparts (ω and ωs). Relative ω reflects the percentage of reliable variance in general factors while accounting for specific factors and in specific factors while accounting for general factors. With the exception of Model E's collapsed general factor (relative ω = 28.5%), relative ω for general factors across all models ranged from 79.6% to 83.2%, showing how most of the reliable variance in unit-weighted total scores is due to the general factor. In contrast, relative ω for specific factors showed greater variability, with values ranging from 18.5% to 100%.

The *H* index was high for general factors across all models (range = .703 to .899); however, specific factors showed greater variability. Whereas the *H* index ranged from .547 to .913 for specific factors, unacceptable *H* index values (i.e., < 0.7) were evident only for the peer problems subscale (see Table 8). FD was above the acceptable threshold for general factors in Models C, D, and F (range .919 to .94). FD for specific factors were lower than for general factors and varied (range .757 to .975).

PUC ranged from 52.6% to 84.2%, showing that across all models the majority of correlations reflect variance from the general factor alone (Bonifay et al., 2015). ARPB varied across models, ranging from 15.6% to 357.1%, indicating some issues with potential parameter bias given that previous work suggests that bias greater than 15% may pose some serious concerns (Muthén et al., 1987; Rodriguez et al., 2016).

3.1.3 External correlates of 'p'

I extracted factor scores from the overall best fitting model in the preceding section in terms of model fit statistics, factor loadings, and reliability statistics. Based on these statistics, factor score estimates for the general factor from Model F were correlated with external variables (Table 9). Gender was significantly correlated with the p-factor from Model F, such that boys scored significantly higher on general psychopathology. Further, greater levels of deprivation were significantly associated with greater levels of general psychopathology. Depression, anxiety, and impulsivity showed weak but significant associations with general psychopathology, whereas prosocial behaviour showed a significant moderate association (r = -.508, p < .001), such that greater levels of general psychopathology were associated with lower levels of prosocial behaviour.

Table 9

Correlations between factor score estimates from the general factor in Model F and external correlates including sociodemographic, behavioural and personality variables.

Correlates	P-factor
Gender (girls = 0)	.156***
Age	-.01
Deprivation Index (Age 8)	.142***
DSIS-C	.240***
Depression (CES-DC)	.235***
Anxiety (PROMIS)	.175***
Prosocial behaviour 8	-.508***

Note. * $p < .05$. ** $p < .01$. ***$p < .001$. Model F: S-1 bifactor model including one general factor with conduct problems subscale set as the reference domain, and three correlated specific factors (hyperactivity/inattention, peer, and emotion problems subscales).

3.2 Analysis 2

3.2.1 Analysis 2: Adversity and total behavioural difficulties

Table 10 shows frequency distributions for the total sample in terms of socio-demographic characteristics as well as for key predictors. Table S1 shows missing data and bivariate correlations for indicators used to construct adversity dimensions. Overall, missing data was low and ranged from 0-4.9% for all items, with the exception of items related to interparental violence (23.3%). Average child age in the sample was approximately 8.5 years (range = 7.75 - 9.75; *SD* = 0.42) and approximately 13% of the sample had TDS within the borderline and abnormal range. The average (*SD*) number of experiences of threat, deprivation, and unpredictability were 0.72 (0.96), 1.51 (1.49), and 1.01 (1.14), respectively. In the present study, 482 (10.4%) child participants experienced no adversity, 196 (4.2%) experienced only threat, 702 (15.2%) experienced only deprivation, and 459 (9.9%) experienced only unpredictability.

Table 10

Sample descriptives and frequency distribution across predictors, sociodemographic covariates, and total behavioural difficulties at age 8 years in the full sample (N = 4632) and mediation analysis subsample (N = 4158).

	Full Sample	Mediation Analysis Subsample
	N (%)	N (%)
Child Gender		
Male	2373 (51.2%)	2105 (50.6%)
Female	2259 (48.8%)	2053 (49.4%)
Maternal Ethnicity at baseline		
NZ European	2995 (64.7%)	2827 (68%)
Maori	531 (11.5%)	457 (11.0%)
Pacific	395 (8.5%)	338 (8.1%)
Asian	559 (12.1%)	469 (11.3%)
Other	147 (3.2%)	66 (1.6%)
DK/RF	4 (.1%)	1 (.0%)
Deprivation Index (Antenatal)		
High	1378 (29.7%)	1216 (29.2%)
Medium	1778 (38.4%)	1615 (38.8%)
Low	1373 (29.6%)	1262 (30.4%)
Missing	103 (2.2%)	65 (1.6%)
SDQ Total Difficulties Score at 8 years		
Abnormal	148 (3.2%)	120 (2.9%)
Borderline	474 (10.2%)	422 (10.1%)
Normal	4010 (86.6%)	3616 (87.0%)
Threat Composite Score		
0	2509 (54.2%)	2266 (54.5%)
1	1264 (27.3%)	1129 (27.2%)
2	573 (12.4%)	509 (12.2%)
3	286 (6.2%)	254 (6.1%)

Deprivation Composite Score		
0	1387 (29.9%)	1253 (30.1%)
1	1337 (28.9%)	1207 (29.0%)
2	886 (19.1%)	790 (19.0%)
3	512 (11.1%)	453 (10.9%)
4	510 (11%)	455 (10.9%)
Unpredictability Composite Score		
0	1912 (41.3%)	1726 (41.5%)
1	1487 (32.1%)	1339 (32.2%)
2	754 (16.3%)	677 (16.3%)
3	479 (10.3%)	416 (10.0%)

Table 11 presents the results of a hierarchical binary logistic regression to test the association of different dimensions of adversity with TDS at 8 years while controlling for socio-demographic covariates. No significant interactions between maternal ethnicity and gender with predictors of interest (threat, deprivation, and unpredictability) on total behavioural difficulties were detected (Table S2). The first block of the model which contained socio-demographic covariates, namely gender and ethnicity, explained a significant proportion of the variance in total behavioural difficulties ($X^2(2) = 64.943$, Nagelkerke $R^2 = .026$, $p < .001$). Gender and maternal ethnicity were both significantly related to behavioural difficulties, such that boys and participants whose mothers were not New Zealand European were 1.83 and 1.45 times, more likely to have behavioural difficulties in the borderline to abnormal range (both p's $< .001$) than girls and those with mothers who were New Zealand European, respectively. The second block, which represents the final model containing the adversity dimensions threat, deprivation, and unpredictability, significantly increased the variance accounted for ($X^2(3) = 271.372$, Nagelkerke R^2 change $= .103$, $p < .001$).

Table 11

Hierarchical binary logistic regression model testing effects of adversity dimensions on total behavioural difficulties at age 8 years in presence of socio-demographic covariates (N = 4628).

	Model 1		Model 2	
Variable	OR	95% CI	OR	95% CI
Maternal Ethnicity (0 = NZ Euro)	1.448***	(1.218, 1.721)	0.871	(0.714, 1.062)
Gender (0 = Girl)	1.832***	(1.537, 2.183)	1.797**	(1.498, 2.156)
Deprivation			1.234***	(1.147, 1.327)
Threat			1.557***	(1.418, 1.711)
Unpredictability			1.362***	(1.249, 1.485)
Nagelkerke R Square	.026		.129	

Note. * $p < .05$. ** $p < .01$. ***$p < .001$. OR = Odds ratio; CI = confidence interval.

Results from the final model show that there was a significant effect of gender, such that boys were approximately twice as likely to have behavioural difficulties in the borderline or abnormal range compared to girls ($p < .001$). All three dimensions of adversity were significantly associated with having behavioural difficulties in the borderline to abnormal range (Table 11). The model showed that each additional experience of deprivation was associated with an approximate 23% increase in the odds of having behavioural difficulties in the borderline to abnormal range ($p < .001$). Additionally, each additional experience of threat was associated with an approximate 55% increase in the odds of having behavioural difficulties in the borderline to abnormal range ($p < .001$). Lastly, each additional experience

of unpredictability was associated with an approximate 36% increase in the odds of having borderline or abnormal behavioural difficulties ($p < .001$).

3.2.2 Analysis 2: Mediating effect of executive control

As noted earlier, a total of four mediation models were conducted using multiple linear regression analysis (Baron & Kenny, 1986) to investigate whether executive control (as measured by the Luria Handclap Task) mediates the association between different adversity dimensions and TDS at age 8 years. First, I examine this mediating effect with each adversity dimension individually with sociodemographic covariates and then examine the mediating effect when all adversity dimensions are included in the model simultaneously (results shown in Tables 12-15).

Multiple linear regression analysis was used to test the hypothesis that executive control mediates the effect of deprivation on behavioural difficulties in the presence of sociodemographic covariates (Table 12). First I regressed executive control onto our measure of deprivation and covariates (i.e., gender, child age and total behavioural difficulties at age 4.5 years, and ethnicity). The model explained a significant 4.37% of the variance in executive control ($F(5, 4147) = 34.92$, $R^2 = .0437$, $p < .001$). As shown in Table 12, gender (dummy coded; girls = 0; $b = -.55$, $SE = .149$, $p = .0002$), previous levels of behavioural difficulties ($b = -.143$, $SE = .017$, $p < .0001$), and ethnicity (dummy coded; NZ European = 0; $b = -0.361$, $SE = 0.178$, $p = .042$) negatively correlated with executive control. Further, child age was significantly associated with executive control performance ($b = 0.188$, $SE = 0.057$, $p = .001$). After controlling for these socio-demographic covariates, deprivation was negatively correlated with executive control ($b = -0.303$, $SE = 0.066$, $p < .0001$), indicating that greater levels of deprivation were associated with poorer performance on an executive control task.

Table 12

Mediated multiple linear regression analysis showing indirect effect of Deprivation (X) on total difficulties scores at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates and previous levels of behavioural difficulties.

Outcome	Predictor	B	SE	t	*p*	95% CI	R^2
Luria Handclap Task	Gender (0 = Girl)	-.550	0.149	-3.700	.0002	(-0.842, -0.258)	.0437
	Total Difficulties at 54 months	-0.143	0.017	-8.571	< .0001	(-0.176, -0.110)	
	Ethnicity (0 = NZ Euro)	-0.361	0.178	-2.025	.0429	(-0.710, -0.012)	
	Child age at 54 months	0.188	0.057	3.273	.0011	(0.075, 0.300)	
	Deprivation	-0.303	0.066	-4.594	< .0001	(-0.433, -0.174)	
Total Difficulties at age 8 (Y)	Gender	0.918	0.136	6.741	< .0001	(0.651, 1.185)	.2846
	Total Difficulties at 54 months	0.506	0.016	31.572	< .0001	(0.475, 0.538)	

Ethnicity (0 = NZ Euro)	-0.526	0.160	-3.290	.001	(-0.839, -0.212)
Child age at 54 months	0.034	0.054	0.630	.5286	(-0.072, 0.140)
Deprivation (X)	0.287	0.059	4.846	< .0001	(0.171, 0.403)
Luria Handclap Task (M)	-0.087	0.014	-5.671	< .0001	(-0.116, -0.057)
Indirect Effect of X on Y	0.026	0.007	-	-	(0.013, 0.042)

Note. N = 4153. Unstandardized regression coefficients are reported.

The second part of the above-mentioned model involved regressing TDS at age 8 years onto executive control, deprivation and covariates (Table 12). The model explained a significant 28.46% of the variance in total behavioural difficulties ($F(6, 4146) = 237.83$, $R^2 = 0.2846$, $p < .001$). As shown in Table 11, gender (girls = 0; $b = 0.918$, $SE = 0.136$, $p < .0001$) and previous levels of behavioural difficulties ($b = 0.506$, $SE = 0.016$, $p < .0001$) were positively correlated with TDS at age 8 years, whereas ethnicity ($b = -0.526$, $SE = 0.16$, $p = .001$) correlated negatively. After adjusting for these covariates, deprivation ($b = 0.287$, $SE = 0.059$, $p < .0001$) positively correlated with TDS whereas executive control ($b = -0.087$, SE - 0.014, $p < .0001$) correlated negatively. This indicates that greater levels of deprivation are associated with higher levels of behavioural difficulties whereas increases in executive control are associated with fewer. Further, I tested the indirect effect of deprivation on TDS through executive control using a percentile bootstrap estimation approach with 10000 samples (Hayes, 2012). The indirect effect of deprivation on TDS through executive control was significant ($b = 0.026$, $SE = 0.007$, 95%BCI = (0.013, 0.042)) as zero was not included in the 95% CI. This supports partial mediation of deprivation on TDS through deficits in an executive control task.

The second mediation model tested the hypothesis that executive control mediates the association between threat and TDS in the presence of socio-demographic covariates (Table 13). First, I regressed executive control onto our measure of threat and covariates (as above). The model explained a significant 3.96% of the variance in executive control ($F(5, 4147) = 31.44$, $R^2 = .0396$, $p < .001$). As presented in Table 13, gender ($b = -0.518$, $SE = 0.15$, $p = .0005$), previous levels of behavioural difficulties ($b = -0.145$, $SE = 0.018$, $p < .0001$), and ethnicity ($b = -0.53$, $SE = 0.174$, $p = .0024$) were negatively correlated with executive control. Additionally, child age was positively correlated with executive control ($b = 0.174$, $SE = 0.058$, $p = .0025$). After controlling for these socio-demographic covariates, threat was

negatively associated with executive control (b = -0.224, SE = 0.1, p = .0243), such that greater experiences of threat were associated with poorer performance on an executive control task.

Table 13

Mediated multiple linear regression analysis showing indirect effect of Threat (X) on total difficulties scores at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates and previous levels of behavioural difficulties.

Outcome	Predictor	B	SE	t	*p*	95% CI	R^2
Luria Handclap Task	Gender	-0.518	0.150	-3.463	.0005	(-0.811, -0.225)	.0396
	Total Difficulties at 54 months	-0.145	0.018	-8.203	< .0001	(-0.180, -0.110)	
	Ethnicity (0 = NZ Euro)	-0.530	0.174	-3.043	.0024	(-0.872, -0.189)	
	Child age at 54 months	0.174	0.058	3.020	.0025	(0.061, 0.287)	
	Threat	-0.224	0.100	-2.253	.0243	(-0.419, -0.029)	
Total Difficulties at age 8 (Y)	Gender	-0.88	0.14	-6.40	< .0001	(-1.14, -0.61)	.2840
	Total Difficulties at 54 months	0.494	0.017	28.821	< .0001	(0.460, 0.527)	

Ethnicity (0 = NZ Euro)	-0.437	0.155	-2.827	.0047	(-0.740, -0.134)
Child age at 54 months	0.044	0.054	0.819	.413	(-0.061, 0.150)
Threat (X)	0.401	0.095	4.232	< .0001	(0.215, 0.587)
Luria Handclap Task (M)	-.089	0.015	-5.868	< .0001	(-0.119, -0.060)
Indirect Effect of X on Y	0.020	0.010	-	-	(0.003, 0.040)

Note. N = 4153. Unstandardized regression coefficients are reported.

Next, I regressed TDS at age 8 years onto executive control, threat, and covariates (Table 13). The model explained a significant 28.4% of the variance in executive control ($F(6, 4146) = 235.15$, $R^2 = 0.284$, $p < .001$). Gender ($b = -0.88$, $SE = 0.14$, $p < .0001$) and ethnicity ($b = -0.437$, $SE = 0.155$, $p = .0047$) were negatively associated with TDS at age 8 years, whereas previous levels of behavioural difficulties correlated positively ($b = 0.494$, $SE = 0.017$, $p < .0001$). After adjusting for these covariates, threat was associated with higher levels of behavioural difficulties ($b = 0.401$, $SE = 0.095$, $p < .0001$), whereas greater executive control was associated with lower behavioural difficulties ($b = -0.089$, $SE = 0.015$, $p < .0001$). Using a percentile bootstrap estimation approach with 10000 samples, the indirect effect of threat on TDS through executive control was significant as zero was not included in the 95% CI ($b = 0.02$, $SE = 0.01$, 95%BCI = (0.003, 0.04)). This supports partial mediation of threat on TDS through executive control in the presence of socio-demographic covariates.

The third mediation model tests the hypothesis that executive control mediates the association between unpredictability and TDS at age 8 years in the presence of socio-demographic covariates (Table 14). As above, executive control was regressed onto unpredictability and socio-demographic covariates, which explained a significant 3.95% of the variance ($F(5, 4147) = 31.45$, $R^2 = 0.0395$, $p < .001$). Results are presented in Table 14, which show that gender ($b = -0.534$, $SE = 0.149$, $p = .0004$), previous levels of behavioural difficulties ($b = -0.153$, $SE = 0.017$, $p < .0001$), and ethnicity (NZ European = 0; $b = -0.614$, $SE = 0.17$, $p = .0003$) were negatively correlated with executive control. However, child age at 4.5 years was positively correlated with executive control performance ($b = 0.177$, $SE = 0.058$, $p = .0022$). After controlling for these socio-demographic covariates, unpredictability was negatively associated with executive control ($b = -0.177$, $SE = 0.071$, $p = .0279$), such that greater levels of unpredictability were associated with poorer performance on an executive control task.

Table 14

Mediated multiple linear regression analysis showing indirect effect of Unpredictability (X) on total difficulties scores at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates and previous levels of behavioural difficulties.

Outcome	Predictor	B	SE	t	*p*	95% CI	R^2
Luria Handclap Task	Gender	-0.534	0.149	-3.578	.0004	(-0.826, -0.241)	.0395
	Total Difficulties at 54 months	-0.153	0.017	-9.227	< .0001	(-0.186, -0.121)	
	Ethnicity (0 = NZ Euro)	-0.614	0.170	-3.611	.0003	(-0.948, -0.281)	
	Child age at 54 months	0.177	0.058	3.062	.0022	(0.064, 0.290)	
	Unpredictability	-0.177	0.081	-2.200	.0279	(-0.335, -0.019)	
Total Difficulties at age 8 (Y)	Gender	0.908	0.136	6.693	< .0001	(0.642, 1.174)	.2895
	Total Difficulties at 54 months	0.499	0.016	31.287	< .0001	(0.0468, 0.531)	
	Ethnicity (0 = NZ Euro)	-0.286	0.151	-1.889	.059	(-0.583, 0.011)	

Child age at 54 months	0.033	0.054	0.599	.5491	(-0.074, 0.139)
Unpredictability (X)	0.518	0.076	6.812	< .0001	(0.369, 0.667)
Luria Handclap Task (M)	-0.088	0.015	-5.813	< .0001	(-0.118, -0.058)
Indirect Effect of X on Y	0.016	0.008	-	-	(0.002, 0.032)

Note. N = 4153. Unstandardized regression coefficients are reported.

As in previous mediation models, TDS at age 8 years was regressed onto unpredictability, executive control, and socio-demographic covariates (Table 14). The model explained a significant 28.95% of the variance in the outcome ($F(6, 4146) = 240.84$, $R^2 = .2895$, $p < .001$). As seen in Table 14, gender ($b = 0.908$, $SE = 0.136$, $p < .0001$) and previous levels of behavioural difficulties ($b = 0.499$, $SE = 0.016$, $p < .0001$) were positively correlated with TDS at age 8 years. After controlling for these potential confounders, unpredictability was positively associated with TDS ($b = 0.518$, $SE = 0.076$, $p < .0001$), whereas executive control correlated negatively ($b = -0.088$, $SE = 0.015$, $p < .0001$). As above, a percentile bootstrap estimation approach with 10000 samples was used to estimate indirect effects which showed a significant indirect effect of unpredictability on TDS through executive control ($b = 0.016$, $SE = 0.008$, 95%BCI = (0.002, 0.032) as zero was not included in the 95% CI. This supports partial mediation of unpredictability on TDS by executive control in the presence of socio-demographic covariates.

The final mediation model tested the mediating effect of deprivation on TDS through executive control, adjusting for other adversity dimensions (i.e., threat and unpredictability) and socio-demographic covariates (Table 15). First, I regressed executive control on all three adversity dimensions and socio-demographic covariates. The model explained a significant 4.45% of the variance in the outcome ($F(7,4145) = 25.17$, $R^2 = 0.0445$, $p < .001$). Gender ($b = -0.542$, $SE = 0.149$, $p = .0003$) and previous levels of behavioural difficulties ($b = -0.13$, $SE = 0.018$, $p < .0001$) were negatively correlated with executive control. In addition, child age was positively associated with executive control ($b = 0.192$, $SE = 0.058$, $p = .009$), whereas ethnicity was not significantly associated with executive control. Controlling for these confounders, threat and deprivation were not significantly associated with executive control. However, deprivation was negatively associated with executive control ($b = -0.27$, $SE =$

0.068, p = .0001), indicating that greater levels of deprivation were associated with poorer performance on the executive control task.

Table 15

Mediated multiple linear regression analysis showing indirect effect of Deprivation (X) on total difficulties scores at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates, previous levels of behavioural difficulties, and other adversity dimensions.

Outcome	Predictor	B	SE	t	p	95% CI	R^2
Luria Handclap Task	Gender	-0.542	0.149	-3.632	.0003	(-0.834, -0.249)	0.0445
	Total Difficulties at 54 months	-0.130	0.018	-7.248	< .0001	(-0.166, -0.095)	
	Ethnicity (0 = NZ Euro)	-0.337	0.181	-1.864	.0624	(-0.692, 0.017)	
	Child age at 54 months	0.192	0.058	3.326	.009	(0.079, 0.305)	
	Deprivation	-0.270	0.068	-3.970	.0001	(-0.404, -0.137)	
	Threat	-0.137	0.101	-1.360	.1738	(-0.335, 0.061)	
	Unpredictability	-0.091	0.082	-1.103	.2701	(-0.252, 0.071)	

Total Difficulties at age 8 (Y)	Gender	0.903	0.136	6.646	< .0001	(0.636, 1.169)	0.2934
	Total Difficulties at 54 months	0.471	0.017	27.324	< .0001	(0.437, 0.505)	
	Ethnicity (0 = NZ Euro)	-0.537	0.160	-3.354	.0008	(-0.851, -0.223)	
	Child age at 54 months	0.019	0.054	0.354	.7234	(-0.087, 0.125)	
	Deprivation (X)	0.173	0.061	2.853	.0044	(0.054, 0.292)	
	Threat	0.293	0.096	3.050	.0023	(0.105, 0.481)	
	Unpredictability	0.444	0.077	5.760	< .0001	(0.293, 0.595)	
	Luria Handclap Task (M)	-0.083	0.015	-5.514	< .0001	(-0.113, -0.054)	
	Indirect Effect of X on Y	0.022	0.007	-	-	(0.010, 0.038)	

Note. N = 4153. Unstandardized regression coefficients are reported.

Lastly, TDS at age 8 years were regressed onto all adversity dimensions, executive control, and socio-demographic covariates (Table 15). The model explained a significant 29.34% of the variance in the outcome ($F(8, 4144) = 182.93$, $R^2 = 0.2934$, $p < .0001$). The results showed that gender ($b = 0.903$, $SE = 0.136$, $p < .0001$) and previous levels of behavioural difficulties ($b = 0.471$, $SE = 0.017$, $p < .0001$) were positively associated with TDS at age 8 years. Further, ethnicity was negatively associated with TDS at age 8 years (dummy coded; NZ European = 0; $b = -0.537$, $SE = 0.16$, $p = .0008$). Controlling for these covariates, all three adversity dimensions had significant direct effects on TDS, such that greater experiences of threat ($b = 0.293$, $SE = 0.096$, $p = .0023$), deprivation ($b = 0.173$, $SE = 0.061$, $p = .0044$), and unpredictability ($b = 0.444$, $SE = 0.077$, $p < .0001$) were associated with higher TDS. A bootstrap estimation approach with 10000 samples showed that the indirect effect of deprivation on TDS through executive control was significant when adjusting for socio-demographic covariates and all adversity dimensions simultaneously ($b = 0.022$, $SE = 0.007$, 95%CI = (0.01, 0.038)), given that confidence intervals did not include zero and thus showing a partial mediation effect.

3.2.4 Sensitivity Analyses

I also ran additional sensitivity analyses to test the robustness of these findings. In the first sensitivity analysis, I compared the mediating effects of deprivation on TDS at age 8 years through executive control when measuring deprivation cognitively or materially. Results are shown in Table S3 and S4 in the supplemental materials and largely follow the same pattern of results. Adjusting for covariates, deprivation, when measured using indicators such as material poverty and family income, was the only adversity dimension associated with executive control ($b = -0.333$, $SE = 0.084$, $p = .0001$). Similarly, when measuring deprivation through cognitive indicators alone, deprivation was the only dimension associated with executive control when adjusting for potential confounders ($b = -0.197$, $SE =$

0.1, p = .0495). Both material (b = 0.028, SE = 0.009, 95%CI = (0.013, 0.047)) and cognitive deprivation (b = .017, SE = .009, 95%CI = (0.0002, 0.0365)) showed significant indirect effects on TDS through executive control using a bootstrap estimation approach with 10000 samples. A notable difference between the two models is the significant direct effect of deprivation on total behavioural difficulties when measured using material indicators (b = 0.208, SE = 0.076, p = .0064), which was not seen for deprivation when measured via cognitive indicators alone.

4 Discussion

There were two main aims of this thesis: a) to assess several structural models of psychopathology on the total difficulties score (TDS) of the Strengths and Difficulties Questionnaire (SDQ) and determine external correlates of the best fitting model; and b) to test whether childhood executive control (EC) mediates the association between different dimensions of adversity and behavioural and emotional problems at age 8 years. I hypothesized that greater levels of threat, deprivation, and unpredictability, will be associated with higher levels of behavioural and emotional problems at age 8 years adjusting for the effects of socio-demographic confounders. Additionally, I hypothesized that threat and unpredictability would be mediated by executive control when examined individually, but not when controlling for other adversity dimensions. Lastly, I hypothesized that the effect of deprivation, but not threat and unpredictability, will have a significant indirect effect on behavioural and emotional problems at age 8 years through executive control in a combined mediation model controlling for socio-demographic covariates, previous levels of behavioural and emotional problems at age 4.5 years, and all adversity dimensions simultaneously.

To help the reader anticipate the structure of this chapter, the first two sections discuss the findings and implications of Analyses 1 and 2 with respect to the literature, respectively. Following those sections, the limitations, strengths, and future directions of my research are outlined and discussed. Some implications of this research are outlined, and final conclusions stated.

In brief and overall, the findings show that bi-factor models of the SDQ yield better fit to the data, but often contain anomalous results (i.e., factor loadings that were not significant, close-to-zero, or negative), especially with regards to the interpretability of specific factors. Further, bi-factor S-1 models offer a suitable and theoretically superior alternative to modelling TDS of the SDQ, demonstrating links between a general factor marked by conduct

problems and behavioural/psychological correlates such as symptoms of depression and anxiety, impulsivity, and prosocial behaviour. Next, the findings showed both direct and indirect effects of different dimensions of adversity on behavioural and emotional problems in middle childhood. When examined individually, each adversity dimension was associated with greater behavioural and emotional problems in middle childhood via poorer performance on an executive control task; however, when examined simultaneously, only indirect effects of deprivation continued to be significant.

4.1 Factor structure and reliability of the SDQ TDS

As noted from the outset, the present study uses the term psychopathology to refer to mental health difficulties rather than diagnosed or severe psychopathology. This is to maintain consistency with the terminology used in this line of research. The extant literature on general and specific forms of psychopathology has been positioned as a first step towards a transdiagnostic approach to psychopathology. Much of the literature on general psychopathology has been investigated with diagnostic-level data (Caspi et al., 2014; Lahey et al., 2012) and symptom-level data (Carragher et al., 2016; Patalay et al., 2015). Within the literature using symptom-level data, comparatively few studies have used the SDQ as a primary instrument, with most studies using the CBCL as the 'gold standard' (Hoffman et al., 2022). The current study tested popular structural models of psychopathology on the TDS of the parent-reported SDQ in a large nationally representative cohort of 8-year-old children. Model fit and reliability varied across model specification, with the S-1 bi-factor model showing the best fit and overall reliability. I elaborate on these findings with regards to the extant literature below.

The results showed that bi-factor models fit the data best, with a one-factor solution and the popular correlated-factors model showing poor fit to the data. This echoes previous findings that show superior fit of bi-factor models compared to a correlated-factors model

(Afzali et al., 2018; Castellanos-Ryan et al., 2016; Carragher et al., 2016; Haltigan et al., 2016; Lacuelle et al., 2015; Lahey et al., 2015; Murray et al., 2016; Patalay et al., 2015). Although showing poor model fit, the traditional correlated-factors model showed a moderate association between internalizing and externalizing dimensions of the SDQ ($r = .57$, $p < .001$), which is consistent with previous findings (Conway et al., 2019; Cosgrove et al., 2011; du Pont et al., 2017; Lahey et al., 2004). Further, the results support previous structural analyses that identify broader internalizing and externalizing dimensions, as correlations between specific factors in Model D showed strong associations between hyperactivity/conduct ($r = .61$, $p < .001$) and peer/emotional problems ($r = .65$, $p < .001$) (Goodman et al., 2010).

Although all bi-factor models in the present study showed superior fit compared to both a one-factor and correlated-factors model (all CFI and TLI > 0.90; RMSEA < 0.80; and SRMR < 0.60), closer observation of factor loadings reveal important differences in interpretability. Bi-factor models C, D, and E showed anomalous results with respect to either general or specific factors. For example, Model C showed an externalizing factor marked strongly by the hyperactivity/inattention problems subscale and an internalizing factor marked strongly by the emotional problems subscale. However, the conduct problems and peer problems subscales included anomalous results, which complicates interpretability. As highlighted by Heinrich et al. (2023), when a specific factor partially collapses, the general factor's interpretation is changed, and specific factors become difficult to interpret. As such, the general factor in Model C appears to primarily reflect a liability to conduct and peer problems, rather than a general factor underlying all aspects of child mental health problems assessed. Similarly, the conduct problems partially collapsed in Model D, evidenced by factor loadings that were not significant or close to zero. In contrast, the general factor partially collapsed in Model E, also evidenced by factor loadings that were negative, not significant, or

close to zero. This is consistent with prior work which has shown that anomalous results are commonly found across symmetrical bi-factor models measuring 'p' or other constructs such as depression or ADHD (Burns et al., 2020; Eid et al., 2017; Heinrich et al., 2023). It is also consistent with general factors from other studies that show a liability for antagonism and distress, rather than a general liability for all psychiatric disorders (Castellanos-Ryan et al., 2016; Lahey et al., 2012).

Previous research of bi-factor models using the SDQ have included the strength-based prosocial scale in their analyses, which complicates comparisons with findings from the present thesis regarding the structure of the TDS. Kobor et al. (2013) found support for a bi-factor model using teacher and parent-reported SDQ data, which was specified with one general factor and five specific factors allowed to freely corelate. Unlike the results of the present thesis, the general factor in their bi-factor model did not collapse when allowing specific factors (including the prosocial subscale) to correlate freely (Kobor et al., 2013). While they included the prosocial score subscale in their analysis, they report some ancillary statistics which show similar strength of the general factor (ECV = 53.3) and low reliability of specific factors (ωHs = .09, .59) (Kobor et al., 2013). In contrast, Ortuno-Sierra et al. (2015) investigated a bi-factor model using self-reported SDQ data but found support for a first order five-factor model rather than a bi-factor model which showed anomalous results. In another study, researchers investigated a bi-factor model with three general factors (externalizing, internalizing, and prosocial dimensions) and four specific factors representing the four difficulties subscales of the teacher-reported SDQ (Caci et al., 2015). While they suggest utility in computing general and specific factor scores evidenced by high reliability estimates (ω = .804 - .926), they do not report other important ancillary statistics such as ωHs or ECV, which precludes any meaningful interpretation of the strength of general factors or the variance attributable to specific factors after partitioning variance due to the general

factor (Rodriguez et al., 2016). Collectively, these findings highlight the need for routine reporting of model-based reliability statistics in order to advance transdiagnostic research and its investigation through bi-factor models using the SDQ.

Keller and Langemeyer (2019) used an item response theory approach to analyse the parent-reported version of the SDQ, specifically the total difficulties score, and thus is one of the few direct comparisons that exist in the literature. They show clear evidence of multidimensionality in the total difficulties score, evidenced by a relatively weak general factor and a substantial proportion of the reliable variance explained by specific factors (Keller & Langemeyer, 2019). In support of these findings, the present results did not show evidence for a strong general factor, as across all models general factors were comprised of moderate to medium sized factor loadings and explained just over one half of the common variance (P factor ECV = 57.8%; Model C). In addition, the common variance explained by the specific factors, after partitioning out variance attributed to the general factor (ωHs), remained substantial and tended to be higher for subscales in the internalizing dimension and thus less reflected in the general factor, which is also consistent with prior work (Keller & Langemeyer, 2019). Adding to Keller and Langemeyer (2019), calculation of the ARPB showed significant differences between unidimensional and bi-factor solutions, with model D showing the lowest bias (15.6%). Prior work has suggested that bias greater than 15% work may pose some serious concerns (Muthén et al., 1987; Rodriguez et al., 2016). Taken together, the results discourage the use of parent-reported SDQ total difficulties scores as a unidimensional construct given clear multidimensionality in the data and a weak general factor.

Another practical issue that may be of interest to researchers is the derivation of factor score estimates or construct replicability of the SDQ subscales. Across all models, FD and H index for general factors were above the required threshold (FD > 0.90; H index > 0.7),

suggesting that factor score estimates of general factors are trustworthy and these latent factors are reliably specified (Rodriguez et al., 2016). However, these indices varied for specific factors, where FD values were considered below the required threshold for most specific factors, and H index being considered unacceptable for the internalising dimension in the symmetrical bi-factor model (Model C) and for the peer problems subscale across other models. Possible explanations could include issues in the categorical thresholds between 'somewhat true' and 'true' for items such as "Has at least one good friend", as this item should be interpreted as a dichotomous 'yes' or 'no' question (Hagquist, 2007; Keller & Langemeyer, 2019).

A novel aim of the present thesis was the estimation of a S-1 bi-factor model of the TDS in the SDQ and its' reliability coefficients and external correlates. The present thesis shows that an S-1 bi-factor model helps to eliminate anomalous results and aids interpretability of specific factors. While reliability statistics show evidence of multidimensionality, most of the variance in the model is explained by the general factor (ECV = 52.2%, PUC = 84.2%). Further, I tested associations between the general factor (marked by conduct disorders) and external correlates. As expected, boys experienced higher levels of general psychopathology, which is consistent with gender differences in the onset and development on children's conduct problems (Alvarez-Voces & Romero, 2024; Gorman-Smith & Loeber, 2005). In addition, there were links between the general factor and area level deprivation measured by the NZ Deprivation Index. While the results could not establish a causal relationship, this association is consistent with prior work demonstrating links between area/neighbourhood level deprivation and conduct problems or delinquent behaviours (Flouri et al., 2010; Galán et al., 2017). To the best of my knowledge, only one study has modelled the SDQ using a bi-factor S-1 model with conduct problems as the reference domain (Chen et al., 2022). Despite showing similarity in factor loadings to the

present results, their study did not report any external validation analyses or reliability statistics (Chen et al., 2022). The present findings address these gaps in the literature by evaluating this bi-factor S-1 model using "riskier tests" prior to their use in SEM, as proposed by prior research (Watts et al., 2019).

Heinrich et al. (2023) argue that defining a general factor a priori in a bi-factor S-1 model is advantageous due to interpretability of general and specific factors. In the present study, the general factor is clearly defined as the common variance captured by conduct problems, whereas, the specific factors reflect hyperactivity/inattention, peer, and emotional problems that cannot be explained by conduct problems. The findings show that this general factor is associated: weakly with self-reported depression, anxiety, and impulsivity; and moderately with lower prosocial behaviour. These associations shed light on the possibility that a transdiagnostic measure of conduct problems in a bi-factor S-1 model might resemble a dysregulated profile, which has been suggested as a screening measure for severe psychopathology (Deutz et al., 2018). Comorbidity between conduct and internalizing problems has been linked with increased delinquent behaviour longitudinally (Polier et al., 2012). Adding to this, researchers used autoregressive latent trajectory models to evaluate developmental cascades between externalizing and internalizing problems (Murray et al., 2020). They showed that externalizing problems became more differentiated in adolescence and were protective of internalizing problems (Murray et al., 2020). Given that the present findings were measured in middle childhood, these results may highlight effects of dynamic mutualism or p-differentiation that culminate later in adolescence, which has shown to be a period for the onset of most high-prevalence disorders (Girolamo et al., 2012; Paus et al., 2008). This highlights the potential to leverage bi-factor S-1 models to study the heterotypic or homotypic continuity of externalizing symptoms, given its advantage over symmetrical bi-

factor models in clearly defining the general factor, thus allowing its replication across different samples, developmental periods, and informants (Heinrich et al., 2023).

Notably, the associations between hyperactivity/inattention and peer/emotional problems in Model D (*r's* = .422 and .424, respectively) were substantially attenuated in a bi-factor S-1 model (*r's* =.149 and .1, respectively). These results may provide evidence for the transdiagnostic features that culminate in the substantial clinical heterogeneity of conduct disorders (Burt, 2013; Klahr & Burt, 2014). Recent research has shown that certain combinations of psychological factors that may be at play, mainly co-occurrence of interpersonal callousness (i.e., deficit in prosocial emotions) with impulsivity, irritability, and/or symptoms of anxiety and depression (Dugre & Potvin, 2022). Other studies have shown that both parent and teacher-reported callous-unemotional traits rather than conduct problems were significantly associated with lower prosocial behaviour (Milledge et al., 2019). As such, the current findings demonstrating stronger associations between the general factor with prosocial behaviour, compared to impulsivity and symptoms of anxiety and depression, could highlight the role of features such as interpersonal callousness as a transdiagnostic feature of conduct problems. However, further construct validation is required.

There are important implications to be noted from this study. Comparison of model fit and ancillary statistics show the necessity of examining both to truly assess the validity and interpretability of bi-factor models. Although many studies have argued that the p-factor replicates well across different studies (Lahey et al., 2015; Noordhof et al., 2015; Snyder et al., 2019), this was solely determined via model fit, which as shown in the present study, is a fallible marker. As such, many studies claim that their p-factor represents a general liability for all psychiatric disorders, when interpretation of their factor loadings show an entirely different meaning. Watts et al. (2019) illustrate these arguments by proposing riskier tests of

validation, requiring the following: a) general factors are equally represented by their indicators; b) specific factors in bi-factor models are reliable and interpretable; and c) bi-factors improve on the external validity of a correlated-factors model. Their results highlight that the general factor in a bi-factor solution was representative of a distress/high externalizing behaviour factor rather than a general liability for all psychiatric disorders, which is similar to previous findings (Castellanos-Ryan et al., 2016; Lahey et al., 2012; Waldman et al., 2018). The present findings add to this literature showing the importance of stringent tests of bi-factor validity and reliability.

In defence of the p-factor literature, Caspi et al. (2023) utilized data from the E-risk Longitudinal Twin Study, based in the United Kingdom, to test a wide range of structural models of psychopathology. They found evidence that p-factors generated across many models (one-factor, higher-order, symmetrical and S-1 bi-factor models) were highly correlated with each other and showed similar reliability when comparing ancillary statistics (Caspi et al., 2023). Moreover, they show that the nomological network of the p-factor in terms of risk factors such as family psychiatric history, deprivation, and inflammation, were similar regardless of the model the p-factor was derived from (Caspi et al., 2023). However, they report that specific factors in hierarchical models should be interpreted with caution. They refer to the extant p-factor literature as a descriptive phase which has shed light that most psychiatric disorders share a factor, or several factors, in common. They conclude that p-factors across different models share an underlying dimension worth investigating, and that specific factors in these models are analogous to studying independent psychiatric disorders (Caspi et al., 2023).

As discussed above, there is still an ongoing debate on the legitimacy of the p-factor, with some arguing that these models are statistical artefacts (Eiko et al., 2021) and others suggesting that these statistical observations have provided the opportunity for a second

phase of research to investigate the putative causes of a general liability to psychiatric disorders (Caspi et al., 2023). In light of this debate, the present findings show the importance of evaluating bi-factor models using riskier tests of validity and reliability (Watts et al., 2019), and also demonstrate the utility of theory-driven bi-factor models in terms of interpretability, such as the S-1 bi-factor model (Heinrich et al., 2023). Given the use of the SDQ as an accessible alternative to the CBCL, the present study highlights how theory-driven S-1 bi-factor models may be leveraged to contribute to the p-factor literature. Screening questionnaires are often used as a first step in large-scale studies, generating insights to be confirmed by 'gold standards' such as structured diagnostic interview (Caci et al., 2015; Kessler et al., 2005). Further assessment of different bi-factor S-1 models using the SDQ and their construct validity may provide an opportunity to further this transdiagnostic research, circumventing the limitations of symmetrical bi-factor models and their issues with interpretability.

4.2 Adversity dimensions and the mediating role of EC

This theoretically informed study aimed to investigate the impact of different dimensions of adversity (i.e., threat, deprivation, and unpredictability) on total behavioural difficulties at age 8 years and the mediating role of EC in this relationship. Consistent with hypotheses, all adversity dimensions were significantly associated with greater likelihood of abnormal/borderline behavioural difficulties in middle childhood when controlling for socio-demographic covariates. Additionally, when examining all adversity dimensions simultaneously, only deprivation continued to be significantly associated with higher TDS via poorer performance on an EC task. These results were also robust to potential confounders such as ethnicity, gender, age of EC assessment, and previous levels of behavioural difficulties. To the best of my knowledge, this is the first study to illustrate these findings when examining dimensions from both the DMAP and Harshness-Unpredictability

framework simultaneously. I elaborate on these findings in relation to the extant literature below.

The results indicate that early childhood experiences of threat, deprivation, and unpredictability are significantly associated with increased odds of having borderline to abnormal behavioural difficulties in middle childhood. This is consistent with my hypotheses that different dimensions of adversity measured in early life would be associated with behavioural difficulties in middle childhood above the effects of socio-demographic covariates. These findings emphasize the indiscriminate nature of adversity in conferring risk for mental health difficulties and is consistent with previous research that documents the effects of different adversity exposures on psychopathology (Wade et al., 2022). Previous research has shown that threatening experiences such as the those measured in this study (i.e., exposure to domestic violence, peer bullying/victimization) are potent predictors of later mental health difficulties (Wade et al., 2022) and show elevated risk across both internalizing and externalizing disorders (Danese et al., 2020; Schaefer et al., 2018). Further supporting these results, both severe and common experiences of deprivation have been consistently linked with later mental health difficulties. For example, while institutionalization shows consistent strong links with the onset of many psychiatric disorders (Humphreys et al., 2020), less severe forms of deprivation such as poverty and low socioeconomic status also show increased risk for psychopathology (Palacios-Barrios et al., 2019; Peverill et al., 2021). In addition, cognitive deprivation associated with the immediate rearing environment, has also shown links with increased psychopathology (Miller et al., 2018; Miller et al., 2021). These findings are consistent with the present study, showing that deprivation defined by cognitive and material indicators, are associated with greater levels of borderline to abnormal behavioural difficulties. Lastly, the findings also showed that unpredictability, when measured using both ancestral cues and household chaos perspectives, was significantly

associated with increased odds of having borderline to abnormal behavioural difficulties in middle childhood. A rich literature supports these results, showing that early life unpredictability when measured according to the ancestral cues or household chaos perspectives, is associated with transdiagnostic risk for psychopathology (Doom et al., 2016; Dwairy, 2008; Hurst et al., 2017; Ross et al., 2016). However, the present study is one of the few studies to highlight associations between multiple adversity dimensions from different dimensional frameworks with later mental health difficulties. In sum, the results add to this limited literature (McGinnis et al., 2022; Phillips et al., 2023; Usacheva et al., 2022), and delineate how associations between myriad adversity dimensions and mental health difficulties are exemplified by equifinality within a sensitive window in early childhood.

Meta-analyses have shown that different forms of adversity have similar effect sizes on future risk for mental health difficulties, suggesting a non-specific relationship between adversity and later psychopathology (Angelakis et al., 2020; Gardner et al., 2019). The current results from a hierarchical binary logistic regression show that threat is more strongly related to later mental health difficulties compared to deprivation and unpredictability (55% more likely to have borderline/abnormal behavioral difficulties compared to 36% and 23%, respectively). Other studies have also shown the stronger associations between threat and psychopathology compared to deprivation (Stein et al., 2021), which could possibly be explained by the proximal mechanisms hypothesized by the DMAP (McLaughlin & Sheridan, 2014). Although effects of threat and deprivation on emotion regulation and cognitive ability, respectively, are evident in early childhood (Machlin et al., 2019; Miu et al., 2022), it is possible that deficits in emotion regulation are associated with immediate effects of psychopathology compared to the effects of cognitive disadvantage due to deprivation. The severity of experiences of deprivation might further compound this effect, where severe forms of deprivation, such as institutionalization, lead to greater deficits in cognitive ability

and, in turn, increases in psychopathology (relative to less severe forms of deprivation such as low cognitive stimulation in the household). One study showed evidence for transdiagnostic comorbidity in the deleterious effects of executive function impairment due to institutionalization, highlighting associations between executive function deficits and externalizing symptoms at age 12 years but no associations with psychopathology at age 16 years (Wade et al., 2021). In the same study, externalizing symptoms at age 12 years predicted symptoms of general psychopathology at age 16 years (Wade et al., 2021), which may suggest a pattern of dynamic mutualism, such that different symptoms reinforce each other over time to establish long-term comorbidity (Murray et al., 2016).

Another possible explanation that may explain different effect sizes for various types of adversity on psychopathology may be the role of unpredictability. Although requiring further research, preliminary findings show that greater unpredictability of threatening experiences may attenuate emotion dysregulation (Herry et al., 2007; Vansteenwegen et al., 2008), whereas greater unpredictability of experiences of deprivation may mitigate deficits in executive functioning by enhancing other cognitive skills such as cognitive flexibility (Fields et al., 2021; Mittal et al., 2015). As such, stronger associations between threat and psychopathology could possibly be explained by their interaction with unpredictability.

Consistent with my hypotheses, all three adversity dimensions examined in the present thesis were associated with later mental health difficulties through EC when examined individually. In a recent meta-analysis, Johnson et al. (2021) found that both experiences of threat and deprivation are associated with reduced executive functioning, with stronger associations for deprivation. Across the three domains of executive functioning, they reported no significant differences between the effects of threat and deprivation on cognitive flexibility, but showed stronger links between deprivation and both lower working memory and inhibitory control, which is the primary mechanism investigated in this thesis (Johnson et

al., 2021). The current research supports these findings, showing that, when examined separately, both threat and deprivation show associations with EC, but that this relationship is mitigated once examined simultaneously. This is also consistent with the DMAP, which holds cognitive ability as the proximal mechanism for experiences of deprivation and emotion regulation as the proximal mechanism for experiences of threat (McLaughlin et al., 2014).

Unlike threat and deprivation, the effects of unpredictability are more varied in the literature. Some studies report that greater levels of early life unpredictability are associated with enhanced effortful control (Warren & Barnett, 2020) or cognitive flexibility (Fields et al., 2021). In contrast, one meta-analysis showed small but significant reductions in executive function in relation to early life unpredictability (Andrews et al., 2021). Notably, the meta-analysis also showed that these associations were stronger when using measures of executive functioning reported by teachers or parents rather than standardized assessments (Andrews et al., 2021). In this study, early life unpredictability was associated with significant decreases in a directly assessed measure of EC. Additionally, I show that this relationship is attenuated when examining all adversity dimensions simultaneously, highlighting further work that needs to be done in this area to elucidate the role of unpredictability and its measurement, neurobiological consequences, and interactions with threat and deprivation. Despite this, the individual relationships between each of threat, deprivation, and unpredictability with EC on later mental health difficulties add to the literature on the utility of executive function as a transdiagnostic mechanism and target for intervention (Fleming et al., 2020; Mason et al., 2020; McNeilly et al., 2021; Zelazo, 2020).

Despite individual associations between each adversity dimension and EC, when examined simultaneously, deprivation was the only adversity dimension associated with small but significant increases in TDS in middle childhood via poorer performance on an EC

task during early childhood. These results are robust to potential confounders such as ethnicity, age at EC assessment, gender, and importantly, show increases in TDS in middle childhood above and beyond those in early childhood. Although the effects were small in size, they are comparable to other studies (Schäfer et al., 2021), and are unsurprising given that the study investigated these effects over a short time interval of 3 years. The results add to the mounting evidence of differential associations between adversity dimensions and development, as hypothesised by the DMAP (McLaughlin et al., 2014; McLaughlin & Sheridan, 2014). Prior work based on the DMAP, has shown that experiences of deprivation, and not threat, are associated with deficits in executive functioning cross-sectionally (Machlin et al., 2019; Sheridan et al., 2017) and longitudinally (Schäfer et al., 2021). DMAP research has also shown that this relationship exists for other markers of cognitive ability, such that only deprivation was associated with risk for psychopathology across childhood and adolescence via deficits in language ability (Miller et al., 2018; Miller et al., 2021).

In line with the present results, Phillips et al. (2023) investigated the role of unpredictability (defined by the ancestral cues perspective, e.g., residential moves) and deprivation (defined as lack of cognitive stimulation) on general psychopathology through preschool executive control. While the effects of deprivation and unpredictability on psychopathology were both mediated by preschool EC when examined separately, only deprivation emerged as a key predictor of later mental health difficulties through EC (Phillips et al., 2023). They also showed the role of distal risk factors in this process, such that deprivation, and not unpredictability, mediated the relationship between socioeconomic status and preschool EC (Phillips et al., 2023). In a recent large-scale longitudinal study, McGinnis et al. (2022) test the impact of threat, deprivation (defined as material deprivation), unpredictability (operationalised as residential moves, , and loss dimensions on myriad proximal and distal outcomes of development. Although they show distinct associations for

deprivation as the only dimension associated with lower IQ in early adulthood, they were unable to conduct mediation analyses (McGinnis et al., 2022). Important differences in these studies arises in their operationalisation of unpredictability. Whereas Phillips et al. (2023) used a frequency count of the total number of residential moves, partner separations, and employment loss; McGinnis et al. (2022) used dichotomous indicators of parental transitions and household moves, as well as other indicators such as reduced standard of living and change of school without friends. As above, this highlights that further research needs to be done regarding the operationalisation of unpredictability. However, the current pattern of results indicate the prominent role of deprivation over unpredictability in the pathway to mental health difficulties through EC. Importantly, these results are robust when using a different operationalisation of unpredictability, such as the use of dichotomised indicators from both the ancestral cues and household chaos perspective, as well as the inclusion of maternal depression as a marker of unpredictability rather than deprivation, which may have had a confounding effect in other studies (Usacheva et al., 2022).

The current results from a sensitivity analysis yielded interesting insights into the relationships between material and cognitive deprivation with both EC and later mental health difficulties. While both material and cognitive deprivation showed unique pathways to later mental health difficulties through poorer performance on an EC task, only material deprivation showed significant direct effects on later mental health difficulties. Similar patterns of results are seen in other studies that used different proxies for cognitive development, such as language abilities (Miller et al., 2021). In their study, Miller et al. (2021) report significant indirect effects of deprivation on psychopathology through lower language ability; however, their structural models showed no direct associations between deprivation and internalising or externalising psychopathology. Furthermore, Phillips et al.

(2023) show no direct effects of maternal education, income, or deprivation on general psychopathology in adolescence while also controlling for unpredictability simultaneously.

There are several explanations that could account for the different patterns of results between cognitive and material deprivation. As demonstrated by prior work (Schäfer et al., 2021; Stein et al., 2021) and the results from the hierarchical logistic regression in this thesis, it is possible that threat plays a more important and immediate role in the development of psychopathology compared to deprivation. Moreover, our finding regarding the significant direct effect of material deprivation on later mental health difficulties is consistent with the Harshness-Unpredictability framework (Ellis et al., 2009). Focusing on the 'why' of development, the Harshness-Unpredictability framework extends the Threat-Deprivation framework to a broader ecological context and suggests that higher levels of harshness (akin to both threat and deprivation) will promote faster life history strategies (e.g., greater impulsivity and number of sexual partners), which in return confers risk for psychopathology (Ellis et al., 2022). Prior work has shown demonstrated links between income harshness on internalising externalising behaviours either directly (Li & Belsky, 2022) or indirectly via mechanisms suggested by life history theory, such as harsh parenting or substance abuse (Doom et al., 2016; Li & Belsky, 2022; Simpson et al., 2012). Further, one study has showed both direct and indirect associations of neighbourhood deprivation with conduct problems in middle childhood through aggressive response generation (Galán et al., 2017). This highlights the potential to examine this relationship through mechanisms proposed by the Harshness-Unpredictability framework. Nevertheless, the current results show that distal and proximal experiences of deprivation confer risk for psychopathology via deficits in EC. Lastly, investigation of other developmental outcomes with psychopathology simultaneously, such as health, may reveal interesting findings regarding life-history trade-offs due to energetic

deprivation, as specified by the integrated model of dimensions of environmental experience (Ellis et al., 2022).

Several implications of these findings should be noted. The patterns of results highlight the utility of dimensional models offering mechanistic explanations underlying the distinct pathways between adversity and psychopathology, which may have been obscured by a CR approach. As such, the findings demonstrate the explicit developmental focus of dimensional models in uncovering the 'why' and 'how' of development (Ellis et al., 2022). The results also demonstrate how the DMAP and Harshness-Unpredictability frameworks are theoretically complementary and allow for testable hypotheses of mechanisms other than commonly used stress-based explanations, emphasizing the interplay environmental influences and learning (McLaughlin & Sheridan, 2016). Further, the current findings represent one of the few studies in the extant literature that examined adversity dimensions both frameworks with proximal mechanisms simultaneously; which addresses previous gaps in the literature (McGinnis et al., 2022; Phillips et al., 2023).

In addition to their theoretical significance, the clear conceptualisation of different forms of adversity could aid policymakers in reducing childhood disadvantage. Given that experiences of early life threat, deprivation, and unpredictability had direct effects on TDS in middle childhood, policies can be designed to address specific forms of adversity and thus reduce disparities in child/adolescent mental health. For example, prior GUiNZ work has shown that the most disadvantaged children continue to face high residential mobility into middle childhood compared to their privileged peers (Davies et al., 2022). The present findings extends this work, demonstrating the co-occurrence between different types of adversity and the extended effects they can have on child behavioural development. Additionally, the results highlight the importance of incorporating different levels of the environment when considering childhood disadvantage. For example, unpredictability was

measured using indicators such as number of residential moves, number of caregiving arrangements, and maternal depression, all of which contributed to later mental health difficulties. Policy-wise, these indicators may reflect financial burden, instability, or issues with the parent-child relationship, and as such the findings echo previous recommendations that highlight the importance of investment into economic, physical, social, and human resources for optimal child development (Davies et al., 2022; Morton et al., 2021). In sum, the current results emphasize the need for early intervention for children who have experienced, threat, deprivation, and unpredictability in early life.

The results also showed direct effects of material deprivation in early life on emotional and behavioural problems years later in middle childhood. This direct effect was illustrated using both area-level and individual-level indicators of deprivation, which has important implications for policy. Previous GUiNZ research has shown insights into neighbourhood deprivation in New Zealand, reporting relatively greater mobility between different levels of deprivation in middle childhood, but that this residential trajectory wasn't seen for those living in the most deprived neighbourhoods (Rudd et al., 2023). Together, these results suggest that policies should aim to reduce economic disadvantage and increase accessibility of mental health care for those experiencing poverty and/or living in the most deprived areas to tackle disparities in child and adolescent mental health. These policies could be aimed especially at families with younger children given association between adversity in early life and later developmental disadvantage, as shown in this thesis.

While the deficits on an EC task were small in magnitude due to the time interval assessed, the results have important public health and clinical implications. The findings highlight that EC is a transdiagnostic mechanism which confers risk for total behavioural difficulties. This contributes to an extant literature that posits executive control as a target of intervention (Zelazo, 2020), given its well-documented associations with school readiness,

quality of life, and health (Diamond & Ling, 2016; Moffitt et al., 2011; Morrison et al., 2010). In addition, interventions targeted at executive functioning have proven efficacious, especially when administered to those with low levels of executive functioning (Diamond & Lee, 2011), and are most successful when simultaneously addressing social, emotional, and physical needs (Diamond & Ling, 2016). Besides the clinical utility of executive control as a target for intervention, the findings suggest the importance of investment to reduce socioeconomic disparities, childhood adversity, and associated deficits in executive functioning, which will help prevent long-term consequences on overall quality of life.

4.3 Limitations

While this study provides insights into dimensional models of both psychopathology and childhood adversity, several limitations should be noted. With regards to Study 1, an important limitation is the reliance on the SDQ alone to investigate hierarchical models of psychopathology, which is best achieved when measuring a wide range of psychopathology and dimensional phenotypes (e.g., thought disorders) (Caspi et al., 2024). This is also a limitation of the larger literature on the structure of psychopathology, which commonly consists of samples not assessed for thought disorders or is assessed via a single instrument, such as the CBCL (Carragher et al., 2016; Hoffman et al., 2022; Patalay et al., 2015). While the GUiNZ study considered the use of the CBCL as it is often regarded the 'gold standard' for the assessment of child/adolescent psychopathology, the SDQ was ultimately utilized given that its freely available and shorter in length.

Another limitation concerns the use of only parent reported SDQ data, which may have biased results given its use as a screener for psychopathology. Moreover, prior work has demonstrated discrepancies between different informants of a child's behaviour, showing that children are better able to report on their internalizing dimensions (Johnston & Murray, 2003), and gender-related expectations of the type of behavioural problems a child might

have (Najman et al., 2001). As such, it is uncertain if the factor structures identified in the current study would emerge with teacher or self-reported data. This study used data from a population cohort study, and as such may not be generalizable to clinic-referred samples. Further, preliminary research has shown that the inclusion of undiagnosed cases influences the structure of psychopathology, biasing models towards a unidimensional structure (Watts et al., 2021). Lastly, testing for model invariance was beyond the scope of the present thesis, and as such results may not be generalizable across age, ethnicity, and gender.

One caveat to our findings concerns the use of dichotomous indicators, using a variety of cut-off thresholds, to construct composite scores for each adversity dimension. Given this approach follows a cumulative risk framework, it may have hindered the ability to examine more ambiguous forms of adversity that have not met the specified threshold. While this may be deemed a limitation, the present study used thresholds indicative of clinically significant adversity where possible (i.e., maternal depression) and "baked in" a degree of severity for other indicators using a 90th percentile threshold. Therefore, this approach may be beneficial to avoid over-pathologizing participants in a privileged sample. Further, it helps circumvent issues with other methodologies such as factor analysis or standardization, which may introduce limitations regarding the co-occurrence between different types of adversity or statistically biasing an individual with low adversity exposure to have higher levels of adversity within a privileged sample (Berman et al., 2022).

The present study also used the Luria Handclap Task as a measure for executive control, which represents only one domain of executive functioning compared to the broader effects hypothesized by the DMAP (McLaughlin & Sheridan, 2014). While the results show utility in EC as a transdiagnostic mechanism for intervention, they are limited given the inability to test the effects of different adversity dimensions on a more cohesive assessment of cognitive ability. As the Luria Handclap Task measures 'cool' executive functioning skills,

the examination of ‘hot’ skills, which are activated in emotionally and motivationally significant situations (Zelazo & Carson, 2012), may lead to different insights. Furthermore, the present thesis did not examine mediators of threat, such as those related to social information processing as hypothesized by DMAP (McLaughlin & Sheridan, 2014). Lastly, the present study did not assess severe experiences of adversity such as sexual abuse.

Lastly, the findings are limited given the use of list-wise deletion as a strategy to handle missing data. This may have biased results and thus limit the ability to generalize conclusions. However, a missing data analysis showed that those with missing data tended to have lower socioeconomic status, levels of parental education, and live in areas with greater deprivation. Different imputation strategies to handle missing values may have revealed different insights, however, it is a possibility that the present strategy to handle missing data had a conservative effect and underestimated results.

4.4 Strengths

Despite the limitations discussed above, this study maintains several strengths. Firstly, although the GUiNZ was not designed to capture the unique effects of threat, deprivation, and unpredictability, the wealth of data collected has allowed the ability to construct composite scores for distinct adversity dimensions as in previous studies (Phillips et al., 2023; McGinnis et al., 2022). Second, these data were collected within the child’s first 4.5 years of life, which allows hypotheses to be tested based on a sensitive window in early childhood. As such, the results are developmentally specific, which limit potential recency effects (Dunn et al., 2018). Third, all adversity dimensions were constructed using prospective mother-reported data. Prior work has demonstrated low agreement between proxy and self-reported child maltreatment (Skar et al., 2021), as well as discrepancies between prospective parent reports and retrospective child reports, which showed two non-overlapping groups of participants but with overall similar rates of maltreatment and associations with psychopathology (Newbury

et al., 2018). This is a strength in the present study as prospective parent reports may capture those that have experienced childhood adversity but are likely to underreport these experiences at a later age (Newbury et al., 2018). Further, the results of the present thesis are robust to underreporting of childhood adversity by parents due to fear or disclosure or social desirability bias (Newbury et al., 2018).

Other strengths of this study are reflective of the multidisciplinary approach of GUiNZ. Core predictions were tested using a large sample size with sufficient ethnic diversity to be reflective of the New Zealand population. This allowed the investigation of the factor structure and reliability of the total difficulties score and its association with external correlates in a larger sample than previous studies (Keller & Langemeyer, 2019; Kobor et al., 2013; Ortuno-Sierra et al., 2015). Further, due to the multidisciplinary approach of GUiNZ, a wide range of indicators were used to construct adversity dimensions, consisting of distal and proximal indicators of childhood adversity, such as the NZDI and NZiDep, which provide a more cohesive assessment than other measures that do not consider consumption patterns (e.g., needs-to-income ratios) (Salmond et al., 2006).

4.5 Future Directions

Evaluating configural, metric, and scalar invariance was beyond the scope of the present thesis but presents a pivotal next step for future research. Given the wealth of data collection and sociodemographic diversity in the GUiNZ study across childhood and adolescence, future research could also evaluate longitudinal invariance across different developmental periods while also investigating homotypic or heterotypic continuity across different dimensions of psychopathology over time. This research would also be able to account for multiple informants of mental health, given the availability of self and parent reported SDQ data at age 12 years (Walker et al., 2023). Furthermore, future studies would also be able to incorporate other measures into their analyses, such as the 10-item Center for

Epidemiological Studies Depression Scale for Children (CES-DC) and the 8-item Patient Reported Outcomes Measurement Information System (PROMIS) Anxiety short form scale (Walker et al., 2013), which are available at 8-, 10-, and 12-year DCW. Data aggregation using item harmonization strategies may be a useful tool to integrate different mental health questionnaires, as evidenced by a study that harmonized the CBCL and SDQ (Hoffman et al., 2024). Lastly, including questions regarding confirmed psychiatric diagnoses in future GUiNZ DCW's may provide the opportunity to test for discriminant validity. This would also allow for the comparison of models with diagnosed case versus undiagnosed cases, which would shed further light on the p-factor debate (Watts et al., 2021).

Other directions for future research involve extending the results of the present thesis to adolescence as well as using more cohesive assessments of executive functioning. At age 8 years, the National Institutes of Health (NIH) Toolbox Cognition Battery was administered to participants (Neumann et al., 2020), which presents an opportunity to test for a broader measure of cognitive ability as a mediator between distinct types of early adversity and adolescent behavioural difficulties. This could highlight further specificity and strength of associations given the wider time interval investigated and that adolescence is a period characterized by the onset of many psychiatric disorders (Girolamo et al., 2012; Paus et al., 2008). Additionally, using a cohesive battery for the assessment of executive functioning may shed light on the compensatory versus attenuating effects of unpredictability on cognitive ability (Herry et al., 2007; Fields et al., 2021). Extending the time interval of this study would also allow the exploration of proximal and distal mechanisms specified in life history theory, such as pubertal effects (Colich et al., 2020).

A key strength of the GUiNZ study is the extensive data collection occurring antenatally and perinatally (Morton et al., 2014). While the study did not control for other types of adversity not specified in the DMAP and Harshness-Unpredictability framework,

future research should evaluate the effects of different adversity dimensions above perinatal predictors known to influence offspring mental health (D'Souza et al., 2015; Slykerman et al., 2015; Theunissen et al., 2022). Another direction of future research that has been highlighted in the literature is the importance of integrating the role of sleep in future investigations (Koss & Gunnar, 2018). Prior work has shown associations between poor sleep and developmental outcomes (Nixon et al., 2008); however, the relationship between sleep and mental health problems is equivocal (Lovato & Gradisar, 2014; Slykerman et al., 2019; Slykerman et al., 2020). Future directions of research could evaluate the utility in conceptualising sleep as an indicator of deprivation and the indirect effects it could have on later mental health difficulties via deficits in cognitive ability. One meta-analysis has shown associations between sleep duration and lower cognitive ability in children (Short et al., 2018). Conceptualising sleep deprivation as an absent expected input required for healthy development, and as such, an indicator of childhood adversity, may be worth exploring in future research.

Different operationalizations of unpredictability could also further our current findings. In a recent study, Farkas et al. (2024) show the utility in classifying unpredictable experiences based on the timescale they are likely to influence. They show that models including stochasticity (variability in days/weeks) and volatility (variability in months/years) better explain associations with internalizing and externalizing symptoms compared to a single measure of unpredictability (Farkas et al., 2024). Their novel findings show that volatility was associated more strongly with externalizing behaviours, whereas greater exposure to stochasticity have subtler effects on psychopathology that require further investigation. GUiNZ data presents a unique opportunity to further these findings given the wealth of prospective longitudinal data, especially in early childhood.

4.6 Summary and Conclusions

The findings of the present thesis contribute knowledge to developmental psychopathology, specifically advancements in dimensional views of both psychopathology and childhood adversity. Several structural models of psychopathology were tested on the total difficulties score of the parent-reported SDQ. In support of previous findings, the present study shows strong correlations between internalizing and externalizing symptoms (Lahey et al., 2004), as well as correlations between specific subscales within the broader internalizing and externalizing dimensions of the SDQ (Goodman et al., 2010). Further, the current study replicates previous findings that show superior fit of bi-factor models, but shed light on the anomalous findings that these models yield. Results also demonstrate that childhood behavioural problems are best described by a bi-factor S-1 model, where a general factor is defined a priori, thus leading to general and specific factors that are easily interpretable. Consistent with the literature, external validation analyses show that a general factor referenced by conduct problems is strongly linked with poorer prosocial behaviour. These results point to important transdiagnostic features that could explain the clinical heterogeneity of conduct disorders, and also show how the use of both bi-factor S-1 models and the SDQ could be leveraged to further the p-factor literature and examine the relationship between adversity and psychopathology with greater specificity.

This is the first study to use longitudinal data to demonstrate the utility in examining associations between multiple dimensions of adversity from different dimensional frameworks with later mental health, and the role of EC in this relationship. Results point to the importance in considering proximal mechanisms unique to distinct types of adverse experiences in early childhood, highlighting limitations in the stress-based approach as a universal mechanism underlying all forms of adversity. Overall, the findings show associations between various forms of early life adversity with TDS in middle childhood, and

the benefits of dimensional models in uncovering specific associations, such as the pathway between deprivation, EC, and TDS. This highlights the clinical utility of executive control as a potential target for intervention or prevention in child mental health difficulties. Policymakers should address these mental health disparities through greater accessibility to mental health resources for those in the most deprived areas, as well as greater investment in economic, physical, social, and human resources to target and reduce specific forms of adversity that are either distal or proximal to the child. Collectively, these policies may benefit child behavioural development and prevent the downstream effects of executive control on psychopathology, which if left unchecked, could exacerbate long-term effects on health and functioning across the lifespan.

Supplementary Materials

Table S1

Percentage missing and bivariate correlations between deprivation, threat, and unpredictability indicators in the main study sample.

Variable	Missing (% from main study sample)	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.
1. Bullying age 1	3%	1.0																		
2. Bullying age 4.5	2.5%	.235**	1.0																	
3. IPV	23.3%	.085**	.098**	1.0																
4. Harsh physical punishment	2.9%	.156**	.129**	.128**	1.0															
5. Emotional abuse	0%	.120**	.104**	.089**	.333**	1.0														
6. Material Hardship age 1	2.2%	.147**	.135**	.155**	.141**	.099**	1.0													

7. Material Hardship age 4.5	1.4%	.141**	.175**	.127**	.149**	.108**	.351**	1.0							
8. NZ deprivation index age 1	2.2%	.147**	.121**	.109**	.160**	.099**	.199**	.201**	1.0						
9. NZ deprivation index age 4.5	4.9%	.184**	.187**	.145**	.201**	.121**	.219**	.246**	.524**	1.0					
10. Cognitive stimulation age 1	2.2%	.060**	.080**	.078**	.106**	.118**	.096**	.084**	.074**	.100**	1.000				
11. Cognitive Stimulation age 4.5	1.4%	0.028	.043**	.041*	.045**	.075**	.083**	.077**	.041**	.063**	.157**	1.000			
12. Hours watched TV	0.3%	.110**	.086**	.081**	.154**	.086**	.106**	.096**	.168**	.196**	.110**	.048**	1.000		
13. Safe play area	1.6%	0.019	-0.008	0.028	.080**	.050**	.036*	.042**	.039**	.043**	.043**	.042**	0.028	1.000	
14. Number of household moves	0%	.100**	.088**	.035*	.038*	.046**	.126**	.122**	.079**	.114**	.064**	.034*	.070**	.035*	1.000
15. Number of caregiving arrangements	.1%	-.043**	-0.021	-0.010	-.036*	-.035*	-.066**	-.076**	-.067**	-.084**	-.040**	-0.007	-.074**	-0.027	0.007	1.000

16. Change in maternal relationship status	.1%	.124**	.135**	.186**	.067**	.045**	.216**	.216**	.128**	.168**	.082**	0.010	.061**	0.016	.191**	-0.024	1.000		
17. Maternal depression age 1	2.2%	.081**	.086**	.139**	.058**	.057**	.150**	.098**	.081**	.078**	.049**	0.023	.065**	0.000	.058**	-0.022	.073**	1.000	
18. Maternal mental health age 4.5	1.9%	.112**	.141**	.111**	.087**	.091**	.199**	.172**	.078**	.090**	.062**	.034*	.070**	.035*	.066**	-.038**	.116**	.162**	1.000
19. Maternal drug use	2.2%	.043**	.055**	.130**	.038**	.048**	.149**	.125**	.074**	.076**	.078**	.034*	.058**	0.018	.134**	-0.008	.115**	.084**	.093**	1.000
20. Financial stress	.7%	.111**	.105**	.114**	.085**	.092**	.302**	.252**	.118**	.142**	.056**	.049**	.070**	.072**	.116**	0.007	.143**	.196**	.189**	.128**

Table S2

Interactions between sociodemographic covariates and adversity dimensions (threat, deprivation, and unpredictability).

	(Gender*Adversity Dimension)	(Ethnicity*Adversity Dimension)
Threat	$X^2(1) = 0.551$, $p = .458$	$X^2(1) = .042$, $p = .837$
Deprivation	$X^2(1) = 1.58$, $p = .209$	$X^2(1) = 0.885$, $p = .347$
Unpredictability	$X^2(1) = 0.62$, $p = .431$	$X^2(1) = 0.315$, $p = .575$

Table S3

Mediated multiple linear regression analysis (N = 4153) showing indirect effect of Cognitive Deprivation (X) on Total Behavioural Difficulties at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates, previous levels of behavioural difficulties, and other adversity dimensions.

Outcome	Predictor	B	SE	t	*p*	95% CI	R^2
Luria Handclap Task	Gender	-0.523	0.149	-3.498	.0005	(-0.815, -0.230)	0.0415
	Total Difficulties at 54 months	-0.137	0.018	-7.645	< .0001	(-0.172, -0.102)	
	Ethnicity (0 = NZ Euro)	-0.489	0.176	-2.785	.0054	(-0.833, -0.145)	
	Child age at 54 months	0.180	0.058	3.118	.0018	(0.067, 0.293)	
	Cognitive Deprivation	-0.197	0.100	-1.965	.0495	(-0.394, -0.001)	
	Threat	-0.183	0.100	-1.828	.0676	(-0.379, 0.013)	
	Unpredictability	-0.148	0.081	-1.823	.0683	(-0.306, 0.011)	
Total Difficulties at age 8 (Y)	Gender	0.890	0.136	6.561	< .0001	(0.624, 1.156)	0.2927
	Total Difficulties at 54 months	0.474	0.017	27.527	< .0001	(0.440, 0.508)	
	Ethnicity (0 = NZ Euro)	-0.452	0.157	-2.888	.0039	(-0.759, -0.145)	
	Child age at 54 months	0.027	0.054	0.495	.6208	(-0.079, 0.133)	
	Cognitive Deprivation (X)	0.170	0.089	1.914	.0557	(-.004, 0.344)	

Threat	0.317	0.095	3.354	.0008	(0.132, 0.503)
Unpredictability	0.478	0.077	6.242	< .0001	(0.328, 0.628)
Luria Handclap Task (M)	-0.085	0.015	-5.636	< .0001	(-0.115, -0.056)
Indirect Effect of X on Y	0.017	0.009	-	-	(0.0002, 0.0365)

Note. N = 4153. Unstandardized regression coefficients are reported.

Table S4

Mediated multiple linear regression analysis showing indirect effect of Material Deprivation (X) on Total Behavioural Difficulties at age 8 years (Y) through Luria Handclap Task (M), controlling for socio-demographic covariates, previous levels of behavioural difficulties, and other adversity dimensions.

Outcome	Predictor	B	SE	t	*p*	95% CI	R^2
Luria Handclap Task	Gender	-0.546	0.149	-3.661	.0003	(-0.838, -0.254)	.0446
	Total Difficulties at 54 months	-0.131	0.018	-7.273	< .0001	(-0.166, -0.096)	
	Ethnicity (0 = NZ Euro)	-0.346	0.182	-1.905	.0569	(-0.702, 0.010)	
	Child age at 54 months	0.197	0.058	3.420	.0006	(0.084, 0.310)	
	Material Deprivation	-0.333	0.084	-3.967	.0001	(-0.498, -0.168)	
	Threat	-0.137	0.101	-1.346	.1784	(-0.335, 0.062)	
	Unpredictability	-0.079	0.083	-0.950	.3422	(-0.242, 0.084)	
Total Difficulties at age 8 (Y)	Gender	0.905	0.136	6.658	< .0001	(0.639, 1.172)	
	Total Difficulties at 54 months	0.471	0.017	27.406	< .0001	(0.438, 0.505)	
	Ethnicity (0 = NZ Euro)	-0.528	0.158	-3.349	.0008	(-0.838, -0.219)	
	Child age at 54 months	0.016	0.054	0.299	.7652	(-0.089, 0.122)	
	Material Deprivation (X)	0.208	0.076	2.726	.0064	(0.059, 0.358)	
	Threat	0.293	0.096	3.044	.0023	(0.104, 0.482)	

Unpredictability	0.438	0.077	5.711	< .0001	(0.287, 0.588)
Luria Handclap Task (M)	-0.084	0.015	-5.512	< .0001	(-0.113, -0.054)
Indirect Effect of X on Y	0.028	.009	-	-	(0.013, 0.047)

Note. N = 4153. Unstandardized regression coefficients are reported.

References

Aarø, L. E., Davids, E. L., Mathews, C., Wubs, A. G., Smith, O. R., & de Vries, P. J. (2022). Internalizing problems, externalizing problems, and prosocial behavior-three dimensions of the strengths and difficulties questionnaire (SDQ): A study among South African adolescents. *Scandinavian Journal of Psychology*, *63*(4), 415–425.

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs: General and Applied*, *80*(7), 1.

Achenbach, T. M., Becker, A., Döpfner, M., Heiervang, E., Roessner, V., Steinhausen, H.-C., & Rothenberger, A. (2008). Multicultural assessment of child and adolescent psychopathology with ASEBA and SDQ instruments: Research findings, applications, and future directions. *Journal of Child Psychology and Psychiatry*, *49*(3), 251–275.

Achenbach, T. M., & Edelbrock, C. (1991). Child behavior checklist. *Burlington (Vt)*, *7*, 371–392.

Afzali, M. H., Sunderland, M., Carragher, N., & Conrod, P. (2018). The Structure of Psychopathology in Early Adolescence: Study of a Canadian Sample: La structure de la psychopathologie au début de l'adolescence: étude d'un échantillon canadien. *The Canadian Journal of Psychiatry*, *63*(4), 223–230. https://doi.org/10.1177/0706743717737032

Ahmad, S., Peterson, E. R., Waldie, K. E., & Morton, S. M. (2019). Development of an index of socio-emotional competence for preschool children in the growing up in New Zealand study. *Frontiers in Education*, *4*, 2. https://www.frontiersin.org/articles/10.3389/feduc.2019.00002/full

al'Absi, M., Ginty, A. T., & Lovallo, W. R. (2021). Neurobiological mechanisms of early life adversity, blunted stress reactivity and risk for addiction. *Neuropharmacology*, *188*, 108519. https://doi.org/10.1016/j.neuropharm.2021.108519

Albott, C. S., Forbes, M. K., & Anker, J. J. (2018). Association of childhood adversity with differential susceptibility of transdiagnostic psychopathology to environmental stress in adulthood. *JAMA Network Open*, *1*(7), e185354–e185354.

Aldao, A., Gee, D. G., De Los Reyes, A., & Seager, I. (2016). Emotion regulation as a transdiagnostic factor in the development of internalizing and externalizing psychopathology: Current and future directions. *Development and Psychopathology*, *28*(4pt1), 927–946.

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy*, *48*(10), 974–983.

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review*, *30*(2), 217–237.

Allegrini, A. G., Cheesman, R., Rimfeld, K., Selzam, S., Pingault, J.-B., Eley, T. C., & Plomin, R. (2020). The p factor: Genetic analyses support a general dimension of psychopathology in childhood and adolescence. *Journal of Child Psychology and Psychiatry*, *61*(1), 30–39.

Álvarez-Voces, M., & Romero, E. (2024). *Gender differences in the development of children's conduct problems: A four-year longitudinal study*. https://europepmc.org/article/ppr/ppr788844

André, J.-B., & Rousset, F. (2020). Does extrinsic mortality accelerate the pace of life? A bare-bones approach. *Evolution and Human Behavior*, *41*(6), 486–492.

Andrews, K., Atkinson, L., Harris, M., & Gonzalez, A. (2021). Examining the effects of household chaos on child executive functions: A meta-analysis. *Psychological Bulletin*, *147*(1), 16.

Andrews, K., Dunn, J. R., Prime, H., Duku, E., Atkinson, L., Tiwari, A., & Gonzalez, A. (2021). Effects of household chaos and parental responsiveness on child executive functions: A novel, multi-method approach. *BMC Psychology*, *9*(1), 147. https://doi.org/10.1186/s40359-021-00651-1

Andrews, K., Khoury, J. E., Tiwari, A., Kirupaharan, S., & Gonzalez, A. (2023). Maternal History of Child Maltreatment and Household Chaos: Examining the Mediating Role of Maternal and Child Psychopathology. *Child Maltreatment, 28*(2), 221–231. https://doi.org/10.1177/10775595221081795

Angelakis, I., Austin, J. L., & Gooding, P. (2020). Association of childhood maltreatment with suicide behaviors among young people: A systematic review and meta-analysis. *JAMA Network Open*, *3*(8), e2012563–e2012563.

Angst, J., Gamma, A., Rössler, W., Ajdacic, V., & Klein, D. N. (2011). Childhood adversity and chronicity of mood disorders. *European Archives of Psychiatry and Clinical Neuroscience*, *261*, 21–27.

Barajas-Gonzalez, R. G., Ayón, C., Brabeck, K., Rojas-Flores, L., & Valdez, C. R. (2021). An ecological expansion of the adverse childhood experiences (ACEs) framework to include threat and deprivation associated with US immigration policies and enforcement practices: An examination of the Latinx immigrant experience. *Social Science & Medicine*, *282*, 114126.

Barker, D. J. P. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine, 261*(5), 412–417. https://doi.org/10.1111/j.1365-2796.2007.01809.x

Barker, D. J., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *The Lancet*, *327*(8489), 1077–1081.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*(6), 1173.

Beauchaine, T. P., & Tackett, J. L. (2020). Irritability as a transdiagnostic vulnerability trait: Current issues and future directions. *Behavior Therapy*, *51*(2), 350–364.

Beauchaine, T. P., & Zisner, A. (2017). Motivation, emotion regulation, and the latent structure of psychopathology: An integrative and convergent historical perspective. *International Journal of Psychophysiology*, *119*, 108–118.

Beauchaine, T. P., Zisner, A. R., & Sauder, C. L. (2017). Trait Impulsivity and the Externalizing Spectrum. *Annual Review of Clinical Psychology*, *13*(1), 343–368. https://doi.org/10.1146/annurev-clinpsy-021815-093253

Belsky, J., Schlomer, G. L., & Ellis, B. J. (2012). Beyond cumulative risk: Distinguishing harshness and unpredictability as determinants of parenting and early life history strategy. *Developmental Psychology*, *48*(3), 662.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*(4), 647–670.

Ben-Shlomo, Y., & Kuh, D. (2002). A life course approach to chronic disease epidemiology: Conceptual models, empirical challenges and interdisciplinary perspectives. In *International journal of epidemiology* (Vol. 31, Issue 2, pp. 285–293). Oxford University Press.

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society*, Series B, 57, 289–300.

Berens, A. E., Jensen, S. K., & Nelson, C. A. (2017a). Biological embedding of childhood adversity: From physiological mechanisms to clinical implications. *BMC Medicine*, *15*(1), 1–12.

Berman, I. S., McLaughlin, K. A., Tottenham, N., Godfrey, K., Seeman, T., Loucks, E., Suomi, S., Danese, A., & Sheridan, M. A. (2022). Measuring early life adversity: A dimensional approach. *Development and Psychopathology*, *34*(2), 499–511.

Bird, A. L., Carr, P. E. A., Reese, E., & Morton, S. M. (2016). Policy translation for early childhood education and care: The Growing Up in New Zealand approach. *International Journal of Child Care and Education Policy*, *10*, 1–18.

Bird, A. L., Grant, C. C., Bandara, D. K., Mohal, J., Atatoa-Carr, P. E., Wise, M. R., Inskip, H., Miyahara, M., & Morton, S. M. (2017). Maternal health in pregnancy and associations with adverse birth outcomes: Evidence from growing up in New Zealand. *Australian and New Zealand Journal of Obstetrics and Gynaecology*, *57*(1), 16–24.

Bird, A., Reese, E., Schaughency, E., Waldie, K., Atatoa-Carr, P., Morton, S., & Grant, C. (2024). Talking, praising and teaching: How parent interaction during a learning task relates to children's early learning. *Early Childhood Research Quarterly*, *66*, 255–268.

Bolten, M., Nast, I., Skrundz, M., Stadler, C., Hellhammer, D. H., & Meinlschmidt, G. (2013). Prenatal programming of emotion regulation: Neonatal reactivity as a differential susceptibility factor moderating the outcome of prenatal cortisol levels. *Journal of Psychosomatic Research*, *75*(4), 351–357.

Bolton, J. L., Molet, J., Ivy, A., & Baram, T. Z. (2017). New insights into early-life stress and behavioral outcomes. *Current Opinion in Behavioral Sciences*, *14*, 133–139.

Bonifay, W. E., Reise, S. P., Scheines, R., & Meijer, R. R. (2015). When Are Multidimensional Data Unidimensional Enough for Structural Equation Modeling? An Evaluation of the DETECT Multidimensionality Index. *Structural Equation Modeling: A Multidisciplinary Journal*, *22*(4), 504–516. https://doi.org/10.1080/10705511.2014.938596

Bonifay, W., Lane, S. P., & Reise, S. P. (2017). Three Concerns With Applying a Bifactor Model as a Structure of Psychopathology. *Clinical Psychological Science*, *5*(1), 184–186. https://doi.org/10.1177/2167702616657069

Bornovalova, M. A., Choate, A. M., Fatimah, H., Petersen, K. J., & Wiernik, B. M. (2020). Appropriate use of bifactor analysis in psychopathology research: Appreciating benefits and limitations. *Biological Psychiatry*, *88*(1), 18–27.

Brieant, A., Clinchard, C., Deater-Deckard, K., Lee, J., King-Casas, B., & Kim-Spoon, J. (2022). Differential Associations of Adversity Profiles with Adolescent Cognitive Control and Psychopathology. *Research on Child and Adolescent Psychopathology*, 1–14.

Brieant, A. E., Sisk, L. M., & Gee, D. G. (2021). Associations among negative life events, changes in cortico-limbic connectivity, and psychopathology in the ABCD Study. *Developmental Cognitive Neuroscience*, *52*, 101022.

Briggs-Gowan, M. J., Grasso, D., Bar-Haim, Y., Voss, J., McCarthy, K. J., Pine, D. S., & Wakschlag, L. S. (2016). Attention bias in the developmental unfolding of post-traumatic stress symptoms in young children at risk. *Journal of Child Psychology and Psychiatry*, *57*(9), 1083–1091. https://doi.org/10.1111/jcpp.12577

Brody, G. H., Yu, T., Chen, E., Miller, G. E., Kogan, S. M., & Beach, S. R. (2013). Is resilience only skin deep? Rural African Americans' socioeconomic status–related risk and competence in preadolescence and psychological adjustment and allostatic load at age 19. *Psychological Science*, *24*(7), 1285–1293.

Brown, J. B., Lent, B., Brett, P. J., Sas, G., & Pederson, L. L. (1996). Development of the Woman Abuse Screening Tool for use in family practice. *Family Medicine-Kansas City-*, *28*, 422–428.

Buckley, J., Peterson, E. R., Underwood, L., D'Souza, S., Morton, S. M., & Waldie, K. E. (2020). Socio-demographic and maternal health indicators of inhibitory control in preschool age children: Evidence from Growing Up in New Zealand. *Longitudinal and Life Course Studies*, *11*(2), 181–201.

Bürgin, D., O'Donovan, A., d'Huart, D., Di Gallo, A., Eckert, A., Fegert, J., Schmeck, K., Schmid, M., & Boonmann, C. (2019). Adverse childhood experiences and telomere length a look into the heterogeneity of findings—A narrative review. *Frontiers in Neuroscience*, *13*, 490.

Burns, G. L., Geiser, C., Servera, M., Becker, S. P., & Beauchaine, T. P. (2020a). Application of the Bifactor S – 1 Model to Multisource Ratings of ADHD/ODD Symptoms: An Appropriate Bifactor Model for Symptom Ratings. *Journal of Abnormal Child Psychology*, *48*(7), 881–894. https://doi.org/10.1007/s10802-019-00608-4

Burt, S. A. (2009). Rethinking environmental contributions to child and adolescent psychopathology: A meta-analysis of shared environmental influences. *Psychological Bulletin*, *135*(4), 608.

Burt, S. A. (2013). Do etiological influences on aggression overlap with those on rule breaking? A meta-analysis. *Psychological Medicine*, *43*(9), 1801–1812.

Bush, N. R., Obradović, J., Adler, N., & Boyce, W. T. (2011). Kindergarten stressors and cumulative adrenocortical activation: The "first straws" of allostatic load? *Development and Psychopathology*, *23*(4), 1089–1106.

Butler, O., Yang, X.-F., Laube, C., Kühn, S., & Immordino-Yang, M. H. (2018). Community violence exposure correlates with smaller gray matter volume and lower IQ in urban adolescents. *Human Brain Mapping*, *39*(5), 2088–2097.

Caci, H., Morin, A. J. S., & Tran, A. (2015). Investigation of a bifactor model of the Strengths and Difficulties Questionnaire. *European Child & Adolescent Psychiatry*, *24*(10), 1291–1301. https://doi.org/10.1007/s00787-015-0679-3

Campbell, O. L., Bann, D., & Patalay, P. (2021). The gender gap in adolescent mental health: A cross-national investigation of 566,829 adolescents across 73 countries. *SSM-Population Health*, *13*, 100742.

Caro-Cañizares, I., Serrano-Drozdowskyj, E., Pfang, B., Baca-García, E., & Carballo, J. J. (2020). SDQ Dysregulation Profile and Its Relation to the Severity of Psychopathology and Psychosocial Functioning in a Sample of Children and Adolescents With ADHD. *Journal of Attention Disorders*, *24*(11), 1557–1564. https://doi.org/10.1177/1087054717691829

Carozza, S., Akarca, D., & Astle, D. (2023). The adaptive stochasticity hypothesis: Modelling equifinality, multifinality and adaptation to adversity. *bioRxiv*, 2023–05.

Carragher, N., Teesson, M., Sunderland, M., Newton, N. C., Krueger, R. F., Conrod, P. J., Barrett, E. L., Champion, K. E., Nair, N. K., & Slade, T. (2016). The structure of adolescent psychopathology: A symptom-level analysis. *Psychological Medicine*, *46*(5), 981–994.

Casey, B. J., Craddock, N., Cuthbert, B. N., Hyman, S. E., Lee, F. S., & Ressler, K. J. (2013). DSM-5 and RDoC: Progress in psychiatry research? *Nature Reviews Neuroscience*, *14*(11), 810–814.

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., & Poulton, R. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science*, *2*(2), 119–137.

Caspi, A., Houts, R. M., Fisher, H. L., Danese, A., & Moffitt, T. E. (2024). The General Factor of Psychopathology (p): Choosing Among Competing Models and Interpreting p. *Clinical Psychological Science*, *12*(1), 53–82. https://doi.org/10.1177/21677026221147872

Caspi, A., & Moffitt, T. E. (2018). All for one and one for all: Mental disorders in one dimension. *American Journal of Psychiatry*, *175*(9), 831–844.

Castellanos-Ryan, N., Brière, F. N., O'Leary-Barrett, M., Banaschewski, T., Bokde, A., Bromberg, U., Büchel, C., Flor, H., Frouin, V., & Gallinat, J. (2016). The structure of psychopathology in adolescence and its common personality and cognitive correlates. *Journal of Abnormal Psychology*, *125*(8), 1039.

Cha, J. E., Waldie, K. E., Neumann, D., Smith, A., & Walker, C. G. (2022). *Psychometric properties and factor structure of the Center for Epidemiologic Studies Depression scale 10-item short form (CES-D-10) in aotearoa New Zealand children*.

Chahal, R., Miller, J. G., Yuan, J. P., Buthmann, J. L., & Gotlib, I. H. (2022). An exploration of dimensions of early adversity and the development of functional brain network connectivity during adolescence: Implications for trajectories of internalizing symptoms. *Development and Psychopathology*, *34*(2), 557–571.

Chang, L., Lu, H. J., Lansford, J. E., Skinner, A. T., Bornstein, M. H., Steinberg, L., Dodge, K. A., Chen, B. B., Tian, Q., & Bacchini, D. (2019). Environmental harshness and unpredictability, life history, and social and academic behavior of adolescents in nine countries. *Developmental Psychology*, *55*(4), 890.

Changeux, J.-P., & Danchin, A. (1976). Selective stabilisation of developing synapses as a mechanism for the specification of neuronal networks. *Nature*, *264*(5588), 705–712.

Chen, C., Lu, Y., Lundström, S., Larsson, H., Lichtenstein, P., & Pettersson, E. (2022). Associations between psychiatric polygenic risk scores and general and specific psychopathology symptoms in childhood and adolescence between and within dizygotic twin pairs. *Journal of Child Psychology and Psychiatry*, *63*(12), 1513–1522. https://doi.org/10.1111/jcpp.13605

Chen, X., Zeng, C., Gong, C., Zhang, L., Wan, Y., Tao, F., & Sun, Y. (2019). Associations between early life parent-child separation and shortened telomere length and psychopathological outcomes during adolescence. *Psychoneuroendocrinology*, *103*, 195–202.

Choate, A. M., Bornovalova, M. A., Hipwell, A. E., Chung, T., & Stepp, S. D. (2022). The general psychopathology factor (p) from adolescence to adulthood: Exploring the developmental trajectories of p using a multi-method approach. *Development and Psychopathology*, 1–19.

Cisler, J. M., Esbensen, K., Sellnow, K., Ross, M., Weaver, S., Sartin-Tarm, A., Herringa, R. J., & Kilts, C. D. (2019). Differential roles of the salience network during prediction error encoding and facial emotion processing among female adolescent assault victims. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *4*(4), 371–380.

Cisler, J. M., & Koster, E. H. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review*, *30*(2), 203–216.

Clark, C., Caldwell, T., Power, C., & Stansfeld, S. A. (2010). Does the influence of childhood adversity on psychopathology persist across the lifecourse? A 45-year prospective epidemiologic study. *Annals of Epidemiology*, *20*(5), 385–394.

Clark, D. A., Hicks, B. M., Angstadt, M., Rutherford, S., Taxali, A., Hyde, L., Weigard, A. S., Heitzeg, M. M., & Sripada, C. (2021). The general factor of psychopathology in the Adolescent Brain Cognitive Development (ABCD) Study: A comparison of alternative modeling approaches. *Clinical Psychological Science*, *9*(2), 169–182.

Colich, N. L., Platt, J. M., Keyes, K. M., Sumner, J. A., Allen, N. B., & McLaughlin, K. A. (2020). Earlier age at menarche as a transdiagnostic mechanism linking childhood trauma with multiple forms of psychopathology in adolescent girls. *Psychological Medicine*, *50*(7), 1090–1098.

Colich, N. L., Rosen, M. L., Williams, E. S., & McLaughlin, K. A. (2020). Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. *Psychological Bulletin*, *146*(9), 721.

Collishaw, S., Goodman, R., Ford, T., Rabe-Hesketh, S., & Pickles, A. (2009). How far are associations between child, family and community factors and child psychopathology informant-specific and informant-general? *Journal of Child Psychology and Psychiatry*, *50*(5), 571–580.

Compton, A. B., Panlilio, C. C., & Humphreys, K. L. (2023). What's the matter with ACEs? Recommendations for considering early adversity in educational contexts. *Child Abuse & Neglect*, 106073.

Conradt, E., Adkins, D. E., Crowell, S. E., Raby, K. L., Diamond, L. M., & Ellis, B. (2018). Incorporating epigenetic mechanisms to advance fetal programming theories. *Development and Psychopathology*, *30*(3), 807–824.

Constantinou, M., & Fonagy, P. (2019). *Evaluating bifactor models of psychopathology using model-based reliability indices*. https://psyarxiv.com/6tf7j/download?format=pdf

Conway, C. C., Mansolf, M., & Reise, S. P. (2019). Ecological validity of a quantitative classification system for mental illness in treatment-seeking adults. *Psychological Assessment*, *31*(6), 730.

Corkin, M. T., Peterson, E. R., Henderson, A. M. E., Bird, A. L., Waldie, K. E., Reese, E., & Morton, S. M. B. (2021). The Predictors of Screen Time at Two Years in a Large Nationally Diverse Cohort. *Journal of Child and Family Studies*, *30*(8), 2076–2096. https://doi.org/10.1007/s10826-021-01985-5

Cosgrove, V. E., Rhee, S. H., Gelhorn, H. L., Boeldt, D., Corley, R. C., Ehringer, M. A., Young, S. E., & Hewitt, J. K. (2011). Structure and Etiology of Co-occurring Internalizing and Externalizing Disorders in Adolescents. *Journal of Abnormal Child Psychology*, *39*(1), 109–123. https://doi.org/10.1007/s10802-010-9444-8

Cox, J. L., Holden, J. M., & Sagovsky, R. (1987). Detection of postnatal depression: Development of the 10-item Edinburgh Postnatal Depression Scale. *The British Journal of Psychiatry*, *150*(6), 782–786.

Cronbach, L. J. (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, *16*(3), 297–334. https://doi.org/10.1007/BF02310555

Crystal, D. S., Ostrander, R., Chen, R. S., & August, G. J. (2001). Multimethod assessment of psychopathology among DSM-IV subtypes of children with attention-deficit/hyperactivity disorder: Self-, parent, and teacher reports. *Journal of Abnormal Child Psychology*, *29*, 189–205.

Danese, A. (2018). Commentary: Biological embedding of childhood adversity: where do we go from here? A reflection on Koss and Gunnar (2018). *Journal of Child Psychology and Psychiatry*, *59*(4), 347–349.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior*, *106*(1), 29–39.

Danese, A., McLaughlin, K. A., Samara, M., & Stover, C. S. (2020). Psychopathology in children exposed to trauma: Detection and intervention needed to reduce downstream burden. *BMJ*, *371*. https://www.bmj.com/content/371/bmj.m3073.abstract

Davies, L., Webber, A., & Timmins, J. (2022). The Nature of Disadvantage. Faced by Children in New Zealand: Implications for policy and service provision. *Policy Quarterly*, *18*(3), 38–43.

Davis, E. P., Stout, S. A., Molet, J., Vegetabile, B., Glynn, L. M., Sandman, C. A., Heins, K., Stern, H., & Baram, T. Z. (2017). Exposure to unpredictable maternal sensory signals influences cognitive development across species. *Proceedings of the National Academy of Sciences*, *114*(39), 10390–10395. https://doi.org/10.1073/pnas.1703444114

De Girolamo, G., Dagani, J., Purcell, R., Cocchi, A., & McGorry, P. D. (2012). Age of onset of mental disorders and use of mental health services: Needs, opportunities and obstacles. *Epidemiology and Psychiatric Sciences*, *21*(1), 47–57.

Deater-Deckard, K., Mullineaux, P. Y., Beekman, C., Petrill, S. A., Schatschneider, C., & Thompson, L. A. (2009). Conduct problems, IQ, and household chaos: A longitudinal multi-informant study. *Journal of Child Psychology and Psychiatry*, *50*(10), 1301–1308. https://doi.org/10.1111/j.1469-7610.2009.02108.x

Del Giudice, M. (2018). *Evolutionary psychopathology: A unified approach*. Oxford University Press.

Del Giudice, M. (2020). Rethinking the fast-slow continuum of individual differences. *Evolution and Human Behavior*, *41*(6), 536–549.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*(7), 1562–1592.

Del Giudice, M., Hinnant, J. B., Ellis, B. J., & El-Sheikh, M. (2012). Adaptive patterns of stress responsivity: A preliminary investigation. *Developmental Psychology*, *48*(3), 775.

Dennison, M. J., Sheridan, M. A., Busso, D. S., Jenness, J. L., Peverill, M., Rosen, M. L., & McLaughlin, K. A. (2016). Neurobehavioral markers of resilience to depression amongst adolescents exposed to child abuse. *Journal of Abnormal Psychology*, *125*(8), 1201.

Deutz, M. H., Shi, Q., Vossen, H. G., Huijding, J., Prinzie, P., Deković, M., van Baar, A. L., & Woltering, S. (2018). Evaluation of the Strengths and Difficulties Questionnaire-Dysregulation Profile (SDQ-DP). *Psychological Assessment*, *30*(9), 1174.

Diamond, A., & Lee, K. (2011). Interventions Shown to Aid Executive Function Development in Children 4 to 12 Years Old. *Science*, *333*(6045), 959–964. https://doi.org/10.1126/science.1204529

Diamond, A., & Ling, D. S. (2016). Conclusions about interventions, programs, and approaches for improving executive functions that appear justified and those that, despite much hype, do not. *Developmental Cognitive Neuroscience*, *18*, 34–48.

Diamond, A., & Taylor, C. (1996). Development of an aspect of executive control: Development of the abilities to remember what I said and to ?Do as I say, not as I do? *Developmental Psychobiology*, *29*(4), 315–334. https://doi.org/10.1002/(SICI)1098-2302(199605)29:4<315::AID-DEV2>3.0.CO;2-T

Doan, S. N., Dich, N., & Evans, G. W. (2014). Childhood cumulative risk and later allostatic load: Mediating role of substance use. *Health Psychology*, *33*(11), 1402.

Doan, S. N., & Evans, G. W. (2020). Chaos and instability from birth to age three. *The Future of Children*, *30*(2), 93–114.

Dodge, K. A., Bates, J. E., & Pettit, G. S. (1990). Mechanisms in the Cycle of Violence. *Science*, *250*(4988), 1678–1683. https://doi.org/10.1126/science.2270481

Doom, J. R., Cicchetti, D., Rogosch, F. A., & Dackis, M. N. (2013). Child maltreatment and gender interactions as predictors of differential neuroendocrine profiles. *Psychoneuroendocrinology*, *38*(8), 1442–1454.

Doom, J. R., & Gunnar, M. R. (2013). Stress physiology and developmental psychopathology: Past, present, and future. *Development and Psychopathology*, *25*(4pt2), 1359–1373.

Doom, J. R., Vanzomeren-Dohm, A. A., & Simpson, J. A. (2016). Early unpredictability predicts increased adolescent externalizing behaviors and substance use: A life history perspective. *Development and Psychopathology*, *28*(4pt2), 1505–1516.

Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., Loo, C. M., & Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect*, *28*(7), 771–784.

Drury, S. S., Theall, K., Gleason, M. M., Smyke, A. T., De Vivo, I., Wong, J. Y. Y., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2012). Telomere length and early severe social deprivation: Linking early adversity and cellular aging. *Molecular Psychiatry*, *17*(7), 719–727.

D'Souza, S., Crawford, C. N., Buckley, J., Underwood, L., Peterson, E. R., Bird, A., Morton, S. M., & Waldie, K. E. (2019). Antenatal determinants of early childhood talking delay and behavioural difficulties. *Infant Behavior and Development*, *57*, 101388.

D'Souza, S., Thompson, J. M., Slykerman, R., Marlow, G., Wall, C., Murphy, R., Ferguson, L. R., Mitchell, E. A., & Waldie, K. E. (2016). Environmental and genetic determinants of

childhood depression: The roles of DAT1 and the antenatal environment. *Journal of Affective Disorders*, *197*, 151–158.

D'Souza, S., Underwood, L., Peterson, E. R., Morton, S. M., & Waldie, K. E. (2020). The association between persistence and change in early childhood behavioural problems and preschool cognitive outcomes. *Child Psychiatry & Human Development*, *51*, 416–426.

D'Souza, S., Waldie, K. E., Peterson, E. R., Underwood, L., & Morton, S. M. (2017). Psychometric properties and normative data for the preschool strengths and difficulties questionnaire in two-year-old children. *Journal of Abnormal Child Psychology*, *45*(2), 345–357.

D'Souza, S., Waldie, K. E., Peterson, E. R., Underwood, L., & Morton, S. M. B. (2019). Antenatal and Postnatal Determinants of Behavioural Difficulties in Early Childhood: Evidence from Growing Up in New Zealand. *Child Psychiatry & Human Development*, *50*(1), 45–60. https://doi.org/10.1007/s10578-018-0816-6

Du Pont, A., Rhee, S. H., Corley, R. P., Hewitt, J. K., & Friedman, N. P. (2018). Rumination and Psychopathology: Are Anger and Depressive Rumination Differentially Associated With Internalizing and Externalizing Psychopathology? *Clinical Psychological Science*, *6*(1), 18–31. https://doi.org/10.1177/2167702617720747

Duckworth, A. L., Quinn, P. D., & Tsukayama, E. (2012). What No Child Left Behind leaves behind: The roles of IQ and self-control in predicting standardized achievement test scores and report card grades. *Journal of Educational Psychology*, *104*(2), 439.

Dugdale, H. L., & Richardson, D. S. (2018). Heritability of telomere variation: It is all about the environment! *Philosophical Transactions of the Royal Society B: Biological Sciences*, *373*(1741), 20160450.

Dugré, J. R., & Potvin, S. (2022). Multiple developmental pathways underlying conduct problems: A multitrajectory framework. *Development and Psychopathology*, *34*(3), 1115–1124.

Dunn, E. C., Soare, T. W., Raffeld, M. R., Busso, D. S., Crawford, K. M., Davis, K. A., Fisher, V. A., Slopen, N., Smith, A. D., & Tiemeier, H. (2018). What life course theoretical models best explain the relationship between exposure to childhood adversity and psychopathology symptoms: Recency, accumulation, or sensitive periods? *Psychological Medicine*, *48*(15), 2562–2572.

Dunn, E. C., Soare, T. W., Zhu, Y., Simpkin, A. J., Suderman, M. J., Klengel, T., Smith, A. D., Ressler, K. J., & Relton, C. L. (2019). Sensitive periods for the effect of childhood adversity on DNA methylation: Results from a prospective, longitudinal study. *Biological Psychiatry*, *85*(10), 838–849.

Duprey, E. B., Handley, E. D., Russotti, J., Manly, J. T., & Cicchetti, D. (2023). A longitudinal examination of child maltreatment dimensions, emotion regulation, and comorbid psychopathology. *Research on Child and Adolescent Psychopathology*, *51*(1), 71–85.

Dwairy, M. A. (2008). Parental Inconsistency Versus Parental Authoritarianism: Associations with Symptoms of Psychological Disorders. *Journal of Youth and Adolescence*, *37*(5), 616–626. https://doi.org/10.1007/s10964-007-9169-3

Edmiston, E. E., Wang, F., Mazure, C. M., Guiney, J., Sinha, R., Mayes, L. C., & Blumberg, H. P. (2011). Corticostriatal-limbic gray matter morphology in adolescents with self-reported exposure to childhood maltreatment. *Archives of Pediatrics & Adolescent Medicine*, *165*(12), 1069–1077.

Eid, M., Geiser, C., Koch, T., & Heene, M. (2017). Anomalous results in G-factor models: Explanations and alternatives. *Psychological Methods*, *22*(3), 541.

Eisenbarth, H. (2017). Environmental Influences: The Special Case of Gender. In L. C. Centifanti & D. M. Williams (Eds.), *The Wiley Handbook of Developmental Psychopathology* (1st ed., pp. 335–342). Wiley. https://doi.org/10.1002/9781118554470.ch16

Ellis, B. J. (2023). *The hidden talents framework: Implications for science, policy, and practice*. University Press. https://doi.org/10.1017/9781009350051

Ellis, B. J., Abrams, L. S., Masten, A. S., Sternberg, R. J., Tottenham, N., & Frankenhuis, W. E. (2022). Hidden talents in harsh environments. *Development and Psychopathology*, *34*(1), 95–113.

Ellis, B. J., & Boyce, W. T. (2008). Biological sensitivity to context. *Current Directions in Psychological Science*, *17*(3), 183–187.

Ellis, B. J., & Del Giudice, M. (2014). Beyond allostatic load: Rethinking the role of stress in regulating human development. *Development and Psychopathology*, *26*(1), 1–20.

Ellis, B. J., & Del Giudice, M. (2019). Developmental adaptation to stress: An evolutionary perspective. *Annual Review of Psychology*, *70*, 111–139.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature*, *20*, 204–268.

Ellis, B. J., Sheridan, M. A., Belsky, J., & McLaughlin, K. A. (2022). Why and how does early adversity influence development? Toward an integrated model of dimensions of environmental experience. *Development and Psychopathology*, *34*(2), 447–471.

Esteves, K. C., Jones, C. W., Wade, M., Callerame, K., Smith, A. K., Theall, K. P., & Drury, S. S. (2020). Adverse childhood experiences: Implications for offspring telomere length and psychopathology. *American Journal of Psychiatry*, *177*(1), 47–57.

Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The Role of Chaos in Poverty and Children's Socioemotional Adjustment. *Psychological Science*, *16*(7), 560–565. https://doi.org/10.1111/j.0956-7976.2005.01575.x

Evans, G. W., Li, D., & Whipple, S. S. (2013). Cumulative risk and child development. *Psychological Bulletin*, *139*(6), 1342.

Farah, M. J., Betancourt, L., Shera, D. M., Savage, J. H., Giannetta, J. M., Brodsky, N. L., Malmud, E. K., & Hurt, H. (2008). Environmental stimulation, parental nurturance and cognitive development in humans. *Developmental Science*, *11*(5), 793–801.

Farah, M. J., Shera, D. M., Savage, J. H., Betancourt, L., Giannetta, J. M., Brodsky, N. L., Malmud, E. K., & Hurt, H. (2006). Childhood poverty: Specific associations with neurocognitive development. *Brain Research*, *1110*(1), 166–174.

Farkas, B. C., Baptista, A., Speranza, M., Wyart, V., & Jacquet, P. O. (2024). Specifying the timescale of early life unpredictability helps explain the development of internalising and externalising behaviours. *Scientific Reports*, *14*(1), 3563.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*(4), 245–258.

Fendrich, M., Weissman, M. M., & Warner, V. (1990). Screening for depressive disorder in children and adolescents: Validating the center for epidemiologic studees depression scale for children. *American Journal of Epidemiology*, *131*(3), 538–551.

Fergusson, D. M., Boden, J. M., & Horwood, L. J. (2008a). Developmental antecedents of interpartner violence in a New Zealand birth cohort. *Journal of Family Violence*, *23*, 737–753.

Fernández De La Cruz, L., Vidal-Ribas, P., Zahreddine, N., Mathiassen, B., Brøndbo, P. H., Simonoff, E., Goodman, R., & Stringaris, A. (2018). Should Clinicians Split or Lump Psychiatric Symptoms? The Structure of Psychopathology in Two Large Pediatric Clinical Samples from England and Norway. *Child Psychiatry & Human Development*, *49*(4), 607–620. https://doi.org/10.1007/s10578-017-0777-1

Fields, A., Bloom, P. A., VanTieghem, M., Harmon, C., Choy, T., Camacho, N. L., Gibson, L., Umbach, R., Heleniak, C., & Tottenham, N. (2021). Adaptation in the face of adversity: Decrements and enhancements in children's cognitive control behavior following early caregiving instability. *Developmental Science*, *24*(6), e13133. https://doi.org/10.1111/desc.13133

Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007). Poly-victimization: A neglected component in child victimization. *Child Abuse & Neglect*, *31*(1), 7–26.

Finlay, S., Roth, C., Zimsen, T., Bridson, T. L., Sarnyai, Z., & McDermott, B. (2022). Adverse childhood experiences and allostatic load: A systematic review. *Neuroscience & Biobehavioral Reviews*, *136*, 104605.

Finney, S. J., & DiStefano, C. (2006). Non-normal and categorical data in structural equation modeling. *Structural Equation Modeling: A Second Course*, *10*(6), 269–314.

Flora, D. B., & Curran, P. J. (2004). An empirical evaluation of alternative methods of estimation for confirmatory factor analysis with ordinal data. *Psychological Methods*, *9*(4), 466.

Flouri, E., & Kallis, C. (2007). Adverse life events and psychopathology and prosocial behavior in late adolescence: Testing the timing, specificity, accumulation, gradient, and moderation of contextual risk. *Journal of the American Academy of Child & Adolescent Psychiatry*, *46*(12), 1651–1659.

Flouri, E., Mavroveli, S., & Tzavidis, N. (2010). Modeling risks: Effects of area deprivation, family socio-economic disadvantage and adverse life events on young children's psychopathology. *Social Psychiatry and Psychiatric Epidemiology*, *45*(6), 611–619. https://doi.org/10.1007/s00127-009-0101-x

Folker, A. E., Deater-Deckard, K., Lansford, J. E., Di Giunta, L., Dodge, K. A., Gurdal, S., Liu, Q., Long, Q., Oburu, P., & Pastorelli, C. (2023). Intraindividual variability in parental acceptance-rejection predicts externalizing and internalizing symptoms across

childhood/adolescence in nine countries. *Journal of Family Psychology*. https://psycnet.apa.org/record/2024-10186-001

Forbes, M. K., Greene, A. L., Levin-Aspenson, H. F., Watts, A. L., Hallquist, M., Lahey, B. B., Markon, K. E., Patrick, C. J., Tackett, J. L., & Waldman, I. D. (2021). Three recommendations based on a comparison of the reliability and validity of the predominant models used in research on the empirical structure of psychopathology. *Journal of Abnormal Psychology*, *130*(3), 297.

Forbes, M. K., Neo, B., Nezami, O. M., Fried, E. I., Faure, K., Michelsen, B., Twose, M., & Dras, M. (2023). *Elemental psychopathology: Distilling constituent symptoms and patterns of repetition in the diagnostic criteria of the DSM-5*. https://psyarxiv.com/u56p2/download?format=pdf

Forbes, M. K., Rapee, R. M., & Krueger, R. F. (2019). Opportunities for the prevention of mental disorders by reducing general psychopathology in early childhood. *Behaviour Research and Therapy*, *119*, 103411. https://doi.org/10.1016/j.brat.2019.103411

Forbes, M. K., Tackett, J. L., Markon, K. E., & Krueger, R. F. (2016). Beyond comorbidity: Toward a dimensional and hierarchical approach to understanding psychopathology across the life span. *Development and Psychopathology*, *28*(4pt1), 971–986.

Forbush, K. T., South, S. C., Krueger, R. F., Iacono, W. G., Clark, L. A., Keel, P. K., Legrand, L. N., & Watson, D. (2010). Locating eating pathology within an empirical diagnostic taxonomy: Evidence from a community-based sample. *Journal of Abnormal Psychology*, *119*(2), 282.

Freitag, G. F., Salem, H., Conroy, K., Busto, C., Adrian, M., Borba, C. P., Brandt, A., Chu, P. V., Dantowitz, A., & Farley, A. M. (2023). The Patient-Reported Outcomes Measurement Information System (PROMIS) pediatric and parent-proxy short forms for anxiety:

Psychometric properties in the Kids FACE FEARS sample. *Journal of Anxiety Disorders*, *94*, 102677.

Fried, E. I. (2020). Lack of Theory Building and Testing Impedes Progress in The Factor and Network Literature. *Psychological Inquiry*, *31*(4), 271–288. https://doi.org/10.1080/1047840X.2020.1853461

Fried, E. I., Greene, A. L., & Eaton, N. R. (2021). The p factor is the sum of its parts, for now. *World Psychiatry*, *20*(1), 69.

Fujiwara, T. (2022). Impact of adverse childhood experience on physical and mental health: A life-course epidemiology perspective. *Psychiatry and Clinical Neurosciences*, *76*(11), 544–551.

Fujiwara, T., Kawakami, N., & Group, W. M. H. J. S. (2011). Association of childhood adversities with the first onset of mental disorders in Japan: Results from the World Mental Health Japan, 2002–2004. *Journal of Psychiatric Research*, *45*(4), 481–487.

Gabard-Durnam, L. J., & McLaughlin, K. A. (2019). Do sensitive periods exist for exposure to adversity? *Biological Psychiatry*, *85*(10), 789–791.

Gabriele, M., Pietro, M., Azzurra, M., Francesca, L., Lisa, P., Laura, R., Filippo, M., & Annarita, M. (2013). Response to treatments in youth with disruptive behaviour disorders. *COMPREHENSIVE PSYCHIATRY*, 1009–1015.

Galán, C. A., Shaw, D. S., Dishion, T. J., & Wilson, M. N. (2017). Neighborhood Deprivation during Early Childhood and Conduct Problems in Middle Childhood: Mediation by Aggressive Response Generation. *Journal of Abnormal Child Psychology*, *45*(5), 935–946. https://doi.org/10.1007/s10802-016-0209-x

Garcia-Moreno, C., Jansen, H. A., Ellsberg, M., Heise, L., & Watts, C. H. (2006). Prevalence of intimate partner violence: Findings from the WHO multi-country study on women's health and domestic violence. *The Lancet*, *368*(9543), 1260–1269.

Garnett, M., Reese, E., Swearingen, I., Peterson, E., Salmon, K., Waldie, K., D'souza, S., Atatoa-Carr, P., Morton, S., & Bird, A. (2023). Maternal Reminiscing and Children's Socioemotional Development: Evidence from a Large Pre-Birth Longitudinal Cohort Study, Growing Up in New Zealand. *Journal of Cognition and Development*, 1–26.

Gassen, J., Prokosch, M. L., Eimerbrink, M. J., Proffitt Leyva, R. P., White, J. D., Peterman, J. L., Burgess, A., Cheek, D. J., Kreutzer, A., & Nicolas, S. C. (2019). Inflammation predicts decision-making characterized by impulsivity, present focus, and an inability to delay gratification. *Scientific Reports*, *9*(1), 4928.

Gee, D. G. (2020). Caregiving influences on emotional learning and regulation: Applying a sensitive period model. *Current Opinion in Behavioral Sciences*, *36*, 177–184.

Gee, D. G. (2021). Early adversity and development: Parsing heterogeneity and identifying pathways of risk and resilience. *American Journal of Psychiatry*, *178*(11), 998–1013.

Gee, D. G. (2022). Neurodevelopmental mechanisms linking early experiences and mental health: Translating science to promote well-being among youth. *American Psychologist*, *77*(9), 1033.

Gettler, L. T., McDade, T. W., Bragg, J. M., Feranil, A. B., & Kuzawa, C. W. (2015). Developmental energetics, sibling death, and parental instability as predictors of maturational tempo and life history scheduling in males from Cebu, Philippines. *American Journal of Physical Anthropology*, *158*(2), 175–184. https://doi.org/10.1002/ajpa.22783

Gilbert, R., Kemp, A., Thoburn, J., Sidebotham, P., Radford, L., Glaser, D., & MacMillan, H. L. (2009). Recognising and responding to child maltreatment. *The Lancet*, *373*(9658), 167–180.

Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Burden and consequences of child maltreatment in high-income countries. *The Lancet*, *373*(9657), 68–81.

Gillespie, M. L., & Rao, U. (2022). Relationships between depression and executive functioning in adolescents: The moderating role of unpredictable home environment. *Journal of Child and Family Studies*, *31*(9), 2518–2534.

Glantz, S. A., Slinker, B. K., & Neilands, T. B. (2001). *Primer of applied regression & analysis of variance, ed* (Vol. 654). McGraw-Hill, Inc., New York. http://mhe-assets-asia.s3.amazonaws.com/professional/newtitles/Professional_New_Titles-Feb2016.pdf

Glover, V. (2015). Prenatal Stress and Its Effects on the Fetus and the Child: Possible Underlying Biological Mechanisms. In M. C. Antonelli (Ed.), *Perinatal Programming of Neurodevelopment* (Vol. 10, pp. 269–283). Springer New York. https://doi.org/10.1007/978-1-4939-1372-5_13

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007). Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology*, *19*(1), 1–19.

Glynn, L. M., & Baram, T. Z. (2019). The influence of unpredictable, fragmented parental signals on the developing brain. *Frontiers in Neuroendocrinology*, *53*, 100736.

Godoy, L. C., Frankfurter, C., Cooper, M., Lay, C., Maunder, R., & Farkouh, M. E. (2021). Association of adverse childhood experiences with cardiovascular disease later in life: A review. *JAMA Cardiology*, *6*(2), 228–235.

Goetschius, L. G., Hein, T. C., McLanahan, S. S., Brooks-Gunn, J., McLoyd, V. C., Dotterer, H. L., Lopez-Duran, N., Mitchell, C., Hyde, L. W., & Monk, C. S. (2020). Association of childhood violence exposure with adolescent neural network density. *JAMA Network Open*, *3*(9), e2017850–e2017850.

Goff, B., Gee, D. G., Telzer, E. H., Humphreys, K. L., Gabard-Durnam, L., Flannery, J., & Tottenham, N. (2013). Reduced nucleus accumbens reactivity and adolescent depression following early-life stress. *Neuroscience*, *249*, 129–138.

Gomez, R., Stavropoulos, V., Vance, A., & Griffiths, M. D. (2019). Re-evaluation of the latent structure of common childhood disorders: Is there a general psychopathology factor (p-factor)? *International Journal of Mental Health and Addiction*, *17*, 258–278.

Goodman, A., Lamping, D. L., & Ploubidis, G. B. (2010). When to Use Broader Internalising and Externalising Subscales Instead of the Hypothesised Five Subscales on the Strengths and Difficulties Questionnaire (SDQ): Data from British Parents, Teachers and Children. *Journal of Abnormal Child Psychology*, *38*(8), 1179–1191. https://doi.org/10.1007/s10802-010-9434-x

Goodman, R. (2001). Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child & Adolescent Psychiatry*, *40*(11), 1337–1345.

Goodman, R., & Scott, S. (1999). Comparing the Strengths and Difficulties Questionnaire and the Child Behavior Checklist: Is small beautiful? *Journal of Abnormal Child Psychology*, *27*, 17–24.

Gorman-Smith, D., & Loeber, R. (2005). Are Developmental Pathways in Disruptive Behaviors the Same for Girls and Boys? *Journal of Child and Family Studies*, *14*(1), 15–27. https://doi.org/10.1007/s10826-005-1109-9

Gorsuch, R. L. (2014). *Factor analysis: Classic edition*. Routledge.

Granger, S. J., Glynn, L. M., Sandman, C. A., Small, S. L., Obenaus, A., Keator, D. B., Baram, T. Z., Stern, H., Yassa, M. A., & Davis, E. P. (2021). Aberrant maturation of the uncinate fasciculus follows exposure to unpredictable patterns of maternal signals. *Journal of Neuroscience*, *41*(6), 1242–1250.

Greene, A. L., & Eaton, N. R. (2017). The temporal stability of the bifactor model of comorbidity: An examination of moderated continuity pathways. *Comprehensive Psychiatry*, *72*, 74–82.

Grummitt, L. R., Kreski, N. T., Kim, S. G., Platt, J., Keyes, K. M., & McLaughlin, K. A. (2021). Association of childhood adversity with morbidity and mortality in US adults: A systematic review. *JAMA Pediatrics*, *175*(12), 1269–1278.

Gueron-Sela, N., Camerota, M., Willoughby, M. T., Vernon-Feagans, L., & Cox, M. J. (2018). Maternal depressive symptoms, mother-child interactions, and children's executive function. *Developmental Psychology*, *54*(1), 71.

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annu. Rev. Psychol.*, *58*, 145–173.

Gunnar, M. R., & Vazquez, D. (2015). Stress Neurobiology and Developmental Psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental Psychopathology* (1st ed., pp. 533–577). Wiley. https://doi.org/10.1002/9780470939390.ch13

Guyon-Harris, K. L., Plamondon, A., Humphreys, K. L., Wade, M., Gleason, M. M., Tibu, F., Nelson, C. A., Fox, N. A., & Zeanah, C. H. (2023). Structure of Psychopathology in Romanian Preschool-Aged Children in an Epidemiological and a High-Risk Sample. *JAACAP Open*.

Hagquist, C. (2007). The psychometric properties of the self-reported SDQ–An analysis of Swedish data based on the Rasch model. *Personality and Individual Differences*, *43*(5), 1289–1301.

Halligan, S. L., Herbert, J., Goodyer, I. M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biological Psychiatry*, *55*(4), 376–381.

Hancock, G. R., & Mueller, R. O. (2001). Rethinking construct reliability within latent variable systems. *Structural Equation Modeling: Present and Future*, *195*, 216.

Hankin, B. L., Davis, E. P., Snyder, H., Young, J. F., Glynn, L. M., & Sandman, C. A. (2017). Temperament factors and dimensional, latent bifactor models of child psychopathology: Transdiagnostic and specific associations in two youth samples. *Psychiatry Research*, *252*, 139–146.

Hanson, J. L., Chung, M. K., Avants, B. B., Shirtcliff, E. A., Gee, J. C., Davidson, R. J., & Pollak, S. D. (2010). Early stress is associated with alterations in the orbitofrontal cortex: A tensor-

based morphometry investigation of brain structure and behavioral risk. *Journal of Neuroscience*, *30*(22), 7466–7472.

Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., Shirtcliff, E. A., Pollak, S. D., & Davidson, R. J. (2015). Behavioral problems after early life stress: Contributions of the hippocampus and amygdala. *Biological Psychiatry*, *77*(4), 314–323.

Harhen, N. C., & Bornstein, A. M. (2024). Interval Timing as a Computational Pathway From Early Life Adversity to Affective Disorders. *Topics in Cognitive Science*, *16*(1), 92–112. https://doi.org/10.1111/tops.12701

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

Heckers, S. (2014). Future in psychopathology research. *Schizophrenia Bulletin*, *40*(Suppl_2), S147–S151.

Heinrich, M., Geiser, C., Zagorscak, P., Burns, G. L., Bohn, J., Becker, S. P., Eid, M., Beauchaine, T. P., & Knaevelsrud, C. (2023). On the meaning of the "P factor" in symmetrical Bifactor models of psychopathology: Recommendations for future research From the Bifactor-(S- 1) perspective. *Assessment*, *30*(3), 487–507.

Heinrich, M., Zagorscak, P., Eid, M., & Knaevelsrud, C. (2020). Giving G a Meaning: An Application of the Bifactor-(S-1) Approach to Realize a More Symptom-Oriented Modeling of the Beck Depression Inventory–II. *Assessment*, *27*(7), 1429–1447. https://doi.org/10.1177/1073191118803738

Heleniak, C., & McLaughlin, K. A. (2020). Social-cognitive mechanisms in the cycle of violence: Cognitive and affective theory of mind, and externalizing psychopathology in children and adolescents. *Development and Psychopathology*, *32*(2), 735–750.

Henry, L. M., Gracey, K., Shaffer, A., Ebert, J., Kuhn, T., Watson, K. H., Gruhn, M., Vreeland, A., Siciliano, R., & Dickey, L. (2021). Comparison of three models of adverse childhood experiences: Associations with child and adolescent internalizing and externalizing symptoms. *Journal of Abnormal Psychology*, *130*(1), 9.

Herry, C., Bach, D. R., Esposito, F., Di Salle, F., Perrig, W. J., Scheffler, K., Lüthi, A., & Seifritz, E. (2007). Processing of temporal unpredictability in human and animal amygdala. *Journal of Neuroscience*, *27*(22), 5958–5966.

Hertzman, C. (2012). Putting the concept of biological embedding in historical perspective. *Proceedings of the National Academy of Sciences*, *109*(supplement_2), 17160–17167.

Herzberg, M. P., Hodel, A. S., Cowell, R. A., Hunt, R. H., Gunnar, M. R., & Thomas, K. M. (2018). Risk taking, decision-making, and brain volume in youth adopted internationally from institutional care. *Neuropsychologia*, *119*, 262–270.

Herzog, J. I., & Schmahl, C. (2018). Adverse childhood experiences and the consequences on neurobiological, psychosocial, and somatic conditions across the lifespan. *Frontiers in Psychiatry*, *9*, 420.

Hill, D., Ameenuddin, N., Reid Chassiakos, Y. L., Cross, C., Hutchinson, J., Levine, A., Boyd, R., Mendelson, R., Moreno, M., & Swanson, W. S. (2016). Media and young minds. *Pediatrics*, *138*(5).

Hill, K., & Kaplan, H. (1999). Life History Traits in Humans: Theory and Empirical Studies. *Annual Review of Anthropology*, *28*(1), 397–430. https://doi.org/10.1146/annurev.anthro.28.1.397

Hodel, A. S., Hunt, R. H., Cowell, R. A., Van Den Heuvel, S. E., Gunnar, M. R., & Thomas, K. M. (2015). Duration of early adversity and structural brain development in post-institutionalized adolescents. *NeuroImage*, *105*, 112–119.

Hoffmann, M. S., Moore, T. M., Axelrud, L. K., Tottenham, N., Pan, P. M., Miguel, E. C., Rohde, L. A., Milham, M. P., Satterthwaite, T. D., & Salum, G. A. (2024). An Evaluation of Item Harmonization Strategies Between Assessment Tools of Psychopathology in Children and Adolescents. *Assessment*, *31*(2), 502–517. https://doi.org/10.1177/10731911231163136

Hoffmann, M. S., Moore, T. M., Axelrud, L. K., Tottenham, N., Zuo, X.-N., Rohde, L. A., Milham, M. P., Satterthwaite, T. D., & Abrahão Salum, G. (2021). Reliability and validity of Bifactor models of dimensional psychopathology in youth from three continents. *medRxiv*, 2021–06.

Holtmann, M., Becker, A., Banaschewski, T., Rothenberger, A., & Roessner, V. (2011). Psychometric validity of the strengths and difficulties questionnaire-dysregulation profile. *Psychopathology*, *44*(1), 53–59.

Hua, J. Y., & Smith, S. J. (2004). Neural activity and the dynamics of central nervous system development. *Nature Neuroscience*, *7*(4), 327–332.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, *6*(1), 1–55. https://doi.org/10.1080/10705519909540118

Humphrey, G., Rossen, F., Greenaway, N., & Bullen, C. (2016). *Parental smoking during pregnancy: Findings from the growing up in New Zealand cohort*.

Humphreys, K. L., Guyon-Harris, K. L., Tibu, F., Wade, M., Nelson III, C. A., Fox, N. A., & Zeanah, C. H. (2020). Psychiatric outcomes following severe deprivation in early childhood: Follow-up of a randomized controlled trial at age 16. *Journal of Consulting and Clinical Psychology*, *88*(12), 1079.

Humphreys, K. L., LeMoult, J., Wear, J. G., Piersiak, H. A., Lee, A., & Gotlib, I. H. (2020). Child maltreatment and depression: A meta-analysis of studies using the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, *102*, 104361.

Humphreys, K. L., & Salo, V. C. (2020). Expectable environments in early life. *Current Opinion in Behavioral Sciences*, *36*, 115–119.

Humphreys, K. L., & Zeanah, C. H. (2015). Deviations from the expectable environment in early childhood and emerging psychopathology. *Neuropsychopharmacology*, *40*(1), 154–170.

Hur, E., Buettner, C. K., & Jeon, L. (2015). Parental Depressive Symptoms and Children's School-Readiness: The Indirect Effect of Household Chaos. *Journal of Child and Family Studies*, *24*(11), 3462–3473. https://doi.org/10.1007/s10826-015-0147-1

Hurst, J. E., & Kavanagh, P. S. (2017). Life history strategies and psychopathology: The faster the life strategies, the more symptoms of psychopathology. *Evolution and Human Behavior*, *38*(1), 1–8.

Hyland, P., Karatzias, T., Ford, J. D., Fox, R., & Spinazzola, J. (2022). The Latent Structure of Child and Adolescent Psychopathology and its Association with Different Forms of Trauma and Suicidality and Self-Harm. *Research on Child and Adolescent Psychopathology*, *50*(11), 1501–1513. https://doi.org/10.1007/s10802-022-00925-1

Ortuño-Sierra, J., Fonseca-Pedrero, E., Aritio-Solana, R., Velasco, A. M., De Luis, E. C., Schumann, G., Cattrell, A., Flor, H., Nees, F., Banaschewski, T., Bokde, A., Whelan, R., Buechel, C., Bromberg, U., Conrod, P., Frouin, V., Papadopoulos, D., Gallinat, J., Lawrence, C. (2015). New evidence of factor structure and measurement invariance of the SDQ across five European nations. *European Child & Adolescent Psychiatry*, *24*(12), 1523–1534. https://doi.org/10.1007/s00787-015-0729-x

Ismail, F. Y., Fatemi, A., & Johnston, M. V. (2017). Cerebral plasticity: Windows of opportunity in the developing brain. *European Journal of Paediatric Neurology*, *21*(1), 23–48.

Iverach, L., Menzies, R. G., & Menzies, R. E. (2014). Death anxiety and its role in psychopathology: Reviewing the status of a transdiagnostic construct. *Clinical Psychology Review*, *34*(7), 580–593.

Janssens, A., & Deboutte, D. (2009). Screening for psychopathology in child welfare: The Strengths and Difficulties Questionnaire (SDQ) compared with the Achenbach System of Empirically Based Assessment (ASEBA). *European Child & Adolescent Psychiatry*, *18*, 691–700.

Johansen, J. P., Cain, C. K., Ostroff, L. E., & LeDoux, J. E. (2011). Molecular mechanisms of fear learning and memory. *Cell*, *147*(3), 509–524.

Johnson, D., Policelli, J., Li, M., Dharamsi, A., Hu, Q., Sheridan, M. A., McLaughlin, K. A., & Wade, M. (2021). Associations of early-life threat and deprivation with executive functioning in childhood and adolescence: A systematic review and meta-analysis. *JAMA Pediatrics*, *175*(11), e212511–e212511.

Johnston, C., & Murray, C. (2003). Incremental validity in the psychological assessment of children and adolescents. *Psychological Assessment*, *15*(4), 496.

Junghänel, M., Rodenacker, K., Dose, C., & Döpfner, M. (2020). Applying the Bifactor S-1 Model to Ratings of ADHD/ODD Symptoms: A Commentary on Burns et al. (2019) and a Re-Analysis. *Journal of Abnormal Child Psychology*, *48*(7), 905–910. https://doi.org/10.1007/s10802-020-00637-4

Juwariah, T., Suhariadi, F., Soedirham, O., Priyanto, A., Setiyorini, E., Siskaningrum, A., Adhianata, H., & Fernandes, A. D. C. (2022). Childhood adversities and mental health problems: A systematic review. *Journal of Public Health Research*, *11*(3), 227990362211066. https://doi.org/10.1177/22799036221106613

Kahl, B. L., Kavanagh, P. S., & Gleaves, D. H. (2022). Testing a life history model of psychopathology: A replication and extension. *Current Psychology*, *41*(9), 6233–6246. https://doi.org/10.1007/s12144-020-01062-y

Kalmakis, K. A., & Chandler, G. E. (2015). Health consequences of adverse childhood experiences: A systematic review. *Journal of the American Association of Nurse Practitioners*, *27*(8), 457–465. https://doi.org/10.1002/2327-6924.12215

Karatekin, C., & Hill, M. (2019). Expanding the original definition of adverse childhood experiences (ACEs). *Journal of Child & Adolescent Trauma*, *12*, 289–306.

Karcher, N. R., Michelini, G., Kotov, R., & Barch, D. M. (2021a). Associations Between Resting State Functional Connectivity and a Hierarchical Dimensional Structure of Psychopathology in Middle Childhood. *Biological Psychiatry. Cognitive Neuroscience and Neuroimaging*, *6*(5), 508–517. https://doi.org/10.1016/j.bpsc.2020.09.008

Karcher, N. R., Michelini, G., Kotov, R., & Barch, D. M. (2021b). Associations between resting-state functional connectivity and a hierarchical dimensional structure of psychopathology in middle childhood. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *6*(5), 508–517.

Kawabe, K., Horiuchi, F., Uno, H., Nakachi, K., Hosokawa, R., Oka, Y., & Ueno, S.-I. (2021). Parent-Adolescent Agreement on Adolescents' Emotional and Behavioral Problems Assessed by the Strengths and Difficulties Questionnaire. *Global Pediatric Health*, *8*, 2333794X2110012. https://doi.org/10.1177/2333794X211001245

Keller, F., & Langmeyer, A. N. (2019). An Item Response Theory Analysis of the Strength and Difficulties Questionnaire (SDQ). *European Journal of Psychological Assessment*, *35*(2), 266–279. https://doi.org/10.1027/1015-5759/a000390

Kersten, P., Czuba, K., McPherson, K., Dudley, M., Elder, H., Tauroa, R., & Vandal, A. (2016). A systematic review of evidence for the psychometric properties of the Strengths and Difficulties Questionnaire. *International Journal of Behavioral Development*, *40*(1), 64–75. https://doi.org/10.1177/0165025415570647

Kessler, R. C., Adler, L., Ames, M., Demler, O., Faraone, S., Hiripi, E. V. A., Howes, M. J., Jin, R., Secnik, K., & Spencer, T. (2005). The World Health Organization Adult ADHD Self-Report Scale (ASRS): A short screening scale for use in the general population. *Psychological Medicine*, *35*(2), 245–256.

Kessler, R. C., Davis, C. G., & Kendler, K. S. (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine*, *27*(5), 1101–1119.

Kessler, R. C., McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., Aguilar-Gaxiola, S., Alhamzawi, A. O., Alonso, J., & Angermeyer, M. (2010). Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *The British Journal of Psychiatry*, *197*(5), 378–385.

Keyes, K. M., Eaton, N. R., Krueger, R. F., McLaughlin, K. A., Wall, M. M., Grant, B. F., & Hasin, D. S. (2012). Childhood maltreatment and the structure of common psychiatric disorders. *The British Journal of Psychiatry*, *200*(2), 107–115.

Kim, H., & Eaton, N. R. (2015). The hierarchical structure of common mental disorders: Connecting multiple levels of comorbidity, bifactor models, and predictive validity. *Journal of Abnormal Psychology*, *124*(4), 1064.

Kim-Spoon, J., Maciejewski, D., Lee, J., Deater-Deckard, K., & King-Casas, B. (2017). Longitudinal associations among family environment, neural cognitive control, and social competence among adolescents. *Developmental Cognitive Neuroscience*, *26*, 69–76.

King, R. B., & Bures, R. M. (2017). How the social environment gets under the skin. *Population Research and Policy Review*, *36*, 631–637.

Klahr, A. M., & Burt, S. A. (2014). Practitioner Review: Evaluation of the known behavioral heterogeneity in conduct disorder to improve its assessment and treatment. *Journal of Child Psychology and Psychiatry*, *55*(12), 1300–1310. https://doi.org/10.1111/jcpp.12268

Kline, T. J. (2005). *Psychological testing: A practical approach to design and evaluation*. Sage publications.

Kóbor, A., Takács, Á., & Urbán, R. (2013). The Bifactor Model of the Strengths and Difficulties Questionnaire. *European Journal of Psychological Assessment*, *29*(4), 299–307. https://doi.org/10.1027/1015-5759/a000160

Kolb, B., & Gibb, R. (2011). Brain plasticity and behaviour in the developing brain. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, *20*(4), 265.

Konzok, J., Henze, G., Peter, H., Giglberger, M., Bärtl, C., Massau, C., Kärgel, C., Schiffer, B., Eisenbarth, H., Wüst, S., & Kudielka, B. M. (2021). Externalizing behavior in healthy young adults is associated with lower cortisol responses to acute stress and altered neural activation in the dorsal striatum. *Psychophysiology*, *58*(12), e13936. https://doi.org/10.1111/psyp.13936

Koss, K. J., & Gunnar, M. R. (2018). Annual research review: Early adversity, the hypothalamic–pituitary–adrenocortical axis, and child psychopathology. *Journal of Child Psychology and Psychiatry*, *59*(4), 327–346.

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., & Clark, L. A. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, *126*(4), 454.

Kraemer, H. C., Lowe, K. K., & Kupfer, D. J. (2005). *To your health: How to understand what research tells us about risk*. Oxford University Press.

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*(10), 921–926.

Krueger, R. F., & Eaton, N. R. (2015). Transdiagnostic factors of mental disorders. *World Psychiatry*, *14*(1), 27.

Kunze, B., Wang, B., Isensee, C., Schlack, R., Ravens-Sieberer, U., Klasen, F., Rothenberger, A., Becker, A., & Group, B.-S. (2018). Gender associated developmental trajectories of SDQ-dysregulation profile and its predictors in children. *Psychological Medicine*, *48*(3), 404–415.

Laceulle, O. M., Vollebergh, W. A., & Ormel, J. (2015). The structure of psychopathology in adolescence: Replication of a general psychopathology factor in the TRAILS study. *Clinical Psychological Science*, *3*(6), 850–860.

Lahey, B. B., Applegate, B., Waldman, I. D., Loft, J. D., Hankin, B. L., & Rick, J. (2004). The structure of child and adolescent psychopathology: Generating new hypotheses. *Journal of Abnormal Psychology*, *113*(3), 358.

Lahey, B. B., Moore, T. M., Kaczkurkin, A. N., & Zald, D. H. (2021). Hierarchical models of psychopathology: Empirical support, implications, and remaining issues. *World Psychiatry*, *20*(1), 57–63.

Lahey, B. B., Rathouz, P. J., Keenan, K., Stepp, S. D., Loeber, R., & Hipwell, A. E. (2015). Criterion validity of the general factor of psychopathology in a prospective study of girls. *Journal of Child Psychology and Psychiatry*, *56*(4), 415–422.

Lahey, B. B., Zald, D. H., Perkins, S. F., Villalta-Gil, V., Werts, K. B., Van Hulle, C. A., Rathouz, P. J., Applegate, B., Class, Q. A., & Poore, H. E. (2018). Measuring the hierarchical general factor model of psychopathology in young adults. *International Journal of Methods in Psychiatric Research*, *27*(1), e1593.

Lauharatanahirun, N., Maciejewski, D., Holmes, C., Deater-Deckard, K., Kim-Spoon, J., & King-Casas, B. (2018). Neural Correlates of Risk Processing Among Adolescents: Influences of Parental Monitoring and Household Chaos. *Child Development*, *89*(3), 784–796. https://doi.org/10.1111/cdev.13036

Lee, T., & Shi, D. (2021). A comparison of full information maximum likelihood and multiple imputation in structural equation modeling with missing data. *Psychological Methods*, *26*(4), 466.

Levin, M. E., MacLane, C., Daflos, S., Seeley, J. R., Hayes, S. C., Biglan, A., & Pistorello, J. (2014). Examining psychological inflexibility as a transdiagnostic process across psychological disorders. *Journal of Contextual Behavioral Science*, *3*(3), 155–163.

Levin-Aspenson, H. F., Watson, D., Clark, L. A., & Zimmerman, M. (2021). What is the general factor of psychopathology? Consistency of the p factor across samples. *Assessment*, *28*(4), 1035–1049.

Li, C.H. (2016). Confirmatory factor analysis with ordinal data: Comparing robust maximum likelihood and diagonally weighted least squares. *Behavior Research Methods*, *48*(3), 936–949. https://doi.org/10.3758/s13428-015-0619-7

Li, Z., & Belsky, J. (2022). Indirect effects, via parental factors, of income harshness and unpredictability on kindergarteners' socioemotional functioning. *Development and Psychopathology*, *34*(2), 635–646.

Liu, S., & Fisher, P. A. (2022). Early experience unpredictability in child development as a model for understanding the impact of the COVID-19 pandemic: A translational neuroscience perspective. *Developmental Cognitive Neuroscience*, *54*, 101091.

Liu, Y., Hatch, S. L., Patalay, P., Schott, J. M., & Richards, M. (2023). A lifecourse approach in examining the association between accumulation of adversity and mental health in older adulthood. *Journal of Affective Disorders*, *339*, 211–218.

Lynch, S. J., Sunderland, M., Newton, N. C., & Chapman, C. (2020). Transdiagnostic risk and protective factors for psychopathology in young people: Systematic review protocol. *JMIR Research Protocols*, *9*(8), e19779.

Lynch, S. J., Sunderland, M., Newton, N. C., & Chapman, C. (2021). A systematic review of transdiagnostic risk and protective factors for general and specific psychopathology in young people. *Clinical Psychology Review*, *87*, 102036.

Machlin, L. (2022). *How Early Adversity Becomes Biologically Embedded and Associated Risk for Psychopathology* [PhD Thesis]. The University of North Carolina at Chapel Hill.

Machlin, L., Miller, A. B., Snyder, J., McLaughlin, K. A., & Sheridan, M. A. (2019). Differential associations of deprivation and threat with cognitive control and fear conditioning in early childhood. *Frontiers in Behavioral Neuroscience*, 80.

Malhi, G. S., Das, P., Bell, E., Mattingly, G., & Mannie, Z. (2019). Modelling resilience in adolescence and adversity: A novel framework to inform research and practice. *Translational Psychiatry*, *9*(1), Article 1. https://doi.org/10.1038/s41398-019-0651-y

Marsh, S., Dobson, R., & Maddison, R. (2020). The relationship between household chaos and child, parent, and family outcomes: A systematic scoping review. *BMC Public Health*, *20*(1), 513. https://doi.org/10.1186/s12889-020-08587-8

Martel, M. M., Markon, K., & Smith, G. T. (2017). Research Review: Multi-informant integration in child and adolescent psychopathology diagnosis. *Journal of Child Psychology and Psychiatry*, *58*(2), 116–128.

Martinez, J. L., Hasty, C., Morabito, D., Maranges, H. M., Schmidt, N. B., & Maner, J. K. (2022). Perceptions of childhood unpredictability, delay discounting, risk-taking, and adult externalizing behaviors: A life-history approach. *Development and Psychopathology*, *34*(2), 705–717.

Martini, J., Li, M., Wright, L., Johnson, D., & Wade, M. (2022). Toward a dimensional model of family-based resilience: Sensitivity, stimulation, and stability as moderators of early-life adversity. *Canadian Psychology/Psychologie Canadienne*, *63*(2), 214.

Marusak, H. A., Etkin, A., & Thomason, M. E. (2015). Disrupted insula-based neural circuit organization and conflict interference in trauma-exposed youth. *NeuroImage: Clinical*, *8*, 516–525.

Masi, G., Milone, A., Pisano, S., Lenzi, F., Muratori, P., Gemo, I., Bianchi, L., Mazzone, L., Postorino, V., & Sanges, V. (2014). Emotional reactivity in referred youth with disruptive behavior disorders: The role of the callous-unemotional traits. *Psychiatry Research*, *220*(1–2), 426–432.

Masi, G., Muratori, P., Manfredi, A., Pisano, S., & Milone, A. (2015). Child behaviour checklist emotional dysregulation profiles in youth with disruptive behaviour disorders: Clinical correlates and treatment implications. *Psychiatry Research*, *225*(1–2), 191–196.

McCrory, E., De Brito, S. A., & Viding, E. (2010). Research Review: The neurobiology and genetics of maltreatment and adversity: Research Review: The neurobiology and genetics of maltreatment and adversity. *Journal of Child Psychology and Psychiatry*, *51*(10), 1079–1095. https://doi.org/10.1111/j.1469-7610.2010.02271.x

McCrory, E. J., De Brito, S. A., Kelly, P. A., Bird, G., Sebastian, C. L., Mechelli, A., Samuel, S., & Viding, E. (2013). Amygdala activation in maltreated children during pre-attentive emotional processing. *The British Journal of Psychiatry*, *202*(4), 269–276.

McElroy, E., Belsky, J., Carragher, N., Fearon, P., & Patalay, P. (2018). Developmental stability of general and specific factors of psychopathology from early childhood to adolescence: Dynamic mutualism or *p* -differentiation? *Journal of Child Psychology and Psychiatry*, *59*(6), 667–675. https://doi.org/10.1111/jcpp.12849

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44.

McEwen, B. S. (2003). Mood disorders and allostatic load. *Biological Psychiatry*, *54*(3), 200–207.

McGinnis, E. W., Sheridan, M., & Copeland, W. E. (2022). Impact of dimensions of early adversity on adult health and functioning: A 2-decade, longitudinal study. *Development and Psychopathology*, *34*(2), 527–538.

McLaughlin, K. A. (2016). Future directions in childhood adversity and youth psychopathology. *Journal of Clinical Child & Adolescent Psychology*, *45*(3), 361–382.

McLaughlin, K. A., Colich, N. L., Rodman, A. M., & Weissman, D. G. (2020). Mechanisms linking childhood trauma exposure and psychopathology: A transdiagnostic model of risk and resilience. *BMC Medicine*, *18*(1), 1–11.

McLaughlin, K. A., DeCross, S. N., Jovanovic, T., & Tottenham, N. (2019). Mechanisms linking childhood adversity with psychopathology: Learning as an intervention target. *Behaviour Research and Therapy*, *118*, 101–109.

McLaughlin, K. A., & Gabard-Durnam, L. (2022). Experience-driven plasticity and the emergence of psychopathology: A mechanistic framework integrating development and the environment into the Research Domain Criteria (RDoC) model. *Journal of Psychopathology and Clinical Science*, *131*(6), 575.

McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychopathology in the National Comorbidity Survey Replication (NCS-R) III: Associations with functional impairment related to DSM-IV disorders. *Psychological Medicine*, *40*(5), 847–859.

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Psychological Science*, *25*(4), 239–245.

McLaughlin, K. A., Sheridan, M. A., Humphreys, K. L., Belsky, J., & Ellis, B. J. (2021). The Value of Dimensional Models of Early Experience: Thinking Clearly about Concepts and Categories. *Perspectives on Psychological Science : A Journal of the Association for Psychological Science*, *16*(6), 1463–1472. https://doi.org/10.1177/1745691621992346

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience & Biobehavioral Reviews*, *47*, 578–591.

McLaughlin, K. A., Weissman, D., & Bitrán, D. (2019). Childhood adversity and neural development: A systematic review. *Annual Review of Developmental Psychology*, *1*, 277–312.

Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R., Rutter, M., & Sonuga-Barke, E. J. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian Adoptees Study Pilot. *Journal of Child Psychology and Psychiatry*, *50*(8), 943–951. https://doi.org/10.1111/j.1469-7610.2009.02084.x

Mehta, M. A., Gore-Langton, E., Golembo, N., Colvert, E., Williams, S. C., & Sonuga-Barke, E. (2010). Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. *Journal of Cognitive Neuroscience*, *22*(10), 2316–2325.

Memmott-Elison, M. K., & Toseeb, U. (2023). Prosocial behavior and psychopathology: An 11-year longitudinal study of inter-and intraindividual reciprocal relations across childhood and adolescence. *Development and Psychopathology*, *35*(4), 1982–1996.

Menzies, R. E., Richmond, B., Sharpe, L., Skeggs, A., Liu, J., & Coutts-Bain, D. (2024). The 'revolving door' of mental illness: A META-ANALYSIS and systematic review of current versus lifetime rates of psychological disorders. *British Journal of Clinical Psychology*, bjc.12453. https://doi.org/10.1111/bjc.12453

Michael, C., Tillem, S., Sripada, C. S., Burt, S. A., Klump, K. L., & Hyde, L. W. (2023). Neighborhood poverty during childhood prospectively predicts adolescent functional brain network architecture. *Developmental Cognitive Neuroscience*, *64*, 101316.

Michelini, G., Barch, D. M., Tian, Y., Watson, D., Klein, D. N., & Kotov, R. (2019). Delineating and validating higher-order dimensions of psychopathology in the Adolescent Brain Cognitive Development (ABCD) study. *Translational Psychiatry*, *9*(1), 261.

Michelini, G., Palumbo, I. M., DeYoung, C. G., Latzman, R. D., & Kotov, R. (2021). Linking RDoC and HiTOP: A new interface for advancing psychiatric nosology and neuroscience. *Clinical Psychology Review*, *86*, 102025. https://doi.org/10.1016/j.cpr.2021.102025

Milledge, S. V., Cortese, S., Thompson, M., McEwan, F., Rolt, M., Meyer, B., Sonuga-Barke, E., & Eisenbarth, H. (2019). Peer relationships and prosocial behaviour differences across disruptive behaviours. *European Child & Adolescent Psychiatry*, *28*(6), 781–793. https://doi.org/10.1007/s00787-018-1249-2

Miller, A. B., Machlin, L., McLaughlin, K. A., & Sheridan, M. A. (2021). Deprivation and psychopathology in the Fragile Families Study: A 15-year longitudinal investigation. *Journal of Child Psychology and Psychiatry*, *62*(4), 382–391.

Miller, A. B., Sheridan, M. A., Hanson, J. L., McLaughlin, K. A., Bates, J. E., Lansford, J. E., Pettit, G. S., & Dodge, K. A. (2018). Dimensions of deprivation and threat, psychopathology, and potential mediators: A multi-year longitudinal analysis. *Journal of Abnormal Psychology*, *127*(2), 160.

Mills-Koonce, W. R., Willoughby, M. T., Garrett-Peters, P., Wagner, N., Vernon-Feagans, L., & Investigators, F. L. P. K. (2016). The interplay among socioeconomic status, household chaos, and parenting in the prediction of child conduct problems and callous–unemotional behaviors. *Development and Psychopathology*, *28*(3), 757–771.

Miočević, M., O'Rourke, H. P., MacKinnon, D. P., & Brown, H. C. (2018). Statistical properties of four effect-size measures for mediation models. *Behavior Research Methods*, *50*(1), 285–301. https://doi.org/10.3758/s13428-017-0870-1.

Mittal, C., Griskevicius, V., Simpson, J. A., Sung, S., & Young, E. S. (2015). Cognitive adaptations to stressful environments: When childhood adversity enhances adult executive function. *Journal of Personality and Social Psychology*, *109*(4), 604.

Miu, A. C., Szentágotai-Tătar, A., Balázsi, R., Nechita, D., Bunea, I., & Pollak, S. D. (2022). Emotion regulation as mediator between childhood adversity and psychopathology: A meta-analysis. *Clinical Psychology Review*, *93*, 102141. https://doi.org/10.1016/j.cpr.2022.102141

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., Houts, R., Poulton, R., Roberts, B. W., Ross, S., Sears, M. R., Thomson, W. M., & Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences*, *108*(7), 2693–2698. https://doi.org/10.1073/pnas.1010076108

Monroe, S. M. (2008). Modern approaches to conceptualizing and measuring human life stress. *Annu. Rev. Clin. Psychol.*, *4*, 33–52.

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, *110*(3), 406.

Morgan, C., Gayer-Anderson, C., Beards, S., Hubbard, K., Mondelli, V., Di Forti, M., Murray, R. M., Pariante, C., Dazzan, P., & Craig, T. J. (2020). Threat, hostility and violence in childhood and later psychotic disorder: Population-based case–control study. *The British Journal of Psychiatry*, *217*(4), 575–582.

Morrison, F. J., Ponitz, C. C., & McClelland, M. M. (2010). *Self-regulation and academic achievement in the transition to school.* https://psycnet.apa.org/record/2009-08923-011

Morton, S., Knowles, S., & Morar, M. (2021). *Academic Perspectives of Wellbeing*. https://swa.govt.nz/assets/Publications/reports/SWA-Academic-Perspectives-of-Wellbeing-Final-for-publication-Oct-2021-2....pdf

Morton, S. M., Atatoa Carr, P. E., Grant, C. C., Robinson, E. M., Bandara, D. K., Bird, A., Ivory, V. C., Kingi, T. K. R., Liang, R., & Marks, E. J. (2013). Cohort profile: Growing up in New Zealand. *International Journal of Epidemiology*, *42*(1), 65–75.

Morton, S. M., Grant, C. C., Carr, P. E. A., Robinson, E. M., Kinloch, J. M., Fleming, C. J., Kingi, T. K. R., Perese, L. M., & Liang, R. (2014). How do you recruit and retain a prebirth cohort? Lessons learnt from Growing Up in New Zealand. *Evaluation & the Health Professions*, *37*(4), 411–433.

Morton, S. M., Napier, C., Morar, M., Waldie, K., Peterson, E., Atatoa Carr, P., Meissel, K., Paine, S.-J., Grant, C. C., & Bullen, P. (2022). Mind the gap–unequal from the start: Evidence from the early years of the Growing Up in New Zealand longitudinal study. *Journal of the Royal Society of New Zealand*, *52*(3), 216–236.

Morton, S. M., Ramke, J., Kinloch, J., Grant, C. C., Carr, P. A., Leeson, H., Lee, A. C. L., & Robinson, E. (2015). Growing Up in New Zealand cohort alignment with all New Zealand births. *Australian and New Zealand Journal of Public Health*, *39*(1), 82–87.

Murray, A. L., Booth, T., Eisner, M., Obsuth, I., & Ribeaud, D. (2019). Quantifying the strength of general factors in psychopathology: A comparison of CFA with maximum likelihood estimation, BSEM, and ESEM/EFA bifactor approaches. *Journal of Personality Assessment*, *101*(6), 631–643.

Murray, A. L., Eisner, M., & Ribeaud, D. (2016). The development of the general factor of psychopathology ‘p factor’through childhood and adolescence. *Journal of Abnormal Child Psychology*, *44*(8), 1573–1586.

Murray, A. L., Eisner, M., & Ribeaud, D. (2020). Within-person analysis of developmental cascades between externalising and internalising problems. *Journal of Child Psychology and Psychiatry*, *61*(6), 681–688. https://doi.org/10.1111/jcpp.13150

Muthén, B. O., & Curran, P. J. (1997). General longitudinal modeling of individual differences in experimental designs: A latent variable framework for analysis and power estimation. *Psychological Methods*, *2*(4), 371.

Muthén, B., Kaplan, D., & Hollis, M. (1987). On structural equation modeling with data that are not missing completely at random. *Psychometrika*, *52*(3), 431–462. https://doi.org/10.1007/BF02294365

Najman, J. M., Williams, G. M., Nikles, J., Spence, S., Bor, W., O'Callaghan, M., Le Brocque, R., Andersen, M. J., & Shuttlewood, G. J. (2001). Bias influencing maternal reports of child behaviour and emotional state. *Social Psychiatry and Psychiatric Epidemiology*, *36*(4), 186–194. https://doi.org/10.1007/s001270170062

Nantel-Vivier, A., Pihl, R. O., Côté, S., & Tremblay, R. E. (2014). Developmental association of prosocial behaviour with aggression, anxiety and depression from infancy to preadolescence. *Journal of Child Psychology and Psychiatry*, *55*(10), 1135–1144. https://doi.org/10.1111/jcpp.12235

Nelson, C. A., & Gabard-Durnam, L. J. (2020). Early adversity and critical periods: Neurodevelopmental consequences of violating the expectable environment. *Trends in Neurosciences*, *43*(3), 133–143.

Nettle, D. (2010). Dying young and living fast: Variation in life history across English neighborhoods. *Behavioral Ecology*, *21*(2), 387–395.

Neumann, D., Peterson, E. R., Underwood, L., Morton, S. M. B., & Waldie, K. E. (2021). Exploring the Factor Structure of the NIH Toolbox Cognition Battery in a Large Sample of 8-Year-Old Children in Aotearoa New Zealand. *Journal of the International Neuropsychological Society*, *27*(8), 825–834. https://doi.org/10.1017/S1355617720001265

Newbury, J. B., Arseneault, L., Moffitt, T. E., Caspi, A., Danese, A., Baldwin, J. R., & Fisher, H. L. (2018). Measuring childhood maltreatment to predict early-adult psychopathology:

Comparison of prospective informant-reports and retrospective self-reports. *Journal of Psychiatric Research*, *96*, 57–64.

Ning, K., Gondek, D., Pereira, S. M. P., & Lacey, R. E. (2023). Mediating mechanisms of the relationship between exposure to deprivation and threat during childhood and adolescent psychopathology: Evidence from the Millennium Cohort Study. *European Child & Adolescent Psychiatry*. https://doi.org/10.1007/s00787-023-02289-3

Nixon, G. M., Thompson, J. M., Han, D. Y., Becroft, D. M., Clark, P. M., Robinson, E., Waldie, K. E., Wild, C. J., Black, P. N., & Mitchell, E. A. (2008). Short sleep duration in middle childhood: Risk factors and consequences. *Sleep*, *31*(1), 71–78.

Nolen-Hoeksema, S., & Watkins, E. R. (2011). A heuristic for developing transdiagnostic models of psychopathology: Explaining multifinality and divergent trajectories. *Perspectives on Psychological Science*, *6*(6), 589–609.

Noordhof, A., Krueger, R. F., Ormel, J., Oldehinkel, A. J., & Hartman, C. A. (2015). Integrating Autism-Related Symptoms into the Dimensional Internalizing and Externalizing Model of Psychopathology. The TRAILS Study. *Journal of Abnormal Child Psychology*, *43*(3), 577–587. https://doi.org/10.1007/s10802-014-9923-4

Nweze, T., Nwoke, M. B., Nwufo, J. I., Aniekwu, R. I., & Lange, F. (2021). Working for the future: Parentally deprived Nigerian Children have enhanced working memory ability. *Journal of Child Psychology and Psychiatry*, *62*(3), 280–288. https://doi.org/10.1111/jcpp.13241

O'brien, R. M. (2007). A Caution Regarding Rules of Thumb for Variance Inflation Factors. *Quality & Quantity*, *41*(5), 673–690. https://doi.org/10.1007/s11135-006-9018-6

O'Connor, T. G., Heron, J., Golding, J., Glover, V., & the AL SPAC Study Team. (2003). Maternal antenatal anxiety and behavioural/emotional problems in children: A test of a programming hypothesis: Genetics and Neurobiology Special Section: Maternal antenatal

anxiety. *Journal of Child Psychology and Psychiatry*, *44*(7), 1025–1036. https://doi.org/10.1111/1469-7610.00187

O'Donnell, E. (2021). *DNA Methylation Mediates the Association Between Early Life Adversity and Childhood Depression* [PhD Thesis]. ResearchSpace@ Auckland.

O'Hare, K., Watkeys, O., Whitten, T., Dean, K., Laurens, K. R., Harris, F., Carr, V. J., & Green, M. J. (2022). Cumulative environmental risk in early life is associated with mental disorders in childhood. *Psychological Medicine*, 1–10.

Oladeji, B. D., Makanjuola, V. A., & Gureje, O. (2010). Family-related adverse childhood experiences as risk factors for psychiatric disorders in Nigeria. *The British Journal of Psychiatry*, *196*(3), 186–191.

Olino, T. M., Dougherty, L. R., Bufferd, S. J., Carlson, G. A., & Klein, D. N. (2014). Testing Models of Psychopathology in Preschool-aged Children Using a Structured Interview-based Assessment. *Journal of Abnormal Child Psychology*, *42*(7), 1201–1211. https://doi.org/10.1007/s10802-014-9865-x

Oltmanns, J. R., Smith, G. T., Oltmanns, T. F., & Widiger, T. A. (2018). General Factors of Psychopathology, Personality, and Personality Disorder: Across Domain Comparisons. *Clinical Psychological Science*, *6*(4), 581–589. https://doi.org/10.1177/2167702617750150

Ortuno-Sierra, J., Chocarro, E., Fonseca-Pedrero, E., i Riba, S. S., & Muñiz, J. (2015). The assessment of emotional and Behavioural problems: Internal structure of The Strengths and Difficulties Questionnaire. *International Journal of Clinical and Health Psychology*, *15*(3), 265–273.

Palumbo, I. M., Patrick, C. J., & Latzman, R. D. (2023). Psychopathology in children: The transdiagnostic contribution of affiliative capacity and inhibitory control. *Development and Psychopathology*, *35*(4), 1627–1642.

Patalay, P., Fonagy, P., Deighton, J., Belsky, J., Vostanis, P., & Wolpert, M. (2015). A general psychopathology factor in early adolescence. *The British Journal of Psychiatry*, *207*(1), 15–22.

Paus, T., Keshavan, M., & Giedd, J. N. (2008). Why do many psychiatric disorders emerge during adolescence? *Nature Reviews Neuroscience*, *9*(12), 947–957.

Peacock, P. J., Lewis, G., Northstone, K., & Wiles, N. J. (2011). Childhood diet and behavioural problems: Results from the ALSPAC cohort. *European Journal of Clinical Nutrition*, *65*(6), 720–726.

Pearson, J., Tarabulsy, G. M., & Bussières, E.-L. (2015). Foetal programming and cortisol secretion in early childhood: A meta-analysis of different programming variables. *Infant Behavior and Development*, *40*, 204–215.

Petanjek, Z., Judaš, M., Šimić, G., Rašin, M. R., Uylings, H. B., Rakic, P., & Kostović, I. (2011). Extraordinary neoteny of synaptic spines in the human prefrontal cortex. *Proceedings of the National Academy of Sciences*, *108*(32), 13281–13286.

Pettersson, E., Lahey, B. B., Larsson, H., & Lichtenstein, P. (2018). Criterion validity and utility of the general factor of psychopathology in childhood: Predictive associations with independently measured severe adverse mental health outcomes in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry*, *57*(6), 372–383.

Peverill, M., Dirks, M. A., Narvaja, T., Herts, K. L., Comer, J. S., & McLaughlin, K. A. (2021). Socioeconomic status and child psychopathology in the United States: A meta-analysis of population-based studies. *Clinical Psychology Review*, *83*, 101933.

Phillips, E. M., Brock, R. L., James, T. D., Nelson, J. M., Espy, K. A., & Nelson, T. D. (2022). Empirical support for a dual process model of the p-factor: Interaction effects between preschool executive control and preschool negative emotionality on general psychopathology. *Journal of Psychopathology and Clinical Science*, *131*(8), 817.

Phillips, E. M., Brock, R. L., James, T. D., Nelson, J. M., Mason, W. A., Espy, K. A., & Nelson, T. D. (2023). Does preschool executive control mediate the impact of early environmental unpredictability and deprivation on the general factor of psychopathology a decade later? *Journal of Child Psychology and Psychiatry*.

Phillips, R. G., & LeDoux, J. E. (1992). Differential contribution of amygdala and hippocampus to cued and contextual fear conditioning. *Behavioral Neuroscience*, *106*(2), 274.

Polier, G. G., Vloet, T. D., Herpertz-Dahlmann, B., Laurens, K. R., & Hodgins, S. (2012). Comorbidity of conduct disorder symptoms and internalising problems in children: Investigating a community and a clinical sample. *European Child & Adolescent Psychiatry*, *21*(1), 31–38. https://doi.org/10.1007/s00787-011-0229-6

Pollak, S. D., Messner, M., Kistler, D. J., & Cohn, J. F. (2009). Development of perceptual expertise in emotion recognition. *Cognition*, *110*(2), 242–247.

Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Developmental Psychology*, *38*(5), 784.

Pollak, S. D., & Smith, K. E. (2021). Thinking clearly about biology and childhood adversity: Next steps for continued progress. *Perspectives on Psychological Science*, *16*(6), 1473–1477.

Potter, L. C., Morris, R., Hegarty, K., García-Moreno, C., & Feder, G. (2021). Categories and health impacts of intimate partner violence in the World Health Organization multi-country study on women's health and domestic violence. *International Journal of Epidemiology*, *50*(2), 652–662.

Rakesh, D., Cropley, V., Zalesky, A., Vijayakumar, N., Allen, N. B., & Whittle, S. (2021). Neighborhood disadvantage and longitudinal brain-predicted-age trajectory during adolescence. *Developmental Cognitive Neuroscience*, *51*, 101002.

Raposo, S. M., Mackenzie, C. S., Henriksen, C. A., & Afifi, T. O. (2014). Time does not heal all wounds: Older adults who experienced childhood adversities have higher odds of mood,

anxiety, and personality disorders. *The American Journal of Geriatric Psychiatry*, *22*(11), 1241–1250.

Raymond, C., Marin, M.-F., Majeur, D., & Lupien, S. (2018). Early child adversity and psychopathology in adulthood: HPA axis and cognitive dysregulations as potential mechanisms. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *85*, 152–160.

Reise, S. P., & Haviland, M. G. (2005). Item Response Theory and the Measurement of Clinical Change. *Journal of Personality Assessment*, *84*(3), 228–238. https://doi.org/10.1207/s15327752jpa8403_02

Reise, S. P., Moore, T. M., & Haviland, M. G. (2010). Bifactor Models and Rotations: Exploring the Extent to Which Multidimensional Data Yield Univocal Scale Scores. *Journal of Personality Assessment*, *92*(6), 544–559. https://doi.org/10.1080/00223891.2010.496477

Reise, S. P., Scheines, R., Widaman, K. F., & Haviland, M. G. (2013). Multidimensionality and Structural Coefficient Bias in Structural Equation Modeling: A Bifactor Perspective. *Educational and Psychological Measurement*, *73*(1), 5–26. https://doi.org/10.1177/0013164412449831

Rhoades, B. L., Greenberg, M. T., & Domitrovich, C. E. (2009). The contribution of inhibitory control to preschoolers' social–emotional competence. *Journal of Applied Developmental Psychology*, *30*(3), 310–320.

Rijlaarsdam, J., Cosin-Tomas, M., Schellhas, L., Abrishamcar, S., Malmberg, A., Neumann, A., Felix, J. F., Sunyer, J., Gutzkow, K. B., & Grazuleviciene, R. (2023). DNA methylation and general psychopathology in childhood: An epigenome-wide meta-analysis from the PACE consortium. *Molecular Psychiatry*, *28*(3), 1128–1136.

Riley, E. N., Atkinson, E. A., & Smith, G. T. (2019). Alcohol consumption as part of the p factor of general psychopathology. *ALCOHOLISM-CLINICAL AND EXPERIMENTAL RESEARCH*, *43*, 200A-200A.

Robinaugh, D. J., Hoekstra, R. H., Toner, E. R., & Borsboom, D. (2020). The network approach to psychopathology: A review of the literature 2008–2018 and an agenda for future research. *Psychological Medicine*, *50*(3), 353–366.

Rodriguez, A., Reise, S. P., & Haviland, M. G. (2016). Applying Bifactor Statistical Indices in the Evaluation of Psychological Measures. *Journal of Personality Assessment*, *98*(3), 223–237. https://doi.org/10.1080/00223891.2015.1089249

Rodriguez-Seijas, C., Eaton, N. R., & Krueger, R. F. (2015). How transdiagnostic factors of personality and psychopathology can inform clinical assessment and intervention. *Journal of Personality Assessment*, *97*(5), 425–435.

Ros, R., & Graziano, P. A. (2020). A transdiagnostic examination of self-regulation: Comparisons across preschoolers with ASD, ADHD, and typically developing children. *Journal of Clinical Child & Adolescent Psychology*, *49*(4), 493–508.

Rosen, M. L., Meltzoff, A. N., Sheridan, M. A., & McLaughlin, K. A. (2019). Distinct aspects of the early environment contribute to associative memory, cued attention, and memory-guided attention: Implications for academic achievement. *Developmental Cognitive Neuroscience*, *40*, 100731.

Rosenfield, P. J., Jiang, D., & Pauselli, L. (2022). Childhood adversity and psychotic disorders: Epidemiological evidence, theoretical models and clinical considerations. *Schizophrenia Research*, *247*, 55–66.

Ross, L. T., Hood, C. O., & Short, S. D. (2016). Unpredictability and Symptoms of Depression and Anxiety. *Journal of Social and Clinical Psychology*, *35*(5), 371–385. https://doi.org/10.1521/jscp.2016.35.5.371

Rudd, G., Meissel, K., & Meyer, F. (2023). Measuring Childhood Exposure to Neighbourhood Deprivation at the Macro-and Micro-level in Aotearoa New Zealand. *Child Indicators Research*, 1–26.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. *Annals of the Academy of Medicine, Singapore*, *8*(3), 324–338.

Rutter, M. (1981). Stress, coping and development: Some issues and some questions. *Journal of Child Psychology and Psychiatry*, *22*(4), 323–356.

Sallis, H., Szekely, E., Neumann, A., Jolicoeur-Martineau, A., Van IJzendoorn, M., Hillegers, M., Greenwood, C. M., Meaney, M. J., Steiner, M., & Tiemeier, H. (2019). General psychopathology, internalising and externalising in children and functional outcomes in late adolescence. *Journal of Child Psychology and Psychiatry*, *60*(11), 1183–1190.

Salmond, C., & Crampton, P. (2002). Heterogeneity of deprivation within very small areas. *Journal of Epidemiology & Community Health*, *56*(9), 669–670.

Salmond, C., Crampton, P., King, P., & Waldegrave, C. (2006). NZiDep: A New Zealand index of socioeconomic deprivation for individuals. *Social Science & Medicine*, *62*(6), 1474–1485.

Saxbe, D., Khoddam, H., Piero, L. D., Stoycos, S. A., Gimbel, S. I., Margolin, G., & Kaplan, J. T. (2018). Community violence exposure in early adolescence: Longitudinal associations with hippocampal and amygdala volume and resting state connectivity. *Developmental Science*, *21*(6), e12686.

Schaefer, J. D., Moffitt, T. E., Arseneault, L., Danese, A., Fisher, H. L., Houts, R., Sheridan, M. A., Wertz, J., & Caspi, A. (2018). Adolescent Victimization and Early-Adult Psychopathology: Approaching Causal Inference Using a Longitudinal Twin Study to Rule Out Noncausal Explanations. *Clinical Psychological Science*, *6*(3), 352–371. https://doi.org/10.1177/2167702617741381

Schäfer, J. L., McLaughlin, K. A., Manfro, G. G., Pan, P., Rohde, L. A., Miguel, E. C., Simioni, A., Hoffmann, M. S., & Salum, G. A. (2023). Threat and deprivation are associated with distinct aspects of cognition, emotional processing, and psychopathology in children and adolescents. *Developmental Science*, *26*(1), e13267.

Schroeder, A., Slopen, N., & Mittal, M. (2018). Accumulation, timing, and duration of early childhood adversity and behavior problems at age 9. *Journal of Clinical Child & Adolescent Psychology*.

Scopel Hoffmann, M., Moore, T. M., Kvitko Axelrud, L., Tottenham, N., Zuo, X.-N., Rohde, L. A., Milham, M. P., Satterthwaite, T. D., & Salum, G. A. (2022). Reliability and validity of bifactor models of dimensional psychopathology in youth. *Journal of Psychopathology and Clinical Science*, *131*(4), 407.

Sheridan, M. A., & McLaughlin, K. A. (2014). Dimensions of early experience and neural development: Deprivation and threat. *Trends in Cognitive Sciences*, *18*(11), 580–585.

Sheridan, M. A., & McLaughlin, K. A. (2016). Neurobiological models of the impact of adversity on education. *Current Opinion in Behavioral Sciences*, *10*, 108–113.

Sheridan, M. A., & McLaughlin, K. A. (2022). Introduction to the special issue on childhood adversity and neurodevelopment. *Developmental Cognitive Neuroscience*, *54*.

Sheridan, M. A., Peverill, M., Finn, A. S., & McLaughlin, K. A. (2017). Dimensions of childhood adversity have distinct associations with neural systems underlying executive functioning. *Development and Psychopathology*, *29*(5), 1777–1794.

Sheridan, M. A., Shi, F., Miller, A. B., Sahali, C., & McLaughlin, K. A. (2020). Network structure reveals clusters of associations between childhood adversities and development outcomes. *Developmental Science*, *23*(5), e12934. https://doi.org/10.1111/desc.12934

Sheridan, M. A., Shi, F., Miller, A. B., Salhi, C., & McLaughlin, K. A. (2020). Network structure reveals clusters of associations between childhood adversities and development outcomes. *Developmental Science*, *23*(5), e12934.

Shonkoff, J. P., Garner, A. S., Child, C. on P. A. of, Family Health, Dependent Care, Developmental, S. on, Pediatrics, B., Siegel, B. S., Dobbins, M. I., Earls, M. F., Garner, A. S., McGuinn, L., Pascoe, J., & Wood, D. L. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*(1), e232–e246.

Shors, T. J., Anderson, M. L., Curlik Ii, D. M., & Nokia, M. S. (2012). Use it or lose it: How neurogenesis keeps the brain fit for learning. *Behavioural Brain Research*, *227*(2), 450–458.

Short, M. A., Blunden, S., Rigney, G., Matricciani, L., Coussens, S., Reynolds, C. M., & Galland, B. (2018). Cognition and objectively measured sleep duration in children: A systematic review and meta-analysis. *Sleep Health*, *4*(3), 292–300.

Silverman, M. N., & Sternberg, E. M. (2012). Glucocorticoid regulation of inflammation and its functional correlates: From HPA axis to glucocorticoid receptor dysfunction. *Annals of the New York Academy of Sciences*, *1261*(1), 55–63.

Simpson, J. A., Griskevicius, V., Kuo, S. I., Sung, S., & Collins, W. A. (2012). Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. *Developmental Psychology*, *48*(3), 674.

Skar, A.-M. S., Jensen, T. K., & Harpviken, A. N. (2021). Who Reports What? A Comparison of Child and Caregivers´ Reports of Child Trauma Exposure and Associations to Post-Traumatic Stress Symptoms and Functional Impairment in Child and Adolescent Mental Health Clinics. *Research on Child and Adolescent Psychopathology*, *49*(7), 919–934. https://doi.org/10.1007/s10802-021-00788-y

Slopen, N., McLaughlin, K. A., & Shonkoff, J. P. (2014). Interventions to improve cortisol regulation in children: A systematic review. *Pediatrics*, *133*(2), 312–326.

Slopen, N., Meyer, C., & Williams, D. R. (2018). Cumulative stress and health. *The Oxford Handbook of Integrative Health Science*, 75–93.

Slopen, N., Williams, D. R., Seedat, S., Moomal, H., Herman, A., & Stein, D. J. (2010). Adversities in childhood and adult psychopathology in the South Africa Stress and Health Study: Associations with first-onset DSM-IV disorders. *Social Science & Medicine*, *71*(10), 1847–1854.

Slykerman, R. F., Budd, C., Thompson, J. M., Bekker, M., Buckley, J., Wall, C., Murphy, R., Mitchell, E. A., & Waldie, K. E. (2020). Physical activity, sleep, body mass index, and associated risk of behavioral and emotional problems in childhood. *Journal of Developmental & Behavioral Pediatrics*, *41*(3), 187–194.

Slykerman, R. F., Thompson, J. Md., Coomarasamy, C., Wall, C. R., Waldie, K. E., Murphy, R., & Mitchell, E. A. (2020). Early adolescent physical activity, sleep and symptoms of depression at 16 years of age. *Acta Paediatrica*, *109*(7), 1394–1399. https://doi.org/10.1111/apa.15140

Slykerman, R. F., Thompson, J., Waldie, K., Murphy, R., Wall, C., & Mitchell, E. A. (2015). Maternal stress during pregnancy is associated with moderate to severe depression in 11-year-old children. *Acta Paediatrica*, *104*(1), 68–74. https://doi.org/10.1111/apa.12787

Smith, K. E., & Pollak, S. D. (2021). Rethinking concepts and categories for understanding the neurodevelopmental effects of childhood adversity. *Perspectives on Psychological Science*, *16*(1), 67–93.

Snyder, H. R., Young, J. F., & Hankin, B. L. (2017). Strong homotypic continuity in common psychopathology-, internalizing-, and externalizing-specific factors over time in adolescents. *Clinical Psychological Science*, *5*(1), 98–110.

Snyder, H. R., Young, J. F., & Hankin, B. L. (2019). Chronic Stress Exposure and Generation Are Related to the P-Factor and Externalizing Specific Psychopathology in Youth. *Journal of*

Clinical Child & Adolescent Psychology, *48*(2), 306–315. https://doi.org/10.1080/15374416.2017.1321002

Southward, M. W., Cheavens, J. S., & Coccaro, E. F. (2023). Defining the p-factor: An empirical test of five leading theories. *Psychological Medicine*, *53*(7), 2732–2743.

Sprooten, E., Franke, B., & Greven, C. U. (2022). The P-factor and its genomic and neural equivalents: An integrated perspective. *Molecular Psychiatry*, *27*(1), 38–48.

Stein, C. R., Sheridan, M. A., Copeland, W. E., Machlin, L. S., Carpenter, K. L. H., & Egger, H. L. (2022). Association of adversity with psychopathology in early childhood: Dimensional and cumulative approaches. *Depression and Anxiety*, *39*(6), 524–535. https://doi.org/10.1002/da.23269

Struck, S., Stewart-Tufescu, A., Asmundson, A. J., Asmundson, G. G., & Afifi, T. O. (2021). Adverse childhood experiences (ACEs) research: A bibliometric analysis of publication trends over the first 20 years. *Child Abuse & Neglect*, *112*, 104895.

Stucky, B. D., & Edelen, M. O. (2014). Using hierarchical IRT models to create unidimensional measures from multidimensional data. *Handbook of Item Response Theory Modeling*, 183–206.

Suh, B. (2022). *The Effects of Adverse Childhood Experiences and Resilience on Child Development: Future Directions in Research, Practice, and Policy* [PhD Thesis]. Arizona State University.

Sumner, J. A., Colich, N. L., Uddin, M., Armstrong, D., & McLaughlin, K. A. (2019). Early experiences of threat, but not deprivation, are associated with accelerated biological aging in children and adolescents. *Biological Psychiatry*, *85*(3), 268–278.

Syed, E. U., Hussein, S. A., Azam, S. I., & Khan, A. G. (2009a). Comparison of Urdu version of Strengths and Difficulties Questionnaire (SDQ) and the Child Behaviour Check List (CBCL)

amongst primary school children in Karachi. *Journal of the College of Physicians and Surgeons Pakistan*, *19*(6), 375.

Syed, E. U., Hussein, S. A., Azam, S. I., & Khan, A. G. (2009b). Comparison of Urdu version of Strengths and Difficulties Questionnaire (SDQ) and the Child Behaviour Check List (CBCL) amongst primary school children in Karachi. *Journal of the College of Physicians and Surgeons Pakistan*, *19*(6), 375.

Szepsenwol, O., Griskevicius, V., Simpson, J. A., Young, E. S., Fleck, C., & Jones, R. E. (2017). The effect of predictable early childhood environments on sociosexuality in early adulthood. *Evolutionary Behavioral Sciences*, *11*(2), 131.

Tackett, J. L., Lahey, B. B., Van Hulle, C., Waldman, I., Krueger, R. F., & Rathouz, P. J. (2013). Common genetic influences on negative emotionality and a general psychopathology factor in childhood and adolescence. *Journal of Abnormal Psychology*, *122*(4), 1142.

Theunissen, G., D'Souza, S., Peterson, E. R., Walker, C., Morton, S. M., & Waldie, K. E. (2022). Prenatal determinants of depressive symptoms in childhood: Evidence from Growing Up in New Zealand. *Journal of Affective Disorders*, *302*, 41–49.

Torvik, F. A., Eilertsen, E. M., McAdams, T. A., Gustavson, K., Zachrisson, H. D., Brandlistuen, R., Gjerde, L. C., Havdahl, A., Stoltenberg, C., & Ask, H. (2020). Mechanisms linking parental educational attainment with child ADHD, depression, and academic problems: A study of extended families in The Norwegian Mother, Father and Child Cohort Study. *Journal of Child Psychology and Psychiatry*, *61*(9), 1009–1018.

Trautmann, S., Wittgens, C., Muehlhan, M., & Kanske, P. (2022). The role of socio-affective and socio-cognitive mechanisms in the processing of witnessed traumatic events. *Frontiers in Psychiatry*, *13*, 320.

Tsukayama, E., Duckworth, A. L., & Kim, B. (2013). Domain-specific impulsivity in school-age children. *Developmental Science*, *16*(6), 879–893. https://doi.org/10.1111/desc.12067

Tyrka, A. R., Parade, S. H., Price, L. H., Kao, H.-T., Porton, B., Philip, N. S., Welch, E. S., & Carpenter, L. L. (2016). Alterations of mitochondrial DNA copy number and telomere length with early adversity and psychopathology. *Biological Psychiatry*, *79*(2), 78–86.

Uddin, L. Q. (2021). Cognitive and behavioural flexibility: Neural mechanisms and clinical considerations. *Nature Reviews Neuroscience*, *22*(3), 167–179.

Usacheva, M., Choe, D., Liu, S., Timmer, S., & Belsky, J. (2022). Testing the empirical integration of threat-deprivation and harshness-unpredictability dimensional models of adversity. *Development and Psychopathology*, *34*(2), 513–526.

van Bork, R., Epskamp, S., Rhemtulla, M., Borsboom, D., & van der Maas, H. L. (2017). What is the p-factor of psychopathology? Some risks of general factor modeling. *Theory & Psychology*, *27*(6), 759–773.

Van De Looij-Jansen, P. M., Goedhart, A. W., De Wilde, E. J., & Treffers, P. D. A. (2011). Confirmatory factor analysis and factorial invariance analysis of the adolescent self-report Strengths and Difficulties Questionnaire: How important are method effects and minor factors? *British Journal of Clinical Psychology*, *50*(2), 127–144. https://doi.org/10.1348/014466510X498174

van Dulmen, M. H., & Egeland, B. (2011). Analyzing multiple informant data on child and adolescent behavior problems: Predictive validity and comparison of aggregation procedures. *International Journal of Behavioral Development*, *35*(1), 84–92.

Verbeek, T., Bockting, C. L., van Pampus, M. G., Ormel, J., Meijer, J. L., Hartman, C. A., & Burger, H. (2012). Postpartum depression predicts offspring mental health problems in adolescence independently of parental lifetime psychopathology. *Journal of Affective Disorders*, *136*(3), 948–954.

Vogel, S. C., Perry, R. E., Brandes-Aitken, A., Braren, S., & Blair, C. (2021). Deprivation and threat as developmental mediators in the relation between early life socioeconomic status and

executive functioning outcomes in early childhood. *Developmental Cognitive Neuroscience*, *47*, 100907.

Wolf, S., & Suntheimer, N. M. (2019). A dimensional risk approach to assessing early adversity in a national sample. *Journal of Applied Developmental Psychology*, *62*, 270–281.

Wade, M., Fox, N. A., Zeanah, C. H., Nelson, C. A., & Drury, S. S. (2020). Telomere length and psychopathology: Specificity and direction of effects within the Bucharest Early Intervention Project. *Journal of the American Academy of Child & Adolescent Psychiatry*, *59*(1), 140–148.

Wade, M., Plamondon, A., & Jenkins, J. M. (2021). A Family Socialization Model of Transdiagnostic Risk for Psychopathology in Preschool Children. *Research on Child and Adolescent Psychopathology*, *49*(8), 975–988. https://doi.org/10.1007/s10802-021-00789-x

Wade, M., Wright, L., & Finegold, K. E. (2022). The effects of early life adversity on children's mental health and cognitive functioning. *Translational Psychiatry*, *12*(1), 244.

Wade, M., Zeanah, C. H., Fox, N. A., & Nelson, C. A. (2020). Global deficits in executive functioning are transdiagnostic mediators between severe childhood neglect and psychopathology in adolescence. *Psychological Medicine*, *50*(10), 1687–1694.

Waldie, K. E., Peterson, E. R., D'Souza, S., Underwood, L., Pryor, J. E., Carr, P. A., Grant, C., & Morton, S. M. (2015). Depression symptoms during pregnancy: Evidence from growing up in New Zealand. *Journal of Affective Disorders*, *186*, 66–73.

Waldman, I. D., & Poore, H. E. (2023). Evaluating alternative models of youth externalizing using quantitative genetic analyses. *Journal of Psychopathology and Clinical Science*, *132*(7), 833.

Waldman, I. D., Poore, H. E., van Hulle, C., Rathouz, P. J., & Lahey, B. B. (2016). External validity of a hierarchical dimensional model of child and adolescent psychopathology: Tests using confirmatory factor analyses and multivariate behavior genetic analyses. *Journal of Abnormal Psychology*, *125*(8), 1053.

Wallander, J. L., Berry, S., Carr, P. A., Peterson, E. R., Waldie, K. E., Marks, E., D'Souza, S., & Morton, S. M. (2019). Patterns of exposure to cumulative risk through age 2 and associations with problem behaviors at age 4.5: Evidence from growing up in New Zealand. *Journal of Abnormal Child Psychology*, *47*, 1277–1288.

Wallander, J. L., Berry, S., Carr, P. A., Peterson, E. R., Waldie, K. E., Marks, E., D'Souza, S., & Morton, S. M. (2021). Patterns of risk exposure in first 1,000 days of life and health, behavior, and education-related problems at age 4.5: Evidence from Growing Up in New Zealand, a longitudinal cohort study. *BMC Pediatrics*, *21*(1), 285.

Walker C.G, Fletcher B.D, Lai, H., Marks, E.J., Yao, E., Evans, R. J., Tait, J., Grant, M., Crosby, K., Gerritsen, S., Pillai, A., Napier, C., Morton, S.M.B., Paine, S.J. (2023) DCW12: Data Release 2023 Data User Guide. Auckland: Growing Up in New Zealand. Available from: www.growingup.co.nz

Walsh, M. C., Joyce, S., Maloney, T., & Vaithianathan, R. (2019). Adverse childhood experiences and school readiness outcomes: Results from the Growing Up in New Zealand study. *The New Zealand Medical Journal (Online)*, *132*(1493), 15–24.

Walsh, M. C., Joyce, S., Maloney, T., & Vaithianathan, R. (2020). Exploring the protective factors of children and families identified at highest risk of adverse childhood experiences by a predictive risk model: An analysis of the growing up in New Zealand cohort. *Children and Youth Services Review*, *108*, 104556.

Wang, X., Lu, J., Liu, Q., Yu, Q., Fan, J., Gao, F., Han, Y., Liu, X., Yao, R., & Zhu, X. (2022). Childhood experiences of threat and deprivation predict distinct depressive symptoms: A parallel latent growth curve model. *Journal of Affective Disorders*, *319*, 244–251.

Warren, S. M., & Barnett, M. A. (2020). Effortful Control Development in the Face of Harshness and Unpredictability. *Human Nature*, *31*(1), 68–87. https://doi.org/10.1007/s12110-019-09360-6

Watts, A. L., Lane, S. P., Bonifay, W., Steinley, D., & Meyer, F. A. C. (2020). Building Theories on Top of, and Not Independent of, Statistical Models: The Case of the *p* -factor. *Psychological Inquiry*, *31*(4), 310–320. https://doi.org/10.1080/1047840X.2020.1853476

Watts, A. L., Meyer, F. A., Greene, A. L., Wood, P. K., Trull, T. J., Steinley, D., & Sher, K. J. (2021). *Spurious empirical support for the p-factor arises with the inclusion of undiagnosed cases*. https://psyarxiv.com/4tazx/download?format=pdf

Watts, A. L., Poore, H. E., & Waldman, I. D. (2019). Riskier Tests of the Validity of the Bifactor Model of Psychopathology. *Clinical Psychological Science*, *7*(6), 1285–1303. https://doi.org/10.1177/2167702619855035

Weissman, D. G., Bitran, D., Miller, A. B., Schaefer, J. D., Sheridan, M. A., & McLaughlin, K. A. (2019a). Difficulties with emotion regulation as a transdiagnostic mechanism linking child maltreatment with the emergence of psychopathology. *Development and Psychopathology*, *31*(3), 899–915.

Weissman, D. G., Bitran, D., Miller, A. B., Schaefer, J. D., Sheridan, M. A., & McLaughlin, K. A. (2019b). Difficulties with emotion regulation as a transdiagnostic mechanism linking child maltreatment with the emergence of psychopathology. *Development and Psychopathology*, *31*(3), 899–915.

Weissman, D. G., Jenness, J. L., Colich, N. L., Miller, A. B., Sambrook, K. A., Sheridan, M. A., & McLaughlin, K. A. (2020). Altered neural processing of threat-related information in children and adolescents exposed to violence: A transdiagnostic mechanism contributing to the emergence of psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry*, *59*(11), 1274–1284.

Wesarg, C., Van Den Akker, A. L., Oei, N. Y., Hoeve, M., & Wiers, R. W. (2020). Identifying pathways from early adversity to psychopathology: A review on dysregulated HPA axis

functioning and impaired self-regulation in early childhood. *European Journal of Developmental Psychology*, *17*(6), 808–827.

White, T., van der Ende, J., & Nichols, T. E. (2019). Beyond Bonferroni revisited: Concerns over inflated false positive research findings in the fields of conservation genetics, biology, and medicine. *Conservation Genetics*, 20(4), 927–937. https://doi.org/10.1007/s10592-019-01178-0

Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, *62*(2), 71.

Wing, R., Gjelsvik, A., Nocera, M., & McQuaid, E. L. (2015). Association between adverse childhood experiences in the home and pediatric asthma. *Annals of Allergy, Asthma & Immunology*, *114*(5), 379–384.

Wintour, E. M., & Owens, J. A. (Eds.). (2006). *Early Life Origins of Health and Disease* (Vol. 573). Springer US. https://doi.org/10.1007/0-387-32632-4

Wright, A. G., Krueger, R. F., Hobbs, M. J., Markon, K. E., Eaton, N. R., & Slade, T. (2013). The structure of psychopathology: Toward an expanded quantitative empirical model. *Journal of Abnormal Psychology*, *122*(1), 281.

Yoon, Y., Eisenstadt, M., Lereya, S. T., & Deighton, J. (2023). Gender difference in the change of adolescents' mental health and subjective wellbeing trajectories. *European Child & Adolescent Psychiatry*, *32*(9), 1569–1578. https://doi.org/10.1007/s00787-022-01961-4

Young, E. S., Frankenhuis, W. E., & Ellis, B. J. (2020). Theory and measurement of environmental unpredictability. *Evolution and Human Behavior*, *41*(6), 550–556.

Young, E. S., Griskevicius, V., Simpson, J. A., Waters, T. E., & Mittal, C. (2018). Can an unpredictable childhood environment enhance working memory? Testing the sensitized-specialization hypothesis. *Journal of Personality and Social Psychology*, *114*(6), 891.

Zelazo, P. D. (2020). Executive Function and Psychopathology: A Neurodevelopmental Perspective. *Annual Review of Clinical Psychology*, *16*(1), 431–454. https://doi.org/10.1146/annurev-clinpsy-072319-024242

Zelazo, P. D., & Carlson, S. M. (2012). Hot and Cool Executive Function in Childhood and Adolescence: Development and Plasticity. *Child Development Perspectives*, *6*(4), 354–360. https://doi.org/10.1111/j.1750-8606.2012.00246.x