# Friendship stress buffering in young people with childhood adversity

König, M.

PhD Dissertation

# Friendship Stress Buffering

*in young people with childhood adversity*

**Maximilian König**

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# Friendship Stress Buffering in Young People with Childhood Adversity

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# Friendship Stress Buffering in Young People with Childhood Adversity

## Doctoral Dissertation

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according to the decision of the doctorate board

to be defended on Thursday, September 25, 2025
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by

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## Trigger Warning

This dissertation discusses childhood trauma, which may evoke distressing emotions or memories in some individuals.

If you find yourself struggling with the content discussed, please consider reaching out to a trusted friend, family member, or mental health professional for support and guidance.

Visit www.findahelpline.com to connect with a trained crisis counselor near you.

To my parents, Meike and Gerd, who inspire me,
my brothers, Robin and Leon, who empower me,
my husband, Fabian, who completes me.

You are my everything.

**Sometimes it is the people who no one imagines anything of who do the things that no one can imagine.**

The Imitation Game

This quote is often associated with the movie *The Imitation Game*, which depicts the life of the pioneering British mathematician and computer scientist Dr. Alan Turing. Dr. Turing played a crucial role in decrypting the German Enigma code during World War II and laid the foundation for modern computing. Reports suggest that Dr. Turing experienced social isolation and bullying in his formative years, and as an adult, he continued to face persecution, discrimination, and harassment due to his homosexuality up until his untimely death in 1954.

This quote shall serve as a reminder of our collective responsibility to foster an environment of openness and inclusivity. To embrace diversity and to recognize the inherent potential in every individual, regardless of their background, identity, or life circumstances. It beckons us to envision a society that challenges stereotypes, dismantles systemic barriers, and advocates for the creation of safe spaces where every individual is being given the opportunity to thrive.

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Childhood adversity is ubiquitous. Approximately 60% of all young people growing up worldwide are exposed to at least one form of childhood adversity by age 18 (Madigan et al., 2023). This includes often co-occurring experiences such as abuse or neglect, parental mental illness, severe poverty, bullying, or exposure to war (Brown et al., 2019; Dong et al., 2004). Chronic and repeated exposure to these toxic stressors has cascading life-altering consequences, leading to increased risks for psychopathology, cognitive deficits, chronic diseases, socioeconomic inequalities, transgressive behaviors, and premature mortality (Gilbert et al., 2009; Grummitt et al., 2021; Kessler et al., 2010). Furthermore, the societal costs are staggering, with adverse childhood experiences contributing to economic burdens and strained healthcare systems (K. Hughes et al., 2021; Peterson et al., 2018). That said, childhood adversity is a preventable public health issue, underscoring the importance to identify, understand, and address the factors that put young people at risk for and protect them from its detrimental effects. This dissertation focuses on the protective role of friendships in youth mental health following childhood adversity, contributing to a deeper mechanistic understanding of how these social relationships help buffer the negative effects of stress.

**Childhood Adversity**

Childhood adversity refers to stressful and potentially traumatic experiences during childhood or adolescence (before age 18) that represent a deviation from what is typically considered a normative environmental context (Cicchetti & Valentino, 2006; McLaughlin, 2016; Nelson & Gabard-Durnam, 2020). This encompasses a range of often co-occurring experiences, from maltreatment, such as exposure to abuse or neglect, to household challenges, which involve exposure to a caregiver experiencing issues such as substance misuse, domestic violence, or divorce, to various other forms of stressful experiences, which can include but is not limited to bullying, discrimination, natural disasters, and refugee or wartime experiences (Brown et al., 2019; K. Hughes et al., 2017; Nelson et al., 2020).

Childhood adversity is the product of several interconnected factors, including social, cultural, economic, environmental, and biological influences, occurring in every society around the world (Madigan et al., 2023; Sethi et al., 2013). Risk factors exist at multiple levels. On an individual level, examples include families with young, single caregivers from lower socioeconomic backgrounds with limited educational opportunities (Crouch et al., 2019; K. Hughes et al., 2017). At the community level, risks are associated with residing in socioeconomically disadvantaged neighborhoods (Kohen et al., 2008), while at the societal level, adverse outcomes may be influenced by cultural norms, such as the approval of physical punishment for disciplining children (Gershoff et al., 2018). Global prevalence estimates from 546,458 adults across 22 countries spanning all continents indicate that approximately six in ten individuals retrospectively self-

reported experiencing at least one form of adversity during childhood or adolescence, with higher rates of particularly severe adversity among those with a history of mental health conditions (47.5%), low-income households (40.5%), and minoritized racial/ethnic groups (26.6%) (Madigan et al., 2023). However, the actual prevalence of childhood adversity is likely higher, as most studies are conducted in the Global North, with many cases unrecorded due to underreporting or societal stigma (Kessler et al., 2009; Meinck et al., 2016; Stoltenborgh et al., 2013).

Two main approaches are currently used to operationalize childhood adversity: cumulative risk and dimensional models of adversity. The prevailing cumulative risk approach focuses on the total number of distinct adverse experiences a young person has encountered, summing these into a cumulative risk score where higher scores signal an increased likelihood of long-term negative health consequences, such as psychopathology (Evans et al., 2013; Felitti et al., 1998). This approach emphasizes stress dysregulation (i.e., altered psychological or physiological responses to stress) as the common, primary mechanism linking childhood adversity with later-life psychopathology (Evans & Kim, 2007; Evans et al., 2013).

More recently, dimensional models of adversity have gained traction as an alternative to the cumulative risk approach. These models aim to expand on the frequently invoked stress pathways by identifying additional mechanisms, particularly learning processes, through which distinct features of adversity shape psychopathology risk (McLaughlin & Sheridan, 2016). This dimensional approach assesses the frequency and severity of childhood adversity and focuses on three core underlying dimensions of experiences that are shared across different types of adversity: threat/harshness (involving harm or threat of harm to oneself and others), deprivation (involving absence of expected cognitive and social stimulation), and, more recently, unpredictability (involving spatial-temporal variation in threat) (Belsky et al., 2012; Berman et al., 2022; McLaughlin et al., 2021; Sheridan & McLaughlin, 2014).

Both approaches have unique strengths and contribute complementary insights (McLaughlin et al., 2021; K. E. Smith & Pollak, 2021). The cumulative risk approach offers a straightforward and widely applicable framework, highlighting the additive effect of adversity on later-life psychopathology. Meanwhile, the dimensional approach offers a more nuanced perspective, elucidating how specific features of adversity differentially affect mechanistic pathways that contribute to increased psychopathology risk. Given that dimensional models are evolving frameworks, further research is needed to refine and update these models based on new insights, particularly as their predictive accuracy regarding later-life functioning remains insufficiently understood (McLaughlin et al., 2021).

**Consequences of Childhood Adversity**

Childhood adversity is associated with a range of deleterious, far-reaching, and long-lasting health and developmental consequences (S. E. Fox et al., 2010; Shonkoff, 2012). It is considered a major contributor to both morbidity and premature mortality (Grummitt et al., 2021; Rod et al., 2020). A systematic review of 19 meta-analyses with more than 20 million participants estimated that childhood adversity accounted for approximately 15% of the total US mortality rate in 2019, translating to around 439,072 deaths (Grummitt et al., 2021). This high mortality rate was associated with several leading causes of death, including suicide attempts, cardiovascular disease, and cancer. Additionally, a systematic review of 35 studies investigating pediatric health outcomes associated with childhood adversity provided prospective evidence of delays in cognitive development, alongside heightened risks for conditions such as asthma, infections, somatic complaints, and sleep disruptions (Oh et al., 2018). To further synthesize evidence on the health risks associated with exposure to multiple childhood adversities, K. Hughes et al. (2017) calculated risk estimates for 23 different health-related outcomes, drawing from a sample of 253,719 participants. Their analysis revealed weak associations with physical inactivity, overweight or obesity, and diabetes, moderate associations with behaviors such as smoking and heavy alcohol use, as well as with conditions like cancer, cardiovascular, and respiratory diseases. Strong associations were identified with high-risk behaviors, including sexual risk-taking, problematic substance use, interpersonal and self-directed violence, and the development of mental health disorders (K. Hughes et al., 2017).

In 2019, the financial burden of health outcomes attributable to childhood adversity was estimated at approximately USD 581 billion in Europe and USD 748 billion in North America, representing 2.7% of Europe's and 3.5% of North America's gross domestic product (Bellis et al., 2019). By calculating the population-attributable fractions (PAFs), Bellis et al. (2019) assessed how much the incidence of a certain condition would be reduced if childhood adversity were eliminated. Notably, the PAFs for mental disorders were among the highest, with childhood adversity being attributed to around 30% of anxiety cases and 40% of depression cases in North America, as well as more than 25% of both conditions in Europe (Bellis et al., 2019).

Since the 1990s, research into the relationship between childhood adversity and the risk of psychopathology has surged (Evans et al., 2013; Felitti et al., 1998). Epidemiological studies consistently demonstrate that exposure to childhood adversity drastically elevates the risk of developing both internalizing (e.g., depression, anxiety) and externalizing (e.g., disruptive behaviors, substance abuse) psychopathology, with these effects often persisting throughout the lifespan (Clark et al., 2010; McLaughlin, 2016). For example, a large-scale survey

involving 51,945 adults across 21 low-, middle-, and high-income countries estimated that roughly one-third of all mental health disorders worldwide are attributable to childhood adversity (Kessler et al., 2010). Moreover, the likelihood of developing psychopathology increases substantially with cumulative exposure to childhood adversity (S. J. Lewis et al., 2021). A recent systematic review and meta-analysis highlighted that individuals exposed to multiple childhood adversities were 3.70 times more likely to develop anxiety, 4.74 times more likely to suffer from depression, and an alarmingly 37.48 times more likely to attempt suicide compared to those without a history of childhood adversity (K. Hughes et al., 2017). Furthermore, individuals who experience multiple childhood adversities also tend to experience more persistent and severe symptoms, alongside heightened resistance to treatment (McLaughlin, Green, et al., 2010; Nanni et al., 2012). Today, the effectiveness of interventions aimed at preventing or reducing childhood adversity remains modest at best (van IJzendoorn et al., 2020). One reason for this could be the cursory understanding of the underlying mechanisms that link childhood adversity exposure to multiple forms of psychopathology.

**Biological Embedding of Childhood Adversity**

Disruptions in stress response systems are thought to be a central mechanism by which childhood adversity becomes biologically embedded or "gets under the skin," ultimately leading to increased vulnerability to psychopathology (Berens et al., 2017; Hertzman, 2012; McLaughlin, Sheridan, et al., 2015).

Stress is the body's coordinated physiological and psychological response to perceived endogenous or exogenous threats or demands. It disrupts homeostatic balance and strains an individual's resources and ability to cope and recover (Goldstein & McEwen, 2002; Selye, 1955, 2013). While stress can be both salubrious and deleterious, its effects are determined by the intensity and duration of the stressor. Mild, infrequent, and short-lived (i.e., positive) stress responses are not only fundamental for survival but are also an essential part of healthy development as they promote adaptation, learning, and growth. However, strong, frequent, and sustained (i.e., toxic) stress responses result in a cumulative "wear and tear" on the body, known as allostatic load, with well-established harmful effects on physical and mental health across the lifespan (Dhabhar, 2014; McEwen, 1998).

Two key neurobiological systems regulate the body's response to stress and are critical in reestablishing homeostasis. The fast-acting sympathetic nervous system (SNS) releases epinephrine (adrenaline), which quickly mobilizes metabolic resources and elicits the fight-or-flight response. Meanwhile, the slower-acting hypothalamus-pituitary-adrenal (HPA) axis triggers the adrenal-driven production of glucocorticoids (mainly cortisol). In turn, glucocorticoids

regulate HPA axis activity, contributing to neural maturation, myelination, and neurogenesis, and serving as potent anti-inflammatory and immunosuppressive agents (Auphan et al., 1995; Gunnar & Quevedo, 2007; Lupien et al., 2009). Additionally, glucocorticoids are thought to exert their prolonged effects on physiology and behavior by influencing gene expression and accelerating epigenetic aging (de Kloet et al., 1996; Marini et al., 2020; Sapolsky et al., 2000; Zannas et al., 2015).

Strong, frequent, and sustained exposure to childhood adversity can exert long-lasting programming effects on the HPA axis, leading to either hyper- or hypo-activation in response to perceived threats (Agorastos et al., 2018; Berens et al., 2017; Lupien et al., 2009; Roberts & Lopez-Duran, 2019). Both chronically elevated and suppressed glucocorticoid levels are indicative of a dysregulated HPA axis. Hyper-reactivity indicates an acquired resistance to glucocorticoid negative feedback mechanisms, leading to heightened stress sensitivity (Danese & McEwen, 2012). In contrast, hypo-reactivity indicates an exaggerated suppression of the HPA axis, resulting in diminished stress sensitivity (Lovallo, 2013). Differential patters of glucocorticoid dysregulation may arise from various factors such as type and timing of adverse experiences, genetic predispositions, current age, or existing psychopathology, with both patterns being linked to negative health and developmental outcomes (Berens et al., 2017; Danese & McEwen, 2012).

Toxic early-life stress is thought to exert its pathogenic effects particularly during sensitive periods of brain development (Berens et al., 2017). These periods of elevated brain plasticity extend into the mid-to-late 20s (Sawyer et al., 2018), with region-specific maturational changes (e.g., synaptogenesis and synaptic pruning) occurring between childhood, adolescence, and early adulthood, creating windows of heightened vulnerability (Andersen, 2003; Foulkes & Blakemore, 2018). Frontolimbic regions, including the hippocampus, amygdala, anterior cingulate cortex, and prefrontal cortex, are particularly susceptible to adverse experiences due to their dense innervation with glucocorticoid receptors and their protracted developmental trajectory (Cohodes et al., 2021; Ioannidis et al., 2020). Adversity-induced alterations in frontolimbic structure and function are believed to play a central role in the biological embedding of childhood adversity, contributing to an increased risk for psychopathology (VanTieghem & Tottenham, 2018). Critical questions remain about how the timing, severity, type, controllability, and predictability of adversity exposure influence frontolimbic development and functioning, and how these effects may, in turn, predict the risk of psychopathology (Cohodes et al., 2021).

**Neurocognitive Adaptation and Poor Social Functioning Linking Childhood Adversity and Psychopathology**

Neuroendocrine stress dysregulation following childhood adversity may serve short-term adaptive purposes by aiding survival in highly stressful and threatening environments, but can become maladaptive in the long-term by disrupting a range of neurocognitive and social processes, eventually increasing latent vulnerability to multiple forms of psychopathology (McCrory et al., 2019; McLaughlin et al., 2020).

The neurocognitive social transactional model of psychiatric vulnerability (Figure 1) highlights how disruptions in key stress-mediating mechanisms link childhood adversity to social stress and heightened psychopathology risk (McCrory et al., 2022). Specifically, this model proposes that adversity-induced neurocognitive adaptation in domains such as threat-, reward-, and autobiographical memory processing might contribute to a social environment characterized by more stressful interpersonal experiences (i.e., stress generation) and fewer protective social relationships (i.e., social thinning), consequently increasing vulnerability to psychopathology.

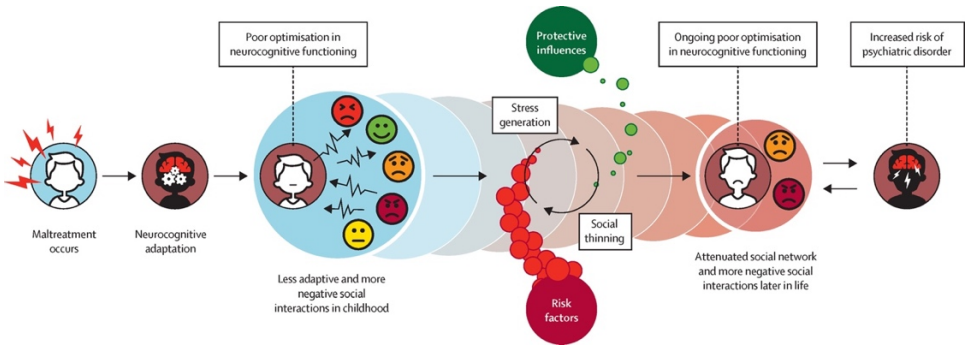


**Figure 1.** The neurocognitive social transactional model of psychiatric vulnerability. Reprinted from McCrory et al. (2022), with permission from Elsevier.

Disruptions in threat processing, including hypervigilance towards and avoidance of threat cues, may hold functional value in adverse environments by enhancing an individual's ability to rapidly detect and respond to potential dangers, thereby promoting safety. However, in less threatening or more normative environments, these biases may become maladaptive through facilitating social stress and increasing the risk for multiple forms of psychopathology, including conduct disorder, anxiety disorder, depression, and post-traumatic stress disorder (PTSD) (Blair & Zhang, 2020; Cisler & Koster, 2010; Etkin & Wager, 2007; Fales et al., 2008). One consistently observed pattern among individuals with childhood adversity, particularly those with threat-related experiences like physical abuse or

violence, is faster attentional engagement and altered neural responses in fronto-amygdala circuits to salient negative cues in the environment, such as angry facial expressions (McLaughlin, DeCross, et al., 2019; McLaughlin, Weissman, et al., 2019; Pollak et al., 2000). These threat processing biases may predispose them to experience interpersonal stress in ambiguous social situations, where they are more likely to respond aggressively or avoidantly to perceived social threats, which can undermine the development and maintenance of supportive social relationships (Dodge et al., 1990; Shackman & Pollak, 2014).

Disruptions in reward processing, including blunted responses to reward anticipation and receipt, may be adaptive in environments offering scares or unpredictable rewards. However, reductions in reward responsiveness may impede individuals from learning which behaviors lead to reward or from experiencing the emotional sensation of reward, which has been identified as a transdiagnostic marker of various forms of psychopathology, including depression, attention-deficit hyperactivity disorder, eating disorder, and schizophrenia (Aldridge-Waddon et al., 2020; Mackin et al., 2019; Sharma et al., 2016). Alterations in reward processing have frequently been observed among individuals with deprivation-related experiences (McLaughlin, DeCross, et al., 2019; McLaughlin, Weissman, et al., 2019; Sheridan et al., 2018). For example, deficits in approach motivation and blunted responses in fronto-striatal circuits during reward anticipation and receipt have been observed following both institutional rearing and neglect (Goff et al., 2013; Hanson, Hariri, et al., 2015). When early-life social interactions are either absent or lack rewarding qualities, long-term tendencies to trust people and expectations regarding the hedonic value of social relationships may be impacted, which will likely affect the formation of stable and supportive social networks (Pitula et al., 2017; Wismer Fries & Pollak, 2017). Additionally, a lack of motivation to follow rules and social norms may increase the risk of experiencing social stress through relational peer-victimization and bullying (Ke et al., 2022).

Disruptions in autobiographical memory processing, including overgeneral recall of single events, may serve as a coping strategy by helping individuals with childhood adversity avoid specific traumatic or distressing memories. However, alterations in how autobiographical memories are represented, recalled, and maintained have been linked to poor problem-solving abilities, negative self-representations, and an increased risk of depression and PTSD (Dalgleish & Werner-Seidler, 2014; Hallford et al., 2022; McCrory et al., 2017; Valentino et al., 2009). While adversity-specific patterns in neural responses during autobiographical memory retrieval are less well-documented, studies involving individuals with mixed adversity exposure have reported increased activation in hippocampal circuits during recall of negative, compared to positive, autobiographical memories (McCrory et al., 2017; McLaughlin, Weissman, et al.,

2019; Puetz et al., 2021). In the context of social functioning, a tendency to recall autobiographical memories in an overgeneral manner can limit access to detailed memory information necessary for navigating interpersonal challenges, such as conflicts with friends (Goddard et al., 1996). By impeding effective conflict resolution, these autobiographical memory patterns can prolong exposure to social stress and ultimately weaken social connections (Puetz et al., 2021; Sutherland & Bryant, 2008).

Together, neurocognitive adaptation following childhood adversity may offer short-term advantages in adverse environments by enhancing survival, but often result in long-term negative (social) consequences in more normative settings, increasing latent vulnerability to psychopathology (McCrory et al., 2022; McCrory & Viding, 2015). To guide prevention efforts and pinpoint specific, malleable intervention targets, future research needs to focus on identifying the neurocognitive systems most critical for predicting maladaptive social functioning and psychopathology risk in young people with childhood adversity. Having said that, not all individuals with childhood adversity are destined to develop psychopathology. Instead, some demonstrate what is referred to as resilient functioning, meaning they fare better than expected given their circumstances (Ioannidis et al., 2020). Resilient functioning following childhood adversity is thought to be facilitated by a diverse range of protective factors residing across multiple psychological, social, and neurobiological levels that help individuals adapt and recover following stress exposure (Fritz, de Graaff, et al., 2018; Ioannidis et al., 2020; Kalisch et al., 2019).

**Social Stress Buffering in Young People with Childhood Adversity**

One important protective factor is social support. Through alleviating the damaging psychological and neurobiological effects of (toxic) stress (Cohen & Wills, 1985; Horan & Widom, 2015), social support plays a critical role in preventing the onset and persistence of psychopathology in young people with childhood adversity (Li et al., 2022; Pine & Cohen, 2002; Trickey et al., 2012; Ungar et al., 2013).

Social stress buffering refers to the process by which the presence and availability of one or more supportive social partners, such as a primary caregiver, friend, teacher, or significant other, mitigate psychological perceptions of stress, dampen neurobiological responses to stress, and promote a faster recovery to baseline stress levels following threat exposure (Gunnar, 2017). For example, through dampening HPA axis activity and consequently lowering the release of glucocorticoids and pro-inflammatory markers into the bloodstream, social support is thought to decrease the physiological burden, or allostatic load, imposed on the body by stress exposure, thereby lowering psychopathology risk (Doan & Evans, 2011; Hennessy et al., 2009; Hostinar et al., 2014b). This

buffering effect occurs across the lifespan in diverse social settings, with its effectiveness influenced by past social experiences and the developmental stage of the recipient (Gunnar & Hostinar, 2015; Hennessy et al., 2009; Hostinar et al., 2014b). While the presence and availability of a supportive caregiver remains a potent stress buffer into late childhood, its effectiveness tends to diminish with the transition to adolescence and young adulthood as friends take on a more central role in providing emotional support and regulating stress (Gunnar & Hostinar, 2015; Gunnar et al., 2015).

Childhood is the developmental stage between infancy and adolescence, characterized by caregiver dependency and marked by significant physical, cognitive, language, and social-emotional growth (Black et al., 2017; Woodhead, 2009). Early research on social buffering of the HPA axis demonstrated that secure attachment relationships with primary caregivers can dampen stress-induced salivary cortisol increases in 2-year-olds (Gunnar et al., 1996). Similar stress buffering effects have been observed in the following years. For example, Seltzer et al. (2010) asked female children (aged 7-12 years) to complete an acute psychosocial stress task and found that following stress exposure, both physical and speech-only contact with their mothers significantly increased urinary levels of oxytocin, a neuropeptide known to inhibit stress-induced glucocorticoid secretion, while also reducing salivary cortisol levels. Research by Hostinar et al. (2015) has demonstrated that children (aged 9-11 years), but not adolescents (aged 15-16 years), exhibited caregiver buffering, as indicated by reduced salivary cortisol responses to acute psychosocial stress. Similarly, Gee et al. (2014) found evidence of maternal buffering in children (aged 4-10 years), but not in adolescents (aged 11-17 years), in the form of suppressed amygdala reactivity and improved affect-related behavioral regulation when viewing maternal compared to stranger stimuli. However, while it seems that caregivers become less central in facilitating emotion regulation and stress buffering after the onset of puberty, their presence and availability remains important, particularly for young people with childhood adversity. For example, Callaghan et al. (2019) found that both children (aged 6-10 years) and adolescents (aged 11-17 years) who had experienced institutional care prior to adoption and reported feeling more secure in their caregiver relationships exhibited reduced amygdala reactivity to caregiver cues, a protective mechanism against long-term anxiety symptoms.

The decreasing effectiveness of caregiver support in stress regulation coincides with key developmental changes typical of adolescence, including the onset of puberty, the maturation of frontolimbic circuits, and the increasing drive to seek independence from caregivers (Blakemore, 2008; Blakemore et al., 2010). Adolescence is the transitional stage between childhood and adulthood, characterized by biological growth and major social role transitions, and nowadays defined as the period between 10 and 24 years of age (Sawyer et al.,

2018). This sensitive period of social development is marked by more time spent with peers and less time spent with family (Lam et al., 2014). The developmental importance of peer companionship and intimacy becomes particularly apparent through dramatic changes in social behavior, such as the growing need for peer approval and the profound influence peers exert on decision-making, both in risky and prosocial contexts (Albert et al., 2013; Buhrmester & Furman, 1987; De Goede et al., 2009; Foulkes et al., 2018). Experiences of peer victimization during adolescence, such as peer rejection or (cyber)bullying, are prospective predictors of negative mental health outcomes (Bowes et al., 2015; Maurya et al., 2022; Prinstein & Aikins, 2004). However, in line with the friendship protection hypothesis (Boulton et al., 1999), high-quality friendship support can protect against future peer victimization and lower the risk of developing mental health problems (Bernasco et al., 2022; Cohen & Wills, 1985; Herman-Stahl & Petersen, 1996; Kendrick et al., 2012).

The availability of safe, stable, reciprocal, and supportive friendships is particularly important for young people with childhood adversity. Not only are those vulnerable young people more likely to experience peer victimization (Benedini et al., 2016; Widom et al., 2008), they are also more likely to victimize others (Fitton et al., 2020; Widom, 1989b). This cycle of victimization is thought to be fueled by adversity-induced neurocognitive adaptation, like altered threat processing, which can compromise an individual's ability to negotiate everyday social stress (Goemans et al., 2023). In turn, this heightened stress susceptibility potentiates mental health vulnerability (Gerin et al., 2019). However, just as not all young people with childhood adversity go on to develop psychopathology, not all will experience or engage in victimization. These resilient individuals likely benefit from protective factors, such as friendship support, that can help break this vicious cycle.

Friendship support has proven to buffer neurobiological stress responses in young people without childhood adversity (Gunnar, 2017; Gunnar & Hostinar, 2015). For example, two studies have demonstrated that following peer exclusion, those with higher levels of perceived social support (e.g., measured by the time spent with friends) exhibited diminished cortisol responses and lower neural activity in frontolimbic regions commonly implicated in responding to social distress (Eisenberger et al., 2007; C. L. Masten et al., 2012). Among young people with childhood adversity, research has demonstrated friendship buffering effects related to the emergence and progression of mental health problems (Powers et al., 2009; van Harmelen et al., 2016, 2021). Preliminary evidence related to friendship stress buffering in young people with childhood adversity suggests that individuals with high-quality friendship support or access to a highly responsive friend following acute psychosocial stress exhibited greater HPA axis recovery, as indicated by a faster return to baseline salivary cortisol levels (Calhoun et al.,

2014). Since disrupted neuroendocrine regulation is a marker of high allostatic load with known pathophysiological consequences (McEwen, 2000), these findings underscore the protective, stress-buffering potential of friendships. However, it remains to be investigated whether friendship support aids mental health and well-being in young people with childhood adversity through dampening psychological and neurobiological stress responses. Ultimately, a nuanced understanding of the stress-related mechanisms linking childhood adversity to later-life psychopathology, along with identifying protective factors that mitigate stress vulnerability, is essential for developing more targeted and effective prevention and intervention strategies serving young people with childhood adversity.

**Dissertation Outline**

This dissertation aims to enhance the mechanistic understanding of friendship stress buffering in young people with childhood adversity. Building on the neurocognitive social transactional model of psychiatric vulnerability (Figure 1; McCrory et al., 2022) and the social stress buffering framework (Gunnar, 2017), its primary goal is to identify psychological, cognitive, and neural stress-related pathways through which friendships mitigate the risk of psychopathology in this vulnerable population.

The **first part** of this dissertation addresses one of the most pervasive societal consequences of childhood adversity, commonly referred to as the cycle of victimization. Drawing on the cycle of violence hypothesis (Widom, 1989b), **Chapter 2** presents a literature review outlining the association between child maltreatment and the increased risk of perpetrating victimization both within and outside the family environment. To shed light on the mechanisms underpinning this cycle, the review detailed three maladaptive neurocognitive mechanisms that link maltreatment experiences with later-life victimization: (1) attentional bias to threat, (2) altered reward processing and feedback learning, and (3) emotion dysregulation. Importantly, the review concluded by emphasizing that not all individuals with a history of child maltreatment engage in victimization, indicating the presence of protective factors like social support that can help mitigate adversity-related vulnerabilities.

The **second part** of this dissertation focuses on the protective role of friendship support by examining psychological, cognitive, and neural mechanisms underlying friendship stress buffering in young people with childhood adversity. This part presents findings from one systematic literature review and three empirical studies: (1) the Resilience After Individual Stress Exposure (RAISE) study, (2) the Resilience after the COVID-19 Threat (REACT) study, and (3) the Towards Health and Resilience in Volatile Environments (THRIVE) study.

The **RAISE study** was a multilevel study at the University of Cambridge, UK, designed to examine psychological, cognitive, and neurobiological mechanisms and protective factors that facilitate resilient functioning in young people with childhood adversity (Moreno-López et al., 2021). Participants ($N$ = 102, $M_{age}$ = 22.24, 64.7% female) were recruited between August 2019 and March 2020 across Cambridgeshire, UK, from the general population through flyers, social media, and prior studies conducted by the University's Department of Psychiatry. Individuals were eligible to participate if they were aged between 16 and 26 years, able to speak, write, and understand English, had a body mass index between 18.5 and 29.9 kg/m², did not currently take medication (e.g., corticosteroids) likely to compromise data interpretation, had no MRI contraindications, and self-reported adverse experiences within the family environment before the age of 16. The RAISE study included three assessment timepoints, with data from the first two analyzed and presented in Chapter 4. At timepoint 1, participants remotely completed online self-report questionnaires assessing current (i.e., past two to four weeks) mental health and well-being, perceived friendship support, and retrospective childhood adversity ($N$ = 102, baseline sample). At timepoint 2, on average one month later, participants attended in-unit assessments at Addenbrooke's Hospital in Cambridge, UK ($n$ = 62, neuroimaging sample). This visit included, among other measures, functional magnetic resonance imaging (fMRI) during which participants completed the Montreal Imaging Stress Task (MIST) (Pruessner et al., 2008). The MIST is a widely used and well-validated acute psychosocial stress paradigm for fMRI that involves a mental arithmetic task performed under time constraints, with an artificially induced failure component and negative verbal feedback delivered by a trained member of the research team. The neuroimaging sample was smaller than the baseline sample due to a University-wide suspension of laboratory research activities in March 2020 in response to the COVID-19 outbreak. However, both groups did not significantly differ in key characteristics, such as age, gender, childhood adversity, or friendship quality. For a comprehensive description of the full study procedure, inclusion and exclusion criteria, and a complete list of all measures, see Moreno-López et al. (2021).

The **REACT study** was a longitudinal follow-up initiated after the COVID-19 outbreak to prospectively examine pandemic-related changes in psychosocial functioning among all RAISE participants ($N$ = 102) who were recruited between August 2019 and March 2020 and had consented to be recontacted for future studies (A. J. Smith et al., 2021). Participants were recruited remotely for three follow-up assessment timepoints. The first follow-up took place during the first national lockdown in the UK ($n$ = 79, April to May 2020), the second during a period of eased restrictions ($n$ = 77, July to August 2020), and the third during another phase of heightened restrictions ($n$ = 73, October to November 2020). Despite retention challenges, participants who completed follow-ups did not

differ significantly from the pre-pandemic baseline sample in key characteristics. At each follow-up, participants completed online self-report questionnaires, assessing variables such as current (i.e., past two weeks) mental health, perceived friendship support, and perceived stress. For a comprehensive description of the full study procedure and a complete list of all measures, see A. J. Smith et al. (2021).

The **THRIVE study** is an ongoing longitudinal study at Leiden University, the Netherlands, also designed to investigate psychological, cognitive, and neurobiological mechanisms and protective factors that facilitate resilient functioning in young people with childhood adversity. Participant recruitment commenced in October 2022 and involves outreach to the general population across the Netherlands through flyer distribution at schools and universities, general practitioner practices, shops, libraries, hospitals, out-patient care facilities, and social media. Eligible participants are aged 18 to 24 years, able to speak, write, and understand Dutch, self-report adverse experiences within or outside the family environment before the age of 18, and have not experienced severe depressive symptoms or suicidal thoughts in the two weeks prior to eligibility screening. Due to the ongoing nature of the study, Chapter 6 presents cross-sectional findings from the first 100 participants ($M_{age}$ = 21.23, 79.0 % female) who completed the initial two (of seven) assessment timepoints. At timepoint 1, participants remotely completed online self-report questionnaires assessing currently perceived friendship support and retrospective childhood adversity. At timepoint 2, on average one month later, participants attended in-unit assessments at the Leiden University Medical Center in Leiden, the Netherlands. This visit included, among other measures, the completion of online self-report questionnaires assessing current (i.e., past two weeks) mental health and perceived stress, as well as an adapted version of the Autobiographical Memory Task (J. M. Williams & Broadbent, 1986), asking participants to recall a memory of a situation or experience with a friend prompted by a positive or negative cue word. A protocol paper providing a detailed description of the study procedure, inclusion and exclusion criteria, and a complete list of all measures is expected to be published soon.

Before outlining the insights gained through the three empirical studies, **Chapter 3** presents a pre-registered systematic literature review that investigated whether greater friendship support reduces neural stress responses in young people with childhood adversity. Building on a growing body of evidence demonstrating the protective effects of friendship support on youth mental health following childhood adversity (van Harmelen et al., 2016, 2021), this review searched for empirical studies published in English through December 2021, involving young people (aged 10-24 years) with childhood adversity, and measures of friendship support and neural stress responses assessed using neuroimaging techniques.

Utilizing cross-sectional behavioral and neuroimaging data from the RAISE study, **Chapter 4** outlines whether greater friendship support predicts reduced neural stress responses in young people with childhood adversity. Specifically, this neuroimaging study examined three hypotheses: (1) whether more severe childhood adversity predicts lower friendship support, which in turn predicts poorer mental health and well-being; (2) whether acute psychosocial stress induced by the MIST elevates state anxiety and neural activity in seven predefined frontolimbic regions of interest (ROIs); and (3) whether greater friendship support predicts reduced frontolimbic ROI reactivity to stress. These hypotheses were tested using both a cumulative risk and dimensional approach, with the expectation that more severe threat-related adversity would be associated with heightened frontolimbic ROI reactivity to stress.

Utilizing longitudinal behavioral data from the REACT study, **Chapter 5** outlines whether greater friendship support predicts reduced mental health symptoms in young people with childhood adversity, both before and at three timepoints during the COVID-19 pandemic. Specifically, this prospective longitudinal study examined three hypotheses: (1) whether the COVID-19 outbreak predicts an overall increase in anxiety and depressive symptoms, along with a decrease in friendship support, with these trends expected to be exacerbated during lockdown periods; (2) whether more severe childhood adversity predicts reduced friendship support and heightened mental health symptoms during the pandemic; and (3) whether greater friendship support predicts fewer mental health symptoms. Additionally, the study explored perceived stress as a potential mechanism linking friendship support to mental health outcomes during the pandemic.

Utilizing cross-sectional behavioral data from the THRIVE study, **Chapter 6** outlines whether greater friendship support predicts autobiographical friendship memory specificity in young people with childhood adversity, as well as its associations with perceived stress and mental health. Specifically, this study examined three hypotheses: (1) whether greater friendship support predicts greater specificity of positive autobiographical friendship memories, lower perceived stress, and fewer depressive symptoms; (2) whether greater specificity of positive memories predicts lower perceived stress and fewer depressive symptoms; and (3) whether lower perceived stress predicts fewer depressive symptoms. To account for potential valence-specific effects, both positive and negative memory associations were analyzed.

This dissertation concludes with an executive summary and general discussion, synthesizing key findings across all chapters, outlining limitations, and proposing directions for future research (**Chapter 7**).

# Chapter 2

## Child Maltreatment and Victimization

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**Abstract**
It is estimated that up to 25% of all children growing up worldwide experience child maltreatment, making it a global emergency with substantial individual and public health consequences. This chapter addresses one of the most societally pervasive consequences of child maltreatment which is known as the "cycle of victimization". This concept depicts the increased risk of maltreated individuals to victimize others later in life, both within and outside the family environment. To understand the architecture of this victimization cycle, the chapter further sheds light on neurocognitive mechanisms aiding different forms of victimization and the buffering role of social support that could help break the cycle of victimization. Advancing our understanding of these complex and interrelated mechanisms will ultimately facilitate the design and implementation of more targeted early treatments and (preventive) interventions and support a move towards a safer society.

*Keywords:* child maltreatment, abuse, neglect, victimization, violence, intergenerational transmission, protective factors, social support

## Introduction

On July 22, 2011, a single perpetrator detonated a bomb at the government headquarters in Oslo before attacking a youth camp on Utøya island, killing 77 civilians, of whom nearly half were under the age of 18. While every mass shooting is different, an alarming number of perpetrators, including the one in Norway, have a documented history of child maltreatment (Densley & Peterson, 2017, 2019; Syse, 2014). For example, various sources have reported about the Utøya perpetrator's early-life involvement with child protection services (CPS) as well as incidences of physical abuse (e.g., being beaten by the mother), sexual abuse (e.g., inappropriate sexual behavior by the mother), emotional abuse (e.g., mother explicitly wishing death on him on multiple occasions), and physical neglect (e.g., being left unsupervised for a prolonged period at an early age) (Olsen, 2016; Syse, 2014).

The World Health Organization (WHO) describes child maltreatment as abusive or neglectful experiences that occur to children under the age of 18. Those experiences include "all forms of physical and/or emotional ill-treatment, sexual abuse, neglect or negligent treatment or commercial or other exploitation, resulting in actual or potential harm to the child's health, survival, development or dignity in the context of a relationship of responsibility, trust or power" (WHO 1999, pp. 14–15). Child maltreatment can be classified into five types: physical, emotional, and sexual abuse, and physical and emotional neglect. The global lifetime prevalence is estimated between 12 and 27% (Stoltenborgh et al., 2015), making child maltreatment a global emergency with substantial individual and public health consequences. Individuals who suffer child maltreatment are known to face life-long effects and challenges on different levels of their development, ranging from vulnerable cognitive and socio-emotional abilities to lower well-being and diminished mental and physical health (Norman et al., 2012; Vachon et al., 2015).

One of the most societally pervasive consequences of child maltreatment is the increased risk of victimizing others, both within and outside the family. Previous research on the intergenerational transmission of child maltreatment shows that offspring of parents who have personally experienced child maltreatment are at 2-3 times greater risk of experiencing maltreatment themselves compared to children of non-maltreated parents (Buisman et al., 2020; Madigan et al., 2019). In addition, the increased risk of (violent) crimes for individuals who have experienced child maltreatment has also been confirmed by empirical research (Salo et al., 2021).

This chapter outlines the link between child maltreatment and victimization within and outside the family environment whilst also shedding light on explanatory mechanisms and the buffering role of social support that could help

break the cycle of victimization (Figure 1). Advancing our understanding of these complex, interrelated pathways will ultimately facilitate the design and implementation of targeted early treatments and (preventive) interventions and support a move towards a safer society.

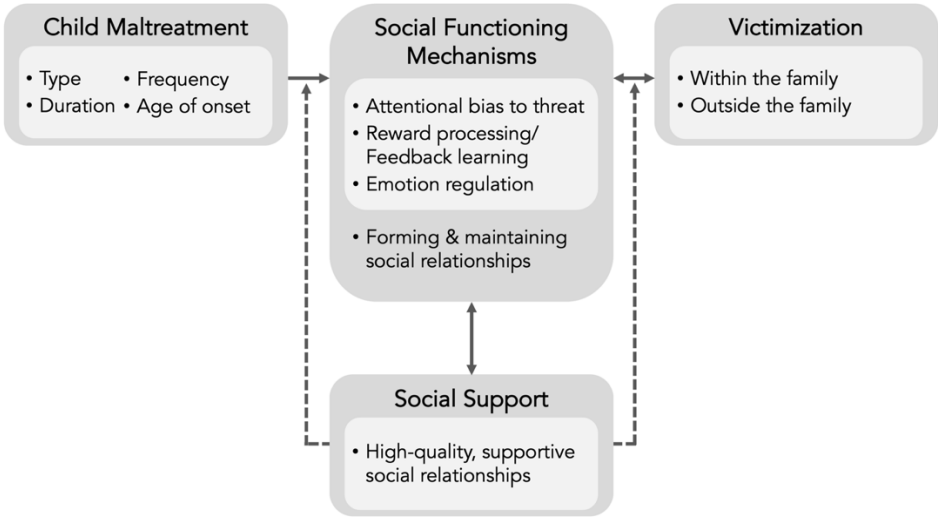


**Figure 1.** Social functioning mechanisms linking child maltreatment to victimization within and outside the family environment. Solid lines represent direct associations between maltreatment characteristics (e.g., type, duration, frequency, and age of onset) and explanatory social functioning mechanisms (e.g., attentional bias to threat, reward processing/feedback learning, and emotion regulation). Vulnerability in these aspects may increase the risk of victimization and impact on social functioning and social support. Dashed lines depict the buffering role of social support moderating the relation between child maltreatment and victimization.

**Intergenerational Transmission of Child Maltreatment**

Experiencing maltreatment during childhood can have a long-term impact on victimization across generations. This intergenerational transmission of maltreatment can be conceptualized from a victim-to-perpetrator and a victim-to-victim perspective (Madigan et al., 2019). According to the victim-to-perpetrator perspective, victims of child maltreatment are at roughly twice the risk of becoming perpetrators of maltreatment once they become parents (Widom, 1989a). The victim-to-victim approach states that children of parents who have been maltreated during their childhood are more likely to become victims of maltreatment themselves. However, according to this perspective their parents do not necessarily act as the perpetrators. For example, several studies have shown that children of mothers who have been sexually abused during their childhood, are at increased risk of being sexually abused by others (i.e.,

perpetrators are most often not their mothers) (e.g., Borelli et al., 2019; K. Kim et al., 2007). The intergenerational transmission of maltreatment hypothesis includes both perspectives (i.e., the victim-to-perpetrator and the victim-to-victim perspective), which are often studied together.

It has long been debated whether intergenerational transmission of child maltreatment actually exists or whether it is merely an artifact of methodologically flawed research (Ertem et al., 2000; Kaufman & Zigler, 1987; Thornberry et al., 2012). This debate led to a number of thorough meta-analyses testing the hypothesis of intergenerational transmission and its association with research quality (Assink et al., 2018; Madigan et al., 2019). The results confirmed that experienced maltreatment during childhood was related to increased risk of maltreatment in the next generation and that this finding is likely not due to methodological weaknesses of those studies. The risk for parents who have experienced child maltreatment to maltreat their own children was estimated to be 2-3 times higher than for non-maltreated parents (Assink et al., 2018; Madigan et al., 2019). In sum, recent meta-analyses support the notion of intergenerational transmission of child maltreatment. However, results also imply that, even though the risk of perpetration is increased, the majority of the maltreated parents do not continue the cycle of victimization.

The victim-to-perpetrator hypothesis was confirmed in a seminal study by Widom et al. (2015). This study was originally set up to focus on archival records to map criminal histories for individuals with and without child maltreatment reports. The study included 902 children with documented histories of abuse and neglect (between the years 1967 and 1971) and matched those with 667 non-maltreated children based on age, sex, race, and social background (Widom, 1989a). These groups were followed for about 40 years during which participants were interviewed and CPS records were searched. Using multiple sources of information, the study showed that parents with child maltreatment experiences were about twice as likely to be reported to CPS compared to the control group. Specifically, 21.4% of the maltreated parents were reported for child maltreatment versus 11.7% of the control group. The study further exposed distinct patterns between different types of child maltreatment. Parents who specifically experienced sexual abuse or neglect during their childhood were at increased risk of maltreating their own children. However, this was not the case for parents who experienced physical abuse. The same was found for perpetrated maltreatment, meaning that parents with histories of maltreatment were generally more likely to sexually abuse or neglect their children, but less likely to physically abuse their children.

Contrary to Widom et al.'s (2015) findings, meta-analytic studies yielded evidence for the transmission of specific types of maltreatment, specifically the

intergenerational transmission of physical abuse. Madigan et al. (2019) found that different types of experienced maltreatment increase the risk of the same as well as other types of maltreatment occurring in the next generation. For example, in case a parent has experienced physical abuse as a child, their offspring is at increased risk of experiencing physical abuse as well and other types of child maltreatment such as neglect, emotional and sexual abuse. Furthermore, Madigan et al. (2019) found that the transmission of child sexual abuse is stronger if the victim or perpetrator is female, however, this was the only type of maltreatment for which gender effects were found. In addition, little evidence was found for differential effects based on the age of the child at the time of the child maltreatment assessment.

Together, the research outlined above indicates that, on the one hand, children of parents who have been maltreated during their childhood are at increased risk of being maltreated by others (victim-to-victim perspective). On the other hand, experiencing child maltreatment increases the risk of maltreating one's own children once victims become parents (victim-to-perpetrator perspective). However, even though maltreated parents are at increased risk of becoming perpetrators of maltreatment, which necessitates the development of targeted prevention and intervention programs, the majority of maltreated parents do not continue the cycle of victimization. In other words, parents who have experienced maltreatment are not destined to maltreat their own children.

**Child Maltreatment and Victimization Outside the Family**

The notion that being a victim of violence feeds the risk of becoming a violent perpetrator has received growing attention during the last couple of decades, partly due to incidences like the 2011 mass shooting in Norway (Jonson-Reid, 1998; Malvaso et al., 2016). Silver et al. (1969) were among the first to study this so-called "violence breeds violence" hypothesis and many researchers followed. For example, overwhelming rates of maltreatment experiences were found in delinquent youth populations (Kratcoski & Kratcoski, 1982; D. O. Lewis et al., 1979). While those studies depicted a strong association between being victimized as a child and becoming a violent perpetrator later in life, they also suffered from methodological limitations, potentially causing an overestimation of the true effects. Many of those studies relied on retrospective designs, used unrepresentative samples, and did not control for confounding variables such as social class differences (Widom, 1989a).

Fortunately, to date a couple of studies were able to overcome these limitations. One of these large-scale prospective longitudinal studies that offered valuable insights into the child maltreatment-offending relation as well as the intergenerational transmission of maltreatment (see previous paragraph) is the study by Widom (1989a). In this study, a large sample of individuals with

substantiated and validated reports of child maltreatment (i.e., abuse or neglect) were matched with a sample of non-maltreated controls. Official records and arrest data were later collected to obtain information about adult criminal behavior for both groups. Although the risk for offending behavior was 1.7 times higher for maltreated individuals (and 1.8 times in a recent meta-analysis by Fitton et al. (2020)) compared to non-maltreated individuals, the vast majority of maltreated adults did not have official records of criminal behaviors, suggesting that the child maltreatment-offending relation is weaker than initially predicted.

Follow-up studies on the same data collected by Widom (1989a) distinguished between different types of child maltreatment and their consequences for violent behavior (Nikulina et al., 2011; Rivera & Widom, 1990; Widom & Maxfield, 1996; Widom, 1989; Widom & Massey, 2015), which happens to be a popular line of research ever since (for a review, see Malvaso et al. (2016)). Despite its popularity, it is still unclear to what extent different types of maltreatment impact on the likelihood of becoming a violent perpetrator later in life. However, recent meta-analyses were not able to show clear differential effects for different types of child maltreatment in association with aggressive and non-aggressive antisocial behavior (Braga et al., 2017; Fitton et al., 2020). Given that subtypes often co-occur, which makes the interpretation of unique effects a lot more complicated (K. Kim et al., 2017; P. M. Sullivan & Knutson, 2000; van Berkel et al., 2020), it can be valuable to also consider contextual features (i.e., frequency, severity, and duration of maltreatment) to gain a clearer understanding of the child maltreatment-offending relation.

The Rochester Youth Development Study (RYDS; C. Smith & Thornberry, 1995) was the first longitudinal study to test for contextual effects. Based on a representative sample of 12-14-year-old American students, a significant association between a history of maltreatment before the age of 12 and delinquent behavior (officially- and self-reported) was found. Specifically, the strength of this association increased as the severity of the maltreatment increased. In other words, more extreme levels of maltreatment lead to higher rates of (violent) delinquency, lending support to the violence breeds violence hypothesis. This association remained significant after controlling for race, gender, socioeconomic status, and family structure and was also found for frequency and duration of maltreatment. In addition to contextual effects, the RYDS data enabled the investigation of how the victims' age at the time of maltreatment is related to later delinquent behavior. Studies that have used this dataset generally showed that maltreatment during adolescence is more strongly related to delinquent behavior compared to maltreatment during childhood (Ireland et al., 2002; Thornberry et al., 2001). However, using a new independent sample, Mersky et al. (2012) could not confirm these findings. Instead, they showed that a history of child maltreatment increases the risk for delinquent behavior at any age.

Reflecting on the literature about the child maltreatment-offending relation within and outside the family clarifies that this relationship is not deterministic. Instead, this association is influenced by a complex interplay of individual, social and contextual factors. Moreover, research shows that there is a broad transmission of different types of maltreatment, indicating that it is not (only) the specific behavior that is transmitted but that broader mechanisms are at stake. In order to be able to break the cycle of victimization within and outside the family, it is crucial to better understand these factors and possible mechanisms that can help explain the maltreatment victim-victimizing relation. Selected mechanisms that could explain the effect of early-life maltreatment experiences on parenting behaviors include neurophysiological, information-processing, and developmental psychopathology models (Alink et al., 2019). These will be discussed in the following paragraphs.

**Mechanisms Connecting Child Maltreatment and Victimization**

Several models exist that can help explain how child maltreatment increases the risk of victimization across the life span. The ecological-transactional perspective on child maltreatment as well as the developmental psychopathology framework suggest that "normative developmental processes" can be elicited by providing a child with an age- and stage-appropriate "average expectable environment" (Cicchetti & Valentino, 2006; Sroufe & Rutter, 1984). However, failure to do so (e.g., exposing the child to maltreatment) can impede development and subsequently lead to transgressive behaviors (e.g., victimization) later in life.

Child maltreatment is a chronic stressor for the victims and chronic activation of the stress system leads to allostatic load, which describes an accumulation of stress response built up in the body (McEwen, 1998). When the human body undergoes allostasis, brain activity shifts from higher order cognitive activation to lower order salience/threat activity (Oei et al., 2012). In addition, this stress exposure activates the hypothalamic–pituitary–adrenal (HPA) axis and the immune system, which subsequently leads to the release of stress hormones (cortisol) and pro-inflammatory markers (cytokines) into the blood stream. Initially, this is an adaptive response that prepares the body to fight or flight. However, on the long-term, a high allostatic load can detrimentally affect neurochemical processes, behavioral responses, and (neuro)physiology. For example, animal studies have shown that early-life stress is associated with alterations in neural morphometry of the animal brain due to, for instance, suppression of neurogenesis and/or neuronal cell-death (Arnsten, 2009; Lupien et al., 2009; Radley et al., 2004; Sanchez et al., 2001). Through this mechanism, chronic stress in the context of child maltreatment is thought to seriously affect the developing brain.

Latent vulnerability and adaptive calibration models suggest that the impact of child maltreatment experiences on the developing brain may aid adaptive behavior in order to survive and reproduce in such high-stress environments. However, as soon as the environment is no longer threatening, such adaptations might create vulnerability to future mental health problems (Del Giudice et al., 2011; McCrory & Viding, 2015). Three latent vulnerability factors are relevant in this respect: an increased attentional bias to threat, reduced reward processing/feedback learning, and emotional (dys)regulation (McCrory & Viding, 2015). These processes eventually place maltreated individuals at risk for maladaptive behavior, including impaired social functioning, and together may underlie the risk for victimization later in life.

### *Attentional Bias to Threat*

Attentional biases reflect an individual's tendency to direct attention to stimuli that match their thoughts and feelings. In the context of child maltreatment, it may be adaptive to rapidly detect threat, such as angry facial emotions of parents. However, over time and in different circumstances this bias to threat could lead to dysfunctional emotions and behavior. Previous work has indeed shown a link between exposure to maltreatment and attentional bias to threat (da Silva Ferreira et al., 2014). Children with a history of maltreatment more rapidly detect and classify emotional faces as threatening. For instance, physically abused children were found to display a response bias towards angry facial expressions, whilst neglected children showed more difficulty discriminating between facial emotions (Pollak et al., 2000). On a neural level, this can be explained by a hyperresponsivity of the amygdala, which is often found in individuals, who were victims of child maltreatment (Hein & Monk, 2017; van Harmelen et al., 2013). The amygdala is a brain region involved in the primary processing of emotional faces, salience detection, fear conditioning and emotional memory (Bremner et al., 2005; Davis & Whalen, 2001; Onur et al., 2009; Todorov & Engell, 2008).

Although adaptive and protective in the context of high stress, an increased attentional bias to threat is thought to contribute to victimization (Crick & Dodge, 1994; Lemerise & Arsenio, 2000; N. V. Miller & Johnston, 2019). For example, an over-attribution of hostile intentions to others' actions might elicit preemptive behavior (e.g., aggression). Hence, parents who attribute hostile intent (i.e., negative parental attributions) towards their child's behavior can be at increased risk for harsh and abusive parenting (Beckerman et al., 2018; Irwin et al., 2014). Furthermore, it has been shown that hostile attributions are a mediating factor for the association between child maltreatment and reactive aggression (Richey et al., 2016), which is why hypervigilant responding to threat might be one potential mechanism linking a history of child maltreatment with victimization later in life.

### *Reward Processing and Feedback Learning*

Sources of reward in a maltreating family environment can be scarce and unpredictable. Reduced anticipation of reward, therefore, may lower the likelihood of continued disappointment and as such represents a positive adaptation in a volatile and uncertain environment. Indeed, both human and animal research have shown that chronic stress exposure early in life can lead to long-term alterations in reward-related behaviors (Birn et al., 2017; Hollon et al., 2015), mediated by changes in the ventral striatum, a subcortical brain structure that plays a role in reward processing and learning (Hanson, Hariri, et al., 2015). Furthermore, research in maltreated children and adolescents (8-14 years) demonstrated reduced sensitivity towards (monetary) rewards (Guyer et al., 2006) as well as a blunted anticipation of rewarding cues on a behavioral and neural level (Dillon et al., 2009).

Reward processing also plays an important role in parenting behaviors. For instance, infant cues, including those of distress, have been found to activate parental reward neurocircuitry (limbic brain regions, striatum, and orbitofrontal cortex), which in turn promotes caregiving responses (Ferrey et al., 2016). This motivated attention to their child's socio-emotional cues is an important driver for sensitive caregiving behavior. Therefore, parents with impaired reward processing (possibly due to their early-life experiences) may lack motivation to attend to their child's needs, which ultimately can put the child at risk for experiencing maltreatment (Strathearn, 2011).

Altered reward processing has also been found to be associated with aggressive behavior. For example, adolescents (16-18 years) with aggressive conduct disorder showed, compared to controls with no conduct disorder, an altered activation in brain regions associated with reward processing (among others in the amygdala and ventral striatum) whilst viewing others in pain (Decety et al., 2009). In other words, highly aggressive youth may enjoy hurting others, which together with an impoverished ability to downregulate one's emotional arousal (see next paragraph) can put them at an even greater risk for victimization (i.e., aggression).

Reward processing is also involved in how individuals learn from feedback, which when growing up in an adverse home environment is either available as an excess of negative feedback and/or a lack of positive feedback. This shapes the way a child incorporates such information and consequently adjusts their behavior. Feedback learning relies in part on the hippocampus (K. C. Dickerson & Delgado, 2015), and early-life stress (e.g., child maltreatment) has been found to reduce hippocampal volume, activation and learning performance (Riem et al., 2015; Schwabe & Wolf, 2012). In support of these findings, child maltreatment has been related to learning difficulties (Hart et al., 1997), impaired spatial working

memory (Majer et al., 2010), impoverished verbal fluency, and reduced cognitive flexibility (Savitz et al., 2008). Children and adolescents (2-17 years) with such (learning) difficulties are known to be at greater risk of experiencing victimization (e.g., bullying, physical abuse, and neglect), which according to the violence breeds violence hypothesis, will also impact on the likelihood of becoming a violent perpetrator later in life (Klomek et al., 2016; Turner et al., 2011). In other words, individuals who have experienced child maltreatment are at greater risk to develop, for example, learning difficulties, which once again put them at greater risk of victimization as well as engaging in (violent) delinquent behavior later in life. Together, altered reward processing and feedback learning might be two additional mechanisms that can put maltreated children at risk for maladaptive behavior, including victimization in later life.

***Emotion Regulation***

The ability to modulate one's emotional arousal (i.e., emotion regulation) is important to be able to respond in a socially acceptable manner to ongoing environmental demands. Poor emotion regulation capacity has been linked to behavioral problems (e.g., internalizing and externalizing symptoms), which can in part be explained by an impoverished ability to downregulate and/or reappraise threat and stress responses (J. Kim & Cicchetti, 2009; Sheppes et al., 2015). According to attachment theory, securely attached children can use caregivers effectively to help learn how to regulate their emotions (Bowlby, 1982). However, in the case of child maltreatment, the absence of a caregiver, who is structuring, explaining, and regulating the emotional world of their child, poses a threat to the optimal development of emotion regulation, which ultimately puts the child in jeopardy of developing psychopathology and behavioral problems (Alink et al., 2009; J. Kim & Cicchetti, 2009).

To identify proximal risk factors for psychopathology and behavioral problems, a study by McLaughlin, Peverill, et al. (2015) investigated how child maltreatment influences neural processes underlying emotion regulation during a time of sensitive neurobiological development. The study showed that maltreated adolescents (13-19 years) exhibited heightened amygdala reactivity in response to viewing negative emotional stimuli. However, this elevated emotional reactivity was also regulated to a greater degree through prefrontal regions causing a down-regulated amygdala comparable to activations observed in non-maltreated adolescents. Whilst in this study maltreated adolescents could successfully modulate their increased vigilance to (negative) emotional stimuli, these findings also shed light on the mechanisms putting maltreated individuals at greater risk for developing aberrant patterns of emotion regulation.

Several studies have confirmed that dysfunctional patterns of emotion regulation in maltreated children (McLaughlin et al., 2020; Shields & Cicchetti, 2001; Teisl

& Cicchetti, 2007) are often accompanied by heightened emotional responses (e.g., aggression) to potential threats in the environment. For example, altered emotional regulation was found to mediate the relation between experiencing child maltreatment and aggressive behavior problems (e.g., bullying and victimization) during childhood (P. M. Cole & Zahn-Waxler, 1992; Shields & Cicchetti, 2001; Teisl & Cicchetti, 2007). Moreover, meta-analytic evidence suggests that problems in anger regulation are a key risk factor for child maltreatment (Stith et al., 2009). Together, altered emotion regulation as a consequence of child maltreatment can increase the risk of developing psychopathology and aggressive behavioral problems, highlighting the importance of exploring emotion (dys)regulation as a mechanism linking child maltreatment and victimization.

***Social Functioning***

The ability to perform and fulfil normative social roles (i.e., social functioning), relies in part on the mechanisms described in the previous paragraphs (i.e., threat reactivity, reward anticipation/feedback learning, and emotion regulation). As such, vulnerability in these aspects may lead to maladaptive social functioning (McCrory et al., 2019). Indeed, individuals with a history of child maltreatment are thought to generate more stress given their increased likelihood to encounter (socially) stressful events (also known as the stress generation model; McCrory et al. (2019)). Consequently, those individuals have greater difficulties forming and/or maintaining high-quality relationships that can help buffer against future stress (Benedini et al., 2016; Gerin et al., 2019; McCrory et al., 2019; van Harmelen et al., 2016).

Forming positive, high-quality social relationships during childhood is particularly important for the mental well-being of adolescents with a history of early-life adverse experiences (van Harmelen et al., 2017). It is therefore not surprising that being socially rejected is a potent risk factor for adjustment problems later in life (Coie & Cillessen, 1993). Experiencing social rejection is central in the context of child maltreatment. It has not only been found that adolescents with a history of child maltreatment are more sensitive to peer rejection on a behavioral and neural level (van Harmelen et al., 2014) but alarmingly maltreated individuals are also more likely to be rejected by their peers in the first place (Bolger & Patterson, 2001). These rejection experiences consequently induce a magnified sensitivity towards future rejection. Specifically, individuals with high rejection sensitivity tend to predict, perceive, and show particularly enhanced distress towards social rejection (DeWall et al., 2009; Riva et al., 2012).

Children who have experienced maltreatment (specifically physical and/or sexual abuse) have been found to be at increased risk for victimization by peers as well

as are more likely to bully others (Shields & Cicchetti, 2001; van Harmelen et al., 2016). These findings are in line with stress susceptibility models, which suggest that child maltreatment contributes to an increased psychiatric vulnerability to future (social) stress (Gerin et al., 2019). Moreover, work by DeWall et al. (2009) on the path between social rejection and aggression has shown that excluded individuals have an increased tendency to attribute hostile intent towards others actions (hostile cognitive bias). Ultimately, social stress vulnerability and generation may increase the likelihood of peer rejection and victimization (Benedini et al., 2016; Gerin et al., 2019; McCrory et al., 2019; van Harmelen et al., 2016), which is why maladaptive social functioning might in part explain how experienced child maltreatment aids later victimization and aggressive behavior.

**Social Support Buffers the Impact of Child Maltreatment**

Not all individuals with a history of child maltreatment will victimize others, which suggests that those individuals benefit from protective (or resilience) factors. Those factors may modulate mechanisms associated with maltreatment related vulnerability (e.g., increased threat reactivity, lower reward anticipation/feedback learning, and dysfunctional emotion regulation), ultimately aiding resilient functioning in the aftermath of child maltreatment (Ioannidis et al., 2020; Kalisch et al., 2019; W. A. Walsh et al., 2010).

Social stress buffering models argue that social support can buffer the negative effects of stress on physical and mental health (Gunnar, 2017). Specifically, a social partner can reduce the physiological impact of stress on the body through attenuating the release of stress hormones (e.g., cortisol), which ultimately may help lower the risk of mental health difficulties (Hostinar et al., 2014b). In line with these models, it was found that friendship and family support reduce depressive symptoms in adolescents with a history of maltreatment (van Harmelen et al., 2016). Additional research has shown that high-quality social relationships influence the development of emotion regulation skills, which act as a protective factor moderating the relation between child maltreatment and psychopathology (Alink et al., 2009; Fritz, de Graaff, et al., 2018; Fritz, Stochl, et al., 2020; Ioannidis et al., 2020).

However, little is known about the neurobiological stress mechanisms that may underlie this relation. For example, a pre-registered, systematic literature review has identified only two studies that directly examined whether friendship support buffers neurobiological stress responses in young people (10-24 years) with a history of childhood adversity (Scheuplein & van Harmelen, 2022). One study tested these mechanisms in previously institutionalized adolescents and found that high-quality friendships at age 12 can buffer the negative effect of blunted sympathetic nervous system reactivity on peer problems at age 16 (Tang et al., 2021), whereas the other identified study was limited by an underpowered sample

of well-functioning adolescents (Fritz, Stretton, et al., 2020). Studies that have investigated social stress buffering in individuals without a recorded history of childhood adversity showed that those with greater levels of perceived social support had reduced neural activity (dorsal anterior cingulate cortex and anterior insula) as well as diminished cortisol responses following social exclusion (Eisenberger et al., 2007; C. L. Masten et al., 2012). However, future research is needed to further investigate these effects in individuals with a history of childhood adversity.

Meta-analytic evidence suggests that safe, stable, and nurturing relationships are critical for breaking the intergenerational transmission of maltreatment (Schofield et al., 2013). In other words, improving social support could lead to a reduction of intergenerational transmission of maltreatment, which ultimately could lower the risk of victimization within and outside the family environment.

**Conclusion and Future Directions**

Experiencing maltreatment during childhood may have a lasting impact on an individual's life trajectory as well as on society at large. Ample evidence suggests that victims of maltreatment are at increased risk for developing psychopathology, getting victimized by others (e.g., bullied), and becoming violent perpetrators themselves (Alink et al., 2009; Buisman et al., 2020; Salo et al., 2021). In this chapter, we outlined the link between child maltreatment and victimization within and outside the family environment and highlighted explanatory mechanisms and the buffering role of social support that could help break the cycle of victimization. The concepts and mechanisms presented in this chapter are modeled in Figure 1.

In the first half of the chapter, we have shown that experiencing maltreatment during childhood can have a long-term impact on victimization across generations. It is clear that experiencing child maltreatment increases the risk of maltreating one's own children once victims become parents (Madigan et al., 2019). Moreover, we have elaborated on the notion that being a victim of violence feeds the risk of becoming a violent perpetrator later in life (violence breeds violence hypothesis) (Silver et al., 1969).

In the second half of the chapter, we have demonstrated that child maltreatment impacts on various interrelated neurocognitive mechanisms aiding different forms of victimization. First, we highlighted that a dysfunctional attentional bias to threat can lead to hypervigilant and aggressive responding (Richey et al., 2016). Second, it was discussed that altered reward processing and feedback learning are known features of maltreatment-related psychopathology, which can lead to maladaptive behavior (e.g., aggression) in novel environments (McCrory et al., 2017). Third, poor emotion regulation capacity was found to link to internalizing

and externalizing symptoms which can in part be explained by an impoverished ability to downregulate and/or reappraise threat and stress responses (J. Kim & Cicchetti, 2009). Partially as a result of these processes, children with a history of child maltreatment are at increased risk for developing maladaptive social functioning, to experience victimization by peers, and to victimize others (McCrory et al., 2019; Shields & Cicchetti, 2001).

An important factor for mitigating the impact of child maltreatment on social functioning mechanisms is a positive and supportive social environment, which can ultimately lower the risk of violent behaviors not only now but also in the next generation. However, we have also shown that individuals who have experienced child maltreatment are more likely to struggle with creating and sustaining this protective social environment.

To break the cycle of victimization, we argue for a greater translation of knowledge about the neurocognitive processes that underlie social functioning (e.g., threat processing, reward processing/feedback learning, and emotion regulation) and that are evidently impacted by child maltreatment. It is necessary to intervene early and focus on improving these neurocognitive processes in order to strengthen social functioning and as a result minimize or even eliminate the risk of victimization later in life. Behavioral interventions targeting social cognitive processes, especially the encoding and interpretation of social cues, represent promising treatment approaches (McLaughlin, DeCross, et al., 2019; Waters & Craske, 2016). For example, by teaching 16-18-year-old juvenile offenders how to positively reframe ambiguous social cues, Ren et al. (2021) were able to show a significant reduction in hostile attribution bias and self-reported aggression. Given that transgressive behaviors (e.g., victimization) are often triggered through the interpretation of other's hostile intent it is powerful to observe that similar low-cost interventions in children (4-9 years) (van Dijk et al., 2019) and adults (Osgood et al., 2021) also reported reduced hostile attribution biases as well as mitigation effects on aggressive behaviors. Furthermore, behavioral activation treatment has been found to effectively improve reward-related functioning (e.g., approach motivation or reward valuation) in clinically depressed adolescents (12-18 years) (McCauley et al., 2016) and adults (18-60 years) (Dimidjian et al., 2006). However, despite its effectiveness, only a few studies have explored the benefits of this intervention in individuals with adverse early-life experiences (Berkowitz et al., 2011; McLaughlin, DeCross, et al., 2019) and more research is needed to establish its effectiveness in reducing victimization later in life.

Another promising treatment approach, specifically for individuals with a history of child maltreatment, is trauma-focused cognitive behavioral therapy (TF-CBT). Among other things, TF-CBT targets heightened emotional responsiveness to

negative stimuli through cognitive reappraisal strategies (e.g., positively reinterpreting emotional stimuli) (Dorsey et al., 2017; McLaughlin, Peverill, et al., 2015). Cognitive reappraisal has been proven to be an effective tool to treat externalizing and internalizing problems in at-risk youth (7-15 years) (Weisz et al., 2017) as well as to modulate arousal and negative emotional reactivity in maltreated children and adolescents (Dorsey et al., 2017; McLaughlin, Peverill, et al., 2015). On a broader level, school-based social and emotional learning (SEL) programs have been successful in enhancing students' behavioral adjustment (i.e., increased prosocial behavior as well as reduced conduct and internalizing problems) through teaching, for example, how to recognize and manage emotions, appreciate the perspectives of others, and maintain positive social relationships (for a thorough meta-analysis see Durlak et al. (2011)). Many of those interventions are promising treatments designed to be flexibly administered in young at-risk populations. However, these interventions may benefit from a stronger focus on the whole range of social functioning mechanisms connecting child maltreatment and victimization.

Even though there is substantial evidence supporting the various neurocognitive mechanisms that we have described, several important aspects are still not fully understood. For example, due to inconsistencies in the literature, it is unclear how type, duration, and frequency of maltreatment as well as age of exposure link to atypical behavioral and neurobiological functioning. Timing of maltreatment also seems to be important. For instance, the life cycle model of stress suggests that across development, brain regions undergo different windows of vulnerability (Lupien et al., 2016). Hence, there might be sensitive periods of development during which the effects of child maltreatment are particularly detrimental (see sensitive periods theory; Teicher & Samson (2016)). This notion has been confirmed in retrospective studies, showing that, for example, hippocampal alterations appeared to be particularly affected by maltreatment exposure during early childhood (3-5 years), whilst amygdala alterations were linked to exposure during early adolescence (10-11 years), and prefrontal cortical deficiencies were related to exposure during mid adolescence (14-16 years) (Andersen et al., 2008; Teicher et al., 2018). However, these findings require further empirical support. A meta-analysis has, for example, shown that the association between child maltreatment and reductions in hippocampal volume was strongest when maltreatment was reported during middle childhood and early adolescence, rather than early childhood (Riem et al., 2015). Furthermore, the literature on how timing of maltreatment affects victimization is inconsistent and merely relying on retrospective designs, which makes it difficult to estimate the frequency as well as to pinpoint the period when maltreatment took place. Therefore, prospective studies are needed to gain a more applicable understanding of the association between maltreatment characteristics (e.g., age of exposure, duration, and frequency) and later maladaptation.

Experiencing maltreatment during childhood will likely have a lasting impact on an individual's life trajectory as well as on the society at large. To give an example, the nonfatal child maltreatment lifetime costs in the U.S. were estimated at $830,928 (2015 USD) per victim and based on investigated incident cases the estimated annual costs for society were considerably higher, estimated at $2 trillion (Peterson et al., 2018). If we consider the indirect effects that maltreatment has on lives of others, as described in this chapter, the costs would likely be much higher. This underscores the need for early detection and intervention approaches to target the mechanisms connecting child maltreatment and victimization. Therefore, the ultimate goal should be to break the cycle of victimization and thereby pave the way for a healthier and more secure society.

**Further Readings**

- Paper by Buisman et al. (2020) on the intergenerational transmission of child maltreatment. https://doi.org/10.1371/journal.pone.0225839
- Paper by Ioannidis et al. (2020) on the complex neurobiology of resilient functioning after childhood adversity. https://doi.org/10.1186/s12916-020-1490-7
- Paper by Currie & Tekin (2012) on the cycle of child maltreatment and the link to future crime. http://dx.doi.org/10.3368/jhr.47.2.509
- Summary information of 2091 studies that have investigated the consequences of child maltreatment. Provided by the WHO: https://apps.who.int/violence-info/child-maltreatment/

# Chapter 3

## The Importance of Friendships in Reducing Brain Responses to Stress in Adolescents Exposed to Childhood Adversity: A Pre-Registered Systematic Review

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**Abstract**
Up to 50% of all children and adolescents growing up worldwide are exposed to at least one form of childhood adversity (CA), which is one of the strongest predictors for later-life psychopathology. One way through which CA confers such vulnerability in later-life is through increased sensitivity to and likelihood of social stress. A growing body of research demonstrates the positive impact of adolescent friendship support on mental well-being after CA, however, the mechanisms that may underlie this relationship are unknown. Neurobiological models of social buffering suggest that social support can reduce perceptions, reactions, and physiological responses to and after stress. Therefore, this pre-registered, systematic literature search examined whether friendships reduce neural stress responses in adolescents with CA.

*Keywords:* friendship buffering, social support, neural stress mechanisms, childhood adversity, adolescence, systematic review.

## Introduction

Up to 50% of all children and adolescents growing up worldwide are exposed to at least one form of childhood adversity (CA; e.g., abuse, neglect, bullying, or poverty) (Bellis et al., 2014; McLaughlin, 2016). CA is a strong predictor of later-life mental health and interpersonal problems. One way through which CA confers such vulnerability is through increased sensitivity to and likelihood of interpersonal stress (e.g., peer relationship problems) (Humphreys et al., 2016; van Harmelen et al., 2014, 2016). As such, to improve well-being, it is imperative that stress vulnerability is reduced in young people with CA.

Safe, stable, and nurturing social relationships can help reduce perceptions, reactions, and physiological responses to and after stress (Gunnar, 2017). Friendships may be a particularly important support source in adolescence, as this is a time when young people start to form more stable, intimate, and reciprocal peer relationships (Orben et al., 2020). Friendship support has indeed been found to improve mental well-being in young people with CA (van Harmelen et al., 2016, 2021). However, it is unknown whether friendship support aids mental well-being through reducing stress responses in these individuals. Therefore, we performed a pre-registered, systematic literature search to examine whether friendship support reduces brain responses to stress in adolescents with CA (see Figure 1).

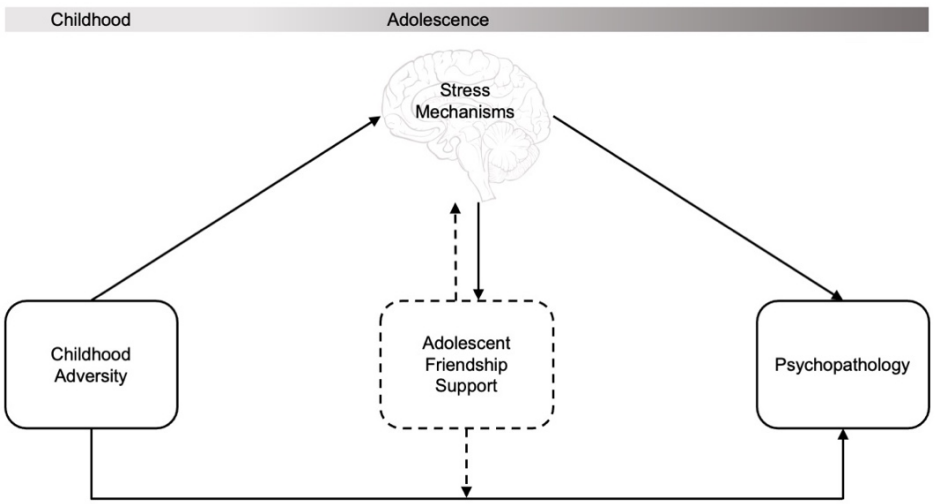


**Figure 1.** Friendship buffering effects on brain responses to stress in adolescents with CA. Adolescent friendship support may help reduce (or buffer) neural stress responses (dashed lines) that are thought to aid psychopathology in young people with CA (solid lines).

**Neurobiological Stress Mechanisms Linking CA and Psychopathology**

Prolonged stress exposure early in life can disrupt the development of psychological and neurobiological processes and thereby increase vulnerability to psychopathology (Y. Chen & Baram, 2016). In humans, CA can impair the responsiveness of the hypothalamic-pituitary-adrenal (HPA) axis; a key stress response system that gets activated when homeostasis (i.e., the body's tendency to maintain a stable internal environment) is threatened. The HPA axis is responsible for producing stress hormones (e.g., cortisol), also known as glucocorticoids (Gunnar et al., 2019; Lupien et al., 2009). Glucocorticoids are potent anti-inflammatory as well as immunosuppressive agents and are important for healthy brain development due to their involvement in neural maturation, myelination, and neurogenesis (Auphan et al., 1995; Lupien et al., 2009). In the context of CA, sustained HPA axis activation can lead to chronically elevated levels of glucocorticoids in the brain and altered frontolimbic development and functioning (Cohodes et al., 2021; McEwen, 2012). Due to their dense innervation with glucocorticoid receptors, brain regions like the hippocampus, amygdala, anterior cingulate cortex, or prefrontal cortex may be particularly impacted resulting, for example, in dysfunctional social information- and emotional processing (Arnsten, 2015; McLaughlin et al., 2020; Tottenham & Sheridan, 2010). According to the theory of latent vulnerability, alterations to these mechanisms may be adaptive in the short-term to support survival in highly stressful and threatening environments. However, in the long-term, such recalibration of the stress system can become maladaptive (McCrory et al., 2019). For example, in the context of an abusive home environment, it may be adaptive to rapidly detect threats (e.g., angry facial expressions). However, in less threatening environments this amygdala-supported attentional bias to threat may aid an over-attribution of hostile intentions to others' action, possibly eliciting preemptive (aggressive or avoidance) behavior (Heuer et al., 2007; N. V. Miller & Johnston, 2019). Indeed, such attentional biases were predictive of future onset of internalizing and externalizing problems in young people with CA (Shackman & Pollak, 2014) and were linked to reduced social interactions and greater difficulties with peers (Humphreys et al., 2016). Forming and maintaining friendships, defined as voluntary, reciprocal, and nurturing relationships, requires social-emotional competence (McCrory et al., 2019), which relies in part on neurocognitive mechanisms (e.g., emotion perception and regulation) known to be altered in adolescents with CA (Benedini et al., 2016; McCrory et al., 2019). Consequently, through this mechanism, young people with CA may be more vulnerable and more likely to experience social stress (i.e., stress generation; McCrory et al. (2019)). CA is therefore thought to shape neurodevelopment in a way that increases vulnerability to social stress (J. Kim & Cicchetti, 2009; McCrory et al., 2019; McLaughlin et al., 2020).

**Social Buffering of Neurobiological Stress Responses across Development**

Social buffering describes the phenomenon in which a social partner can attenuate acute physiological stress responses (Gunnar, 2017). This leads to a reduction in the release of glucocorticoids and proinflammatory markers into the bloodstream (Hostinar et al., 2014a; R. M. Sullivan & Perry, 2015). In humans, this decreased allostatic load (i.e., the physiological impact of stress on the body) may help protect against the emergence of psychopathology (S. Cohen & Wills, 1985; McLaughlin et al., 2020). Indeed, trauma exposed young people with high levels of social support are less likely to develop psychopathology (Trickey et al., 2012).

Social buffering occurs throughout the lifespan and its effectiveness is influenced by previous social experiences, as well as the developmental stage of the recipient (Hennessy et al., 2009). During early childhood, the caregiver is the most potent stress buffer. Animal models have demonstrated that maternal presence can attenuate glucocorticoid release and block amygdala-dependent threat learning in rodent pups (Raineki et al., 2014; R. M. Sullivan & Perry, 2015). Similarly, in humans, maternal availability after a social evaluative performance stressor was found to facilitate greater oxytocin release, a neuropeptide capable of inhibiting glucocorticoid secretion in response to stress, as well as a more rapid decrease and lower levels of peak cortisol in children (Seltzer et al., 2010). Across social species, high-quality caregiving, characterized by predictable caregiving that signals safety, can improve the effectiveness of social buffering (Ainsworth et al., 1974; Gee & Cohodes, 2021). In humans, high-quality caregiving modulates children's frontolimbic circuitry and contributes to healthy socioemotional functioning (Gee & Cohodes, 2021). For example, greater feelings of child-reported security in the caregiver-child attachment relationship buffers amygdala reactivity, enhances affective behavior, and mental health (Callaghan et al., 2019; Gee et al., 2014).

While caregivers remain potent stress buffers throughout childhood, evidence suggests that their effectiveness diminishes with the transition to adolescence (Gee et al., 2014; Hostinar et al., 2015). One potential mechanism proposes that with the maturation of frontolimbic circuitry caregivers lose their active role in facilitating emotion regulation and buffering amygdala reactivity (Gee & Cohodes, 2021; Gee et al., 2014; Hostinar et al., 2015). This makes space for other attachment figures to take over the stress-alleviating role of social support. At the same time, adolescents learn to navigate the world more independently and start to increasingly form and maintain emotionally intimate peer relationships (Orben et al., 2020). Hence, peers take on a more central role in social-emotional buffering (Gee & Cohodes, 2021).

Adolescent friendship support is a potent protective factor, capable of buffering threat-related processing (see Gunnar (2017) for review). Specifically, adolescents with heightened levels of perceived social support (e.g., measured through the time spent interacting with friends) had diminished cortisol responses and lower neural activity in brain regions commonly associated with social distress following social exclusion (Eisenberger et al., 2007; C. L. Masten et al., 2012), providing initial evidence that adolescent friendships may buffer neural stress responses in young people without CA (Eisenberger et al., 2007). However, it is yet unknown whether friendship support similarly buffers neural stress responses in vulnerable adolescents with CA.

## Do Friendships Reduce Neural Stress Responses in Adolescents with CA?

While there is a growing body of research demonstrating the positive impact of adolescent friendship support on mental well-being after CA (Fritz, Stretton, et al., 2020; van Harmelen et al., 2016, 2021), very little is known about the neural mechanisms that aid this relationship. Therefore, we performed a pre-registered, systematic literature review to examine whether friendship support buffers neural stress responsivity in adolescents with CA (Prospero: CRD42021233949).

### *Systematic Review: Study Selection and Data Extraction*

In accordance with the PRISMA guidelines (Page et al., 2021), we searched for empirical studies (peer-reviewed articles, proceedings papers, and conference papers) published in English and involving human subjects by using internet databases (Web of Science Core Collection, PubMed, and PsycINFO) through December 2021.

We included studies assessing friendships and neural stress responses in adolescents with CA. Specifically, we included studies in which at least a portion of the adolescent sample (sample mean age 10-24) had experienced or reported a history of CA. CA was defined as any event assessed prior to the age of 18, which is "likely to require significant adaption by an average child and that represent[s] a deviation from the expectable environment" (McLaughlin, 2016, p. 363), such as abuse, neglect, or bullying. Friendships had to be assessed between the (sample mean) age of 10-24 and were defined as self- or other-reported, subjective or objective peer relationships, excluding support from family, pets, community ties, or co-workers. Measures of neural stress responses needed to be induced (e.g., stress exposure or negative feedback) in the lab, and assessed using neuroimaging techniques (e.g., fMRI or EEG).

These categories were included in search terms encompassing friendship support, study population, neural domains, CA, and stress exposure (see Table S1 for all search terms used). To identify studies missed in this targeted search, we

performed backward reference searches, and used Google Scholar for forward searching.

A total of 6,260 articles were identified. After removing duplicates, two independent reviewers screened titles, abstracts, and keywords of 4,297 articles based on the PI(C)OS concept: population (P; adolescents between the age of 10-24 (sample mean age) with CA (assessed prior to the age of 18)), intervention (I; friendships assessed between the age of 10-24 (sample mean age)), outcome (O; neural stress mechanisms), and study design (S; empirical study) (Liberati et al., 2009). This screening resulted in adequate inter-rater reliability of Kappa = .58, disagreement was resolved through discussion. Next, 66 full-text articles were selected and subsequently assessed for eligibility, however, only two articles matched all search criteria (Kappa = .79) (Figure 2). Therefore, we allowed stress responses in any neurobiological system (incl. endocrine and sympathetic nervous system) and included two additional studies. For a detailed summary of all excluded studies see Tables S2-6. A risk of bias (quality) assessment was performed for the four included studies, in which studies could score one point for each quality marker they met (e.g., "Is the sample representative of the defined population?"). The overall quality score (QA score) for each study was calculated by adding up all nine items (see Table S7 for all assessment questions).

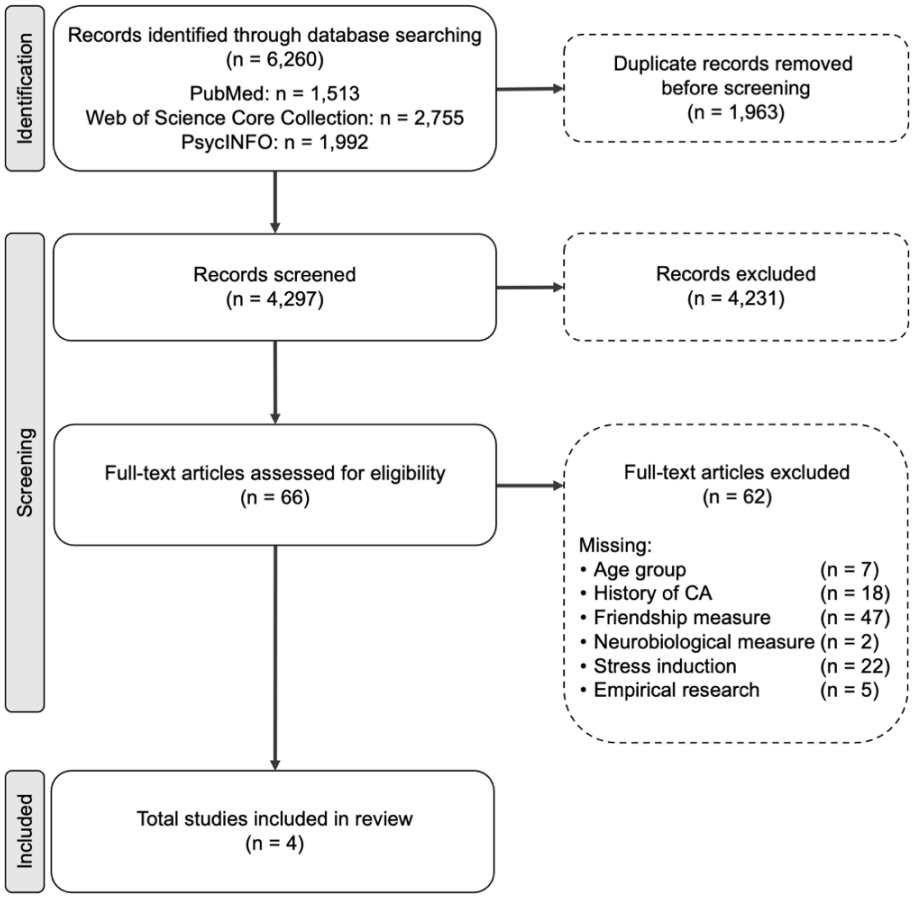


**Figure 2.** *PRISMA flow diagram.* Adapted from (Page et al., 2021).

### *Results*

This pre-registered, systematic review identified four eligible studies (Fritz, Stretton, et al., 2020; Kelly et al., 2015; Negriff et al., 2020; Alva Tang et al., 2021) (see Figure 2). Only two of those studies did directly test whether friendships buffer neurobiological stress responses in adolescents with CA (Fritz, Stretton, et al., 2020; Tang et al., 2021). Tang et al. (2021) showed that high-quality friendships at age 12 can buffer the indirect effect of maladaptive stress physiology (blunted sympathetic nervous system reactivity to social rejection feedback) on peer problems at age 16 in 217 adolescents (136 with CA) who had been institutionalized. In contrast, Fritz, Stretton, et al. (2020) found that friendship support at ages 14 or 17 was not associated with affective behavioral or neural responses to social rejection at age 18 in a small sample of 55 adolescents (26 with CA). Although, adolescents with CA reported more friendship support at age 14, suggesting a particularly well-functioning sample with possibly normalized stress responses. Indeed, Schweizer et al. (2016) reported enhanced emotion regulation

capacity in the same cohort of adolescents with CA, perhaps obscuring the ability to comprehensively examine friendship stress buffering effects.

The remaining two studies included in the review did not directly test the model of interest (Kelly et al., 2015; Negriff et al., 2020). Kelly et al. (2015) found that adolescents (aged 10-14) with documented maltreatment experiences displayed increased emotional reactivity, an attentional bias away from threat, and reduced gray matter volume (GMV) in the left medial orbitofrontal cortex (mOFC), a brain region implicated in empathic, social functioning. In addition, reduced GMV in the left mOFC mediated the relationship between maltreatment and peer relationship problems providing support for neural stress generation and mechanisms in adolescents with CA. Similarly, Negriff et al. (2020) found that 10-year-old adolescents with CA reported a smaller perceived friendship support network and showed blunted cortisol responses to social stress. However, in both studies it was not specifically tested whether friendship support or network characteristics (size or interconnections) were associated with reduced neurobiological stress responses.

These findings add to studies that were excluded from the current review due to missing search criteria (summarized in Tables S2-6). For example, studies investigating friendship stress buffering in individuals without CA demonstrated that adolescents who spent more time with friends showed reduced neural activity (dACC and anterior insula) during social exclusion (C. L. Masten et al., 2012). Whereas adults with below average levels of perceived social support showed a positive correlation between threat-related amygdala reactivity and trait anxiety (Hyde et al., 2011).

Moreover, three excluded studies supported the notion of friendship buffering on neurobiological mechanisms, despite not investigating stress responses (Gu et al., 2020; Malhi et al., 2020; Reid et al., 2021). First, Reid et al. (2021) found that the quality of social support, but not previous institutionalization experiences, predicted changes in diurnal cortisol patterns across early adolescence. Specifically, higher levels of social support were associated with lower bedtime cortisol levels. Second, Gu et al. (2020) tested adolescents orphaned by parental HIV/AIDS who displayed decreased cortical resting state activity (elevated theta-to-beta ratio) in fronto-central regions, which was also associated with greater learning and behavioral problems as well as difficulties making friends. Similarly, Malhi et al. (2020) showed that adolescents (12-18 years) with severe emotional trauma had smaller left hippocampal volumes as well as less perceived social support from friends, family, and significant others, compared to individuals with minimal trauma exposure. In addition, the former two studies (Gu et al., 2020; Malhi et al., 2020) lend further support to the stress generation idea through which friendship support may also be less available to young people with CA.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings | QA Score |
|---|---|---|---|---|---|---|---|---|
| Fritz, Stretton, et al. (2020) | Total: 55<br><br>CA: 26<br>Control: 29 | Three timepoints: 14, 17, 18<br><br>*Scanning at $T_3$* | Intrafamily adverse events<br><br>CAMEEI performed with primary caregiver | Perceived friendship support<br><br>CFQ | Brain function | Social feedback task | CA predicted higher levels of perceived friendship support at age 14, which was associated with perceived friendship support at age 17. However, friendship support at either age did not mediate the link between CA and affective behavioral or neural (AI and dACC) responses to social rejection. | 7 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Kelly et al. (2015) | Total: 122<br><br>CA: 62<br>Control: 60 | 10-14 | Maltreatment<br><br>Child Protection Service records | Perceived access of support from others<br><br>Perceived sense of relatedness scale of the RSCA | Brain structure | Threat process-ing task | Maltreated adolescents showed increased behavioral threat reactivity, greater peer problems, which were partly mediated by reduced GMV in the left mOFC as well as no group specific differences in perceived friendship support. However, it was not specifically tested whether friendship support was associated with reduced mOFC GMV, emotional reactivity, or changes in attentional threat bias in this sample. | 6 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Negriff et al. (2020) | Total: 303<br><br>CA: 303<br>Control: 151 | Four timepoints: 10, 12, 14, 18 | Maltreatment<br><br>Child Protection Service records | Perceived social support (incl. friendships)<br><br>Social Network Interview; CSSQ; MOS-SS | Salivary cortisol, salivary alpha amylase | TSST | At age 10, maltreated adolescents not only reported a smaller perceived friendship support network, compared to the age-matched non-maltreated comparison group, they also showed a blunted cortisol response to social stress, which was especially pronounced for adolescents, who experienced physical or sexual abuse. However, it was not specifically investigated whether the size and interconnections of the friendship support network were associated with lower cortisol responses to social stress. | 5 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Tang et al. (2021) | Total: 217<br><br>CA: 136<br>Control: 135 | Two timepoints: 12, 16 | Institutional rearing<br><br>Bucharest Early Intervention Project | Friendship quality | Sympathetic nervous system reactivity | Social evaluation task | High-quality friendships at age 12 can buffer the indirect effect of blunted SNS reactivity to social rejection feedback on peer problems at age 16 following early institutionalization. | 8 |

**Table 1.** Summary of studies included in the systematic review. Summary of four studies included in the systematic review. Abbreviations: CAMEEI = cambridge early experiences interview; CFQ = cambridge friendship questionnaire; CA = childhood adversity; AI = anterior insula; (d)AAC = (dorsal) anterior cingulate cortex; QA Score = quality assessment score; RSCA = resiliency scale for children and adolescents; GMV = gray matter volume; (m)OFC = (medial) orbitofrontal cortex; CSSQ = child social support questionnaire; MOS-SS = MOS social support survey; TSST = trier social stress test; $T_3$ = Timepoint three; SNS = sympathetic nervous system.

**Discussion**

This pre-registered, systematic review identified only four studies that could have examined whether friendship support buffers neurobiological stress responses in adolescents with CA (Fritz, Stretton, et al., 2020; Kelly et al., 2015; Negriff et al., 2020; Tang et al., 2021). One study found support for friendship stress buffering in a large sample of previously institutionalized adolescents (Tang et al., 2021), whereas two studies did not directly test this model (Kelly et al., 2015; Negriff et al., 2020), and another was limited by an underpowered sample of well-functioning adolescents with mild to moderate CA (Fritz, Stretton, et al., 2020). Previous research (incl. Negriff et al. (2020)) classified individuals with CA exposure as more sensitive and likely to experience interpersonal stress due to compromised social-emotional functioning (Benedini et al., 2016; Humphreys et al., 2016; Kelly et al., 2015; McCrory et al., 2019). Through this mechanism, it is suggested that friendship support may also be less available to young people with CA. However, Kelly et al. (2015) and Fritz, Stretton, et al. (2020) demonstrated that adolescents with CA can have normative or even increased levels of friendships support. This is promising, considering that greater friendship support has been proven to promote mental well-being in this population (van Harmelen et al., 2021) as well as reduce neurobiological responses to social stress in adolescents without CA (Eisenberger et al., 2007; C. L. Masten et al., 2012). Given that friendship stress buffering was only studied in two samples, future research is clearly needed to investigate whether friendships aid mental well-being through reducing neurobiological stress responses in adolescents with CA.

Future research should explore the heterogeneity in CA exposure as well as the types of assessment. For example, a dimensional approach could be used to conceptualize complex CA experiences along distinct dimensions of threat and deprivation in order to capture their impact on neurobiological stress mechanisms (McLaughlin & Sheridan, 2016). However, whether this approach allows the field to ultimately advance from cumulative measures of risk remains to be further investigated (see Pollak & Smith (2021)). Furthermore, previous empirical and meta-analytic evidence has confirmed that prospective documentation (objective) and retrospective self-report (subjective) measures of CA identify individuals with differential neural outcomes and psychopathological risk trajectories (Baldwin et al., 2019; Danese & Widom, 2020). Specifically, understanding and measuring variability in subjective life experiences appears crucial for identifying maladaptive neurobiological stress mechanisms linking CA exposure and risk of psychopathology.

Differential dimensions of friendships (e.g., intimacy or support network size) as well as developmental differences should also be considered. For example, (A. M. Smith et al., 2009) showed that the degree of psychological closeness between same-sex adolescent stranger pairs modulates cortisol responses during a social

stress task. Moreover, Hostinar et al. (2015) showed that parental support becomes less effective in reducing cortisol stress responses (i.e., HPA reactivity) from childhood to adolescence. Hence, future studies should include well powered samples to allow for the investigation of heterogeneity of CA and its assessments as well as friendship dimensions and developmental timing on friendship stress buffering in young people with CA.

In sum, this systematic review identified only two studies that specifically tested whether friendship support buffers neural stress responses in adolescents with CA. Both studies provided divergent evidence for the stress buffering role of friendship support, which is why future research is clearly needed to investigate whether friendships reduce stress vulnerability in young people with CA.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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**Supplementary Information**

| **Search Terms** | |
|---|---|
| *Search category: title, abstract & keywords* | |
| | (friend* OR peer* OR "social support") |
| AND | (child* OR adolescen* OR teen* OR youth* OR "development") |
| AND | ("brain" OR "neural" OR "grey matter" OR "white matter" OR structur* OR function* OR neurobiolog* OR "fmri" OR "mri" OR "diffusion mri" OR "dti" OR "eeg" OR "tms" OR "diffusion tensor imaging" OR "transcranial magnetic stimulation" OR "electroencephalography" OR "magnetic resonance imaging" OR "functional magnetic resonance imaging") |
| AND | (advers* OR maltreat* OR mistreat* OR abuse* OR assault* OR molest* OR neglect* OR victim* OR orphan* OR institutional* OR trauma* OR "deprivation" OR "early life stress" OR "corporal punishment" OR "domestic violence" OR "witnessing intimate partner violence" OR "family conflict" OR "abandonment" OR "physical discipline" OR "bullying") |
| AND | (stress* OR "exclusion" OR "rejection" OR "negative feedback" OR "peer pressure" OR "distress") |
| **Documents** | |
| | peer-reviewed articles, proceedings papers and conference papers |
| **Databases** | |
| | Web of Science Core Collection, PubMed, PsycINFO |

**Table S1.** Search terms and databases used for the systematic review. Overview of search terms and databases used for the pre-registered, systematic review (Prospero: CRD42021233949). Five categories were included in the search strategy to encompass friendship support, study population, neural domains, CA, and stress exposure. In line with the PRISMA guidelines (Page et al., 2021), databases were searched for empirical studies published in English and involving human subjects for all available years through December 2021.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings |
|---|---|---|---|---|---|---|---|
| Kiefer et al. (2021) | 29 | *M* = 13.5 | Peer victimization | - | Brain function | Cyberball | Adolescents with a history of chronic peer victimization showed higher neural activity in frontal brain areas (sg/pgACC, left IFG, and dlPFC) during social exclusion. In addition, this exclusion-specific signal increase was positively related to the severity of prior victimization experience. |
| McIver et al. (2018) | 45 | 17-19 | Peer victimization | - | Brain function | Cyberball | Adolescents with a history of peer victimization showed heightened neural activity (left amygdala, left parahippocampal gyrus, left inferior frontal operculum, and right fusiform gyrus) during social exclusion. |
| McIver et al. (2019) | 45 | *M* = 17.7 | Peer victimization | - | Brain function | Cyberball | In adolescents with CA, functional connectivity between the left amygdala-ACC and left amygdala-mPFC was greater during social inclusion than exclusion, which also predicted a greater risk for depressive symptoms. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Puetz et al. (2014) | 51 | 7-14 | Early separation experiences | - | Brain function | Cyberball | During social exclusion, individuals with CA showed increased affective behavioral and threat-related neural responses (middle temporal gyrus, thalamus, and ventral tegmental area) as well as reduced activity in brain regions implicated in affect regulation (dlPFC and dACC). |
| Rudolph et al. (2016) | 47 | *M* = 15.46 | Peer victimization | - | Brain function | Cyberball | Adolescents with a history of peer victimization showed higher neural activity (dACC) during social exclusion, which was also associated with internalizing symptoms. |
| van Harmelen et al. (2014) | 46 | *M* = 19.2 | Emotional maltreatment | - | Brain function | Cyberball | Severity of maltreatment was positively associated with increased neural responsivity (dorsal mPFC) to social exclusion. |
| Will, van Lier, et al. (2016) | 44 | 12-15 | Chronic peer rejection | - | Brain function | Cyberball | Adolescents with a history of chronic peer rejection showed higher neural activity (dAAC) during social exclusion. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Jarcho et al. (2019) | 47 | *M* = 10.91 | Peer victimization | - | Brain function | Social evaluation paradigm | During unpredictable peer evaluation, adolescents with more severe peer victimization experiences showed greater wariness, which was linked to stronger neural responses (right amygdala). |
| Laceulle et al. (2017) | 141 | Two timepoints: 16, 19 | Childhood adversity | - | Endocrine measures | TSST | Adolescents with a history of social defeat showed increased basal cortisol levels and decreased reactions to the stress task from age 16 to 19, compared to peers with a history of loss/illness as well as those with no CA. |
| Oppenheimer et al. (2020) | 36 | 11-16 | Peer victimization | - | Brain function | Social acceptance & rejection task | Adolescents with greater severity of suicidal ideation exhibited increased neural activation (right anterior insula) during social rejection as well as reported high levels of peer victimization or EMA-measured daily negative social experiences. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Pegg et al. (2019) | 231 | *M* = 18.16 | Lifetime stress exposure | - | ERPs | Island Getaway task (Vote to accept/ reject peers & receive acceptance/ rejection feedback) | Adolescents with greater lifetime social stress exposure and reduced social reward responsiveness showed greater depressive symptoms. |
| Puetz et al. (2016) | 41 | 10-14 | Maltreatment | - | Brain function | Social rejection-themed emotional Stroop task | Maltreated adolescents displayed hypoactivation to rejection cues in regions previously implicated in abuse-related posttraumatic stress disorder (e.g., left anterior insula). |

**Table S2.** Excluded studies examining the impact of childhood adversity on neurobiological stress mechanisms. Twelve articles investigating the impact of childhood adversity on neurobiological stress mechanisms were excluded from the review due to missing friendship support measures. Most studies used social rejection paradigms and found that adolescent with childhood adversity showed greater affective behavioral and neural responsivity during social exclusion. However, these studies were unable to investigate whether friendships buffer neurobiological stress responses. Abbreviations: (sg/pg)ACC = (sub/perigenual) anterior cingulate cortex; IFG = inferior frontal gyrus; (dl)PFC = (dorsolateral) prefrontal cortex; CA = childhood adversity; (d)ACC = (dorsal) anterior cingulate cortex; (d)(m)PFC = (dorsal) (medial) prefrontal cortex; TSST = trier social stress task; EMA = ecological momentary assessment; ERPs = event-related potentials.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings |
|---|---|---|---|---|---|---|---|
| Baddam et al. (2016) | 46 | 8-13 | - | Best friend pairs<br><br>*Note: friendship support was not directly assessed* | ERPs | Cyberball | Youth rejected by a stranger showed greater brain responses (P2 and positive slow wave ERP) compared to being rejected by a friend. |
| C. L. Masten et al. (2012) | 21 | *M* = 17.77 | - | Time spent with friends outside of school each day for 2 weeks | Brain function | Cyberball | Adolescents who spent more time with friends showed reduced neural activity (dACC and anterior insula) during social exclusion. |

**Table S3.** Excluded studies examining friendship buffering of neurobiological stress responses in adolescents without childhood adversity. Two articles investigating friendship buffering of neurobiological stress responses in adolescents were excluded from the review due to a sample without a recorded history of childhood adversity. Only one study did directly assess friendship support and provided initial evidence that friendships may buffer neural stress responses in adolescents without childhood adversity. Abbreviations: ERPs = event-related potentials; (d)ACC = (dorsal) anterior cingulate cortex.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings |
|---|---|---|---|---|---|---|---|
| **Learning** | | | | | | | |
| Gu et al. (2020) | 128 | 9-17 | Orphaned by parental HIV/AIDS | Ability to make friends (i.e., peer social skills) | ERPs | - | Adolescents orphaned by parental HIV/AIDS displayed an increased theta-to-beta power ratio (an EEG marker of decreased cortical activity) in fronto-central regions linked with greater learning and behavioral problems as well as difficulties making friends. However, the authors did not further investigate whether reduced peer social skills impacted the quality of already existing friendships and whether those friendships were associated with reduced cortical resting state activity. |
| Silvers et al. (2016) | 89 | 7-16 | Institutional rearing | - | Brain function | Aversive learning task | Youth who experienced prior institutionalization specifically recruited the hippocampus during aversive learning. Also, connectivity between the hippocampus and PFC prospectively predicted a reduction in anxiety symptoms 2 years later. |

**Reward Processing**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Casement et al. (2014) | 120 | Two timepoints: 11/12, 16 | Peer victimization & low parental warmth | - | Brain function | - | Adolescents who experienced peer victimization showed reduced neural responses (mPFC) to potential rewards, which also mediated the association with depressive symptoms. |
| Eckstrand et al. (2019) | 46 | 14-18 | Peer victimization | - | Brain function | Social reward task | Sexual minority adolescents reported greater interpersonal depressive symptoms and showed blunted neural responses (right mPFC, left anterior insula, right temporoparietal junction) to social, but not monetary, reward. |
| Ethridge et al. (2018) | 61 | 18-25 | Peer victimization | - | ERPs | Doors task (forced choice guessing task) | Adolescents with past-year relational, but not physical, victimization experiences showed blunted brain responses to rewards. |
| Rappaport et al. (2019) | 56 | *M* = 18.05 | Peer victimization | - | ERPs | Social reward task | Adolescents exposed to peer victimization showed reduced brain responses (reward positivity) to social than monetary rewards. |

**Emotion Processing**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Gerin et al. (2019) | 196 | *M* = 19.49 | Childhood adversity | - | Brain function | Face-matching paradigm | Adolescents with CA showed increased baseline neural reactivity (amygdala) to threat and had a greater likelihood of reporting elevated post-baseline exposure to major stressful life events as well as internalizing symptoms at follow-up. |
| Loth et al. (2014) | 1445 | 12-15 | Stressful life events | - | Brain function | Angry faces task | Adolescents showed genotype-dependent neural sensitivity (ventral striatum) to negative social cues as well as the oxytocin receptor gene-genotype rs237915 was found to moderate social and emotional problems after stressful experiences. |
| Weissman et al. (2019) | 179 | 10-16 | Experienced neighborhood and school crime, peer victimization, or discrimination | - | Brain function | Emotional faces task | Adolescents with greater threat exposure showed more internalizing problems as well as stronger negative coupling between the vmPFC and respiratory sinus arrhythmia, which is associated with decreased internalizing problems. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Wymbs et al. (2020) | 55 | 7-16 | Childhood adversity | - | Brain function | Emotional Go-NoGo task | Youth with CA showed increased neural reactivity (amygdala, ACC, insula, nucleus accumbens, and frontal pole) to threat stimuli and the impact of CA on threat processing was found to be moderated by maternal support. |
| **Emotion Regulation** | | | | | | | |
| Curtis & Cicchetti (2007) | 87 | 6-12 | Maltreatment | - | ERPs | - | Maltreated youth showed greater right hemisphere activity in the parietal region of the cortex, whilst non-maltreated youth showed increased activity in left hemisphere activity. |
| Rudolph, Skymba, et al. (2020) | 43 | *M* = 15.44 | Peer victimization | - | Brain function | Emotion regulation task | Adolescents exposed to high levels of peer victimization demonstrated poor emotion regulation (more positive amygdala-right vlPFC connectivity) in the context of negative emotional stimuli. |

**Endocrine Response**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Reid et al. (2021) | 290 | 7-15 | Institutional rearing | Social support (incl. friendships) | Salivary cortisol | - | Quality of social support, but not necessarily previous institutionalization experiences, predicted changes in measures of diurnal cortisol patterns across early adolescence. Specifically, higher levels of social support were associated with lower bedtime cortisol levels. |

**Table S4.** Excluded studies examining the impact of childhood adversity on adolescent functioning across neurobiological, psychological, and social domains. Thirteen articles investigating the impact of childhood adversity on adolescent neurobiological-, psychological-, and social functioning were excluded from the review due to missing friendship support or stress measures. Studies found that adolescents with childhood adversity show greater behavioral (social) problems, blunted neural responses to rewards, maladaptive emotional processing as well as poorer emotion regulation. Abbreviations: ERPs = event related potentials; EEG = electroencephalogram; (m)PFC = (medial) prefrontal cortex; CA = childhood adversity; ACC = anterior cingulate cortex; (vl)-, (vm)PFC = (ventrolateral)-, (ventromedial) prefrontal cortex.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings |
|---|---|---|---|---|---|---|---|
| du Plessis et al. (2019) | 50 | Two timepoints: 9, 14 | Peer victimization | - | Brain structure at 14 & endocrine measures | - | Youth with low daily cortisol output showed greater levels of peer victimization which was associated with smaller right vlPFC surface area at age 14. |
| K. H. Lee et al. (2020) | 152 | 12-17 | Peer victimization | - | Brain structure | - | Adolescent peer problems may have an indirect effect on depressive symptoms through altered (increased) nucleus accumbens volume. |
| Malhi et al. (2019) | 202 | 12-18 | Emotional trauma (abuse, neglect) | - | Brain structure & endocrine measures | - | Smaller left hippocampal volumes were found in adolescents with high emotional trauma carrying the NR3C1 ‘ G’ allele compared to adolescents with mild emotional trauma carrying the same genes and allelic variant. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Malhi et al. (2020) | 202 | 12-18 | Emotional trauma (abuse, neglect) | Perceived social support (incl. friends) | Brain structure | - | Adolescents with severe emotional trauma had smaller left hippocampal volumes as well as less perceived social support from friends, family, and significant others, compared to adolescents with minimal trauma exposure. |
| Teicher et al. (2010) | 848 | 18-25 | Childhood adversity | - | Brain structure | - | Adolescents with CA showed increased mean radial diffusivity and decreased fractional anisotropy in the corpus callosum and the corona radiata. |
| Vargas et al. (2019) | 104 | *M* = 18.02 | Peer victimization | - | Brain structure | - | Adolescents who experienced peer victimization reported greater depressive symptoms and showed smaller mOFC volumes. |

**Table S5.** Excluded studies examining the impact of childhood adversity on adolescent brain structure. Six articles investigating the impact of childhood adversity on adolescent brain structure were excluded from the review due to missing friendship support or stress measures. All studies reported altered brain structure in adolescents with childhood adversity. For example, studies investigating adolescents with severe emotional trauma found smaller left hippocampal volumes as well as less perceived social support in individuals with greater trauma exposure. Moreover, studies investigating adolescents with experienced peer victimization reported surface area reductions particularly surrounding frontal cortical volumes. Abbreviations: (vl)PFC = (ventrolateral) prefrontal cortex; CA = childhood adversity; (m)OFC = (medial) orbitofrontal cortex.

| Paper | *N* | Age (years) | Adversity | Friendship Support | Neuro-biology | Stress | Main Findings |
|---|---|---|---|---|---|---|---|
| Herd et al. (2018) | 167 | Three timepoints: 14, 15, 16 | - | Perceived secure relationship quality (incl. friendships) | Brain structure | - | Adolescent behavioral inhibitory control was found to mediate the link between perceived stress and adolescent secure relationship quality with parents, but not with peers. |
| Holmes et al. (2020) | 91 | 25 | - | - | Brain function | - | Adolescent (deviant) peer relationships at ages 20 and 21 were found to predict frontostriatal resting state connectivity at age 25. |
| Kumar et al. (2019) | 55 | 12-14 | - | - | Brain function | Peer evaluation task | During peer evaluations, adolescents encoded social values in the mPFC. |
| C. L. Masten et al. (2010) | 20 | 12-13 | - | - | Brain function | Cyberball | Adolescents showed greater neural activity (dmPFC, mPFC, precuneus, and pSTS) during observed social exclusion relative to inclusion, which was linked to greater prosocial behavior following exclusion. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| C. L. Masten, Eisenberger, Pfeifer, & Dapretto (2013) | 23 | 12-13 | - | - | Brain function | Cyberball | Adolescents with (heightened sensitivity to) firsthand rejection experiences were more likely to showed greater neural activity in threat-related processing brain regions (incl. dACC and amygdala). |
| C. L. Masten, Eisenberger, Pfeifer, Colich, et al. (2013) | 16 | Two timepoints: 10, 13 | - | - | Brain function | Cyberball | Adolescent pubertal development from age 10 to 13 was positively related to empathic concern and personal distress at age 13 as well as predicted heightened neural responses (dmPFC and temporal pole) when observing social exclusion. |
| McQuaid et al. (2019) | 83 | 11-14 | - *Note: maternal report of negative major life event stress was assessed during pregnancy* | - | Brain structure | - | Adolescents who experienced prenatal stress showed greater gray matter density in the bilateral posterior parietal cortex as well as displayed greater risk for psychiatric symptoms and family system dysfunction. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Rudolph, Davis, et al. (2020) | 45 | 14-16 | - | - | Brain function | Cyberball | Adolescent girls with stressful parent-child relationships reported greater depressive symptoms as well as reduced neural (dACC, subgenual ACC, and anterior insular) responses during social exclusion. |
| Sebastian et al. (2011) | 35 | 14-16 | - | - | Brain function | Cyberball | Compared to adults, adolescents showed attenuated neural reactivity (vlPFC) during social exclusion. |
| Tang et al. (2019) | 166 | 14-28 | - | - | ERPs | Cyberball | Young adolescents (10-17 years) showed greater theta power to social exclusion, compared to older adolescents (18-28 years) and the functional link between theta power to exclusion and self-reported distress was strongest in young adolescents (14-17 years). |
| Tousignant et al. (2018) | 40 | 12-17 | - | - | Brain function | Cyberball | Compared to adults (22-30 years), adolescents (12-17 years) showed reduced neural activation (inferior frontal gyrus) whilst observing social exclusion as well as showed less prosocial behavior towards the excluded player. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Will, Crone, et al. (2016) | 43 | *M* = 14.10 | - | - | Brain function | Cyberball | Adolescents showed higher neural activity (dorsal striatum and lateral PFC) during social exclusion as well as when sharing monetary gains with the excluders. |
| Yeates et al. (2014) | 143 | 8-13 | - | Peer acceptance and friendships | Brain structure | - | Adolescent perspective taking abilities (theory of mind) significantly predicted rejection and victimization by peers, which in turn predicted an increased likelihood of poorer psychosocial outcomes (incl. friendships). |
| **Incorrect Age** | | | | | | | |
| H. J. Chen et al. (2019) | 90 | *M* = 48.6<br><br>*Note: sample mean age outside the adolescent range* | Natural disaster | - | Brain function | - | Traumatized adults showed decreased low-frequency fluctuation in the dACC as well as reduced dACC functional connectivity with the right hippocampus and left cerebellum, compared to controls. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Daniels et al. (2011) | 70 | *M* = 36.24<br><br>*Note: sample mean age outside the adolescent range* | Childhood adversity | - | Brain function | Symptom provocation paradigm | Emotion regulation abilities mediated the link between CA and posttraumatic adjustment. |
| Hyde et al. (2011) | 103 | 31-54<br><br>*Note: sample mean age outside the adolescent range* | - | Perceived availability of social support (incl. friends) | Brain function | Amygdala reactivity paradigm | Individuals with below average levels of perceived social support showed a significant positive correlation between threat-related amygdala reactivity and trait anxiety. |
| Kungl et al. (2017) | 37 | 1-5<br><br>*Note: sample mean age outside the adolescent range* | Foster care | - | ERPs | - | Children in foster care showed dampened brain responses (N170) when viewing stranger and caregiver faces, which was also linked to insecure attachment. |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Lapp et al. (2018) | 90 | *M* = 32.12<br><br>*Note: sample mean age outside the adolescent range* | Childhood adversity | - | Endocrine measures | TSST | Adults with severe CA showed blunted salivary cortisol response to the TSST as well as elevated cortisol levels at pretest, as well as 20- and 40-min post-TSST. |
| Leicht-Deobald et al. (2018) | 31 | 18-65<br><br>*Note: sample mean age outside the adolescent range* | Childhood adversity | - | Brain function | MIST | Individuals with CA who reported higher levels of co-worker social support showed reduced neural reactivity (amygdala) during an arithmetic stress task. |
| Muetzel et al. (2019) | 2602 | Two timepoints: 7/8, 10<br><br>*Note: sample mean age outside the adolescent range* | Peer victimization | - | Brain structure | - | Children who experienced peer victimization showed thicker cortex in the fusiform gyrus compared to those without victimization experiences. |

**Traumatic Brain Injury**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Bigler et al. (2013) | 12 | 8-12 | - | Peer acceptance and friendships | Brain structure | - | Four of the nine youth with TBI had no mutual friends, four also received low peer acceptance ratings, and two were perceived by peers as having high levels of rejection/victimization in the classroom.<br><br>*Note: Empirical studies only including participants with TBI were excluded.* |
| Yeates et al. (2013) | 87 | 8-13 | - | Peer acceptance and friendships | Brain structure | - | Youth with severe TBI demonstrated greater peer relationship problems (incl. higher levels of peer rejection and victimization) as well as a lower likelihood of having a mutual friend, which was related to white matter volumes in several posterior brain regions. |

**Missing Multiple Search Criteria**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Say et al. (2014) | 92 | 11-18 | Childhood adversity | - | - | - | Adolescents with psychogenic non-epileptic seizures were more likely to report CA as well as lower levels of self-esteem. |
| van Harmelen et al. (2016) | 771 | Two timepoints: 14, 17 | Childhood adversity | Friendship quality | - | - | Friendship support in 14-year-old adolescents with CA may reduce depressive symptoms at age 17. |

**Missing Empirical Research**

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Beauchamp et al. (2020) | - | - | - | - | - | - | *Note: No findings available due to protocol paper.* |
| Bruijel et al. (2018) | - | - | - | - | - | - | *Note: No findings available due to protocol paper.* |
| Cryan & Dinan (2013) | - | - | - | - | - | - | *Note: No findings available due to commentary.* |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Hoffman et al. (2019) | - | - | - | - | - | - | *Note: No findings available due to protocol paper.* |
| McLaughlin et al. (2020) | - | - | - | - | - | - | *Note: No findings available due to review paper.* |

**Table S6.** Excluded studies with multiple missing search criteria. Twenty-nine articles were excluded from the review due to multiple missing search criteria. Thirteen studies recruited adolescents without a recorded history of childhood adversity. The sample mean age of seven articles fell outside the predefined adolescent age range (10-24 years). Two studies only included patients with traumatic brain injury and two more studies did not collect any neurobiological stress measures. Finally, five studies were excluded due to missing empirical research (incl. protocol, commentary, or review papers). Abbreviations: (dm)PFC = dorsomedial prefrontal cortex; pSTS = posterior superior temporal sulcus; (d)ACC = (dorsal) anterior cingulate cortex; (vl)PFC = (ventrolateral) prefrontal cortex; CA = childhood adversity; ERPs = event-related potentials; TSST = trier social stress task; MIST = montreal imaging stress task; TBI = traumatic brain injury.

| | Risk of Bias (Quality) Assessment |
|---|---|
| 1. | Did the study address a clearly defined question? |
| 2. | Is the sample representative of the defined population? |
| 3. | Was the target group appropriately matched to a control group? |
| 4. | Were well-established measure(s) used to assess friendship support? |
| 5. | Were well-established measure(s) used to assess CA? |
| 6. | Did the authors account for all confounding factors? |
| 7. | Did the authors provide a justification that their sample size is appropriate beyond just citing convention in the literature? |
| 8. | Did the authors report effect sizes or confidence intervals for the main findings? |
| 9. | Did the authors ensure the reproducibility of their research findings? |

**Table S7.** Summary of nine risk of bias (quality) assessment questions. For each available quality marker articles could score one point and up to nine points in total. Assessments were performed by two independent reviewers.

# Chapter 4

## The Stress-Buffering Role of Friendships in Young People with Childhood Threat Experiences: A Preliminary Report

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**Abstract**

**Background:** High-quality friendships have a positive impact on the mental health of young people with childhood adversity (CA). Social stress buffering, the phenomenon of a social partner attenuating acute stress responses, is a potential yet unexplored mechanism that may underlie this relationship. **Objective:** This study examined whether perceived friendship quality was related to better mental health and lower neural stress response in young people with CA. **Method:** A total of $N = 102$ young people (aged 16-26) with low to moderate CA were included in the study. We first investigated associations between friendship quality, mental health, and CA. In a representative subset ($n = 62$), we assessed neural stress responses using the Montreal Imaging Stress Task. In our sample, CA was best described along two dimensions resembling threat or deprivation like experiences. Hence, we investigated both cumulative and dimensional effects of CA. **Results:** We found no support for social thinning after CA, meaning that the severity of CA (cumulative or dimensional) did not differentially impact friendship quality. High-quality friendships, on the other hand, were strongly associated with better mental health. Furthermore, acute stress increased state anxiety and enhanced neural activity in five frontolimbic brain regions, including the left hippocampus. We found weak support that threat experiences interacted with friendship quality to predict left hippocampal reactivity to stress. However, this effect did not survive multiple comparison correction. **Conclusion:** The absence of social thinning in our sample may suggest that the risk of developing impoverished social networks is low for rather well-functioning young people with low to moderate CA. Regardless, our findings align with prior research, consistently showing a strong association between high-quality friendships and better mental health in young people with CA. Future research is needed to examine whether friendships aid neural stress responses in young people with childhood threat experiences.

*Keywords*: childhood adversity, threat experiences, hippocampus, neural stress mechanisms, friendship quality, young people

**Highlights**

- Young people with childhood adversity underwent acute stress induction, eliciting frontolimbic reactivity.
- High-quality friendships were strongly associated with better mental health.
- Weak support for friendship stress buffering did not survive multiple comparison correction.

## Introduction

Up to half of children and adolescents worldwide experience at least one form of childhood adversity (CA), such as abuse, neglect, bullying, or poverty (Bellis et al., 2014). Exposure to CA represents a deviation from the "expectable" environment, which requires young people to adapt their psychological, social, and neurobiological functioning, ultimately putting them at greater risk for prolonged mental health problems (Cicchetti & Valentino, 2006; Clark et al., 2010; Kessler et al., 2010; McLaughlin, 2016). In fact, around a third of lifetime mood, anxiety, and substance use disorders can be attributed to CA (Green et al., 2010). Hence, investigating neurodevelopmental mechanisms that underlie mental health vulnerability and identifying protective factors that buffer these risk pathways is crucial for informing the development of effective psychosocial interventions.

Chronic or repeated exposure to CA has programming effects on key stress response systems, which can increase later-life mental health vulnerability (Lupien et al., 2009). For example, repeated activation of the hypothalamic-pituitary-adrenal (HPA) axis leads to elevated levels of stress hormones (glucocorticoids) in the body (McEwen, 2017). This neuroendocrine response to prolonged psychosocial stress may promote adaptive functioning (e.g., increased alertness) in the short-term to support survival in stressful environments. However, over time, sustained activation of this stress response system may be detrimental to the structure and function of stress-sensitive brain regions (Arnsten, 2009; Y. Chen & Baram, 2016; Danese & McEwen, 2012). Frontolimbic brain regions, including the hippocampus, amygdala, and prefrontal cortex (PFC), may be particularly sensitive to stress in the context of chronic CA exposure due to their dense innervation with glucocorticoid receptors (Cohodes et al., 2021; Ioannidis et al., 2020; Tottenham & Sheridan, 2010).

Given the importance of the frontolimbic system for social information and emotional processing (Humphreys et al., 2016; McLaughlin et al., 2020), alterations to that system following CA may increase mental health vulnerability, as suggested by McCrory et al. (2022). Their social transactional model of mental health vulnerability posits that through neurocognitive adaptations to high stress environments, young people with CA might become more likely to subsequently experience (interpersonal) stress (i.e., "stress generation"; McCrory et al. (2019)) and attenuation in their support networks (i.e., "social thinning"; Nevard et al. (2021); Sheikh et al. (2016)), contributing to greater mental health vulnerability.

Critically, social support is a key protective factor against the emergence of mental health problems in young people with CA (Li et al., 2022; Trickey et al., 2012). However, little is known about the underlying mechanisms of this relationship. Social stress buffering models suggest that the presence of a social partner can reduce physiological responses to acute psychosocial stress, measured by

glucocorticoid blood levels (Gunnar, 2017; R. M. Sullivan & Perry, 2015). During childhood, caregiver support suppresses cortisol secretion to acute psychosocial stress (Hostinar et al., 2015), dampens amygdala reactivity, and promotes emotion regulation (Gee et al., 2014) in children without CA. In addition, previously institutionalized children with greater self-reported feelings of caregiver security exhibited reduced amygdala reactivity to caregiver cues, which was also predictive of a greater decrease in future anxiety symptoms (Callaghan et al., 2019). During adolescence, a unique time of social reorientation and increased sensitivity to peers (Cosme et al., 2022), friendship support takes on a more potent stress buffering role, capable of protecting against the emergence and progression of mental health problems following CA (van Harmelen et al., 2016, 2021). Preliminary evidence for friendship stress buffering has shown that the more time spent interacting with supportive friends was associated with diminished neurobiological stress responses (i.e., reduced cortisol, dorsal anterior cingulate cortex (dACC), and anterior insula reactivity) in young people without CA (Eisenberger et al., 2007; C. L. Masten et al., 2012). To date, only two studies have examined friendship stress buffering in young people with CA and reporting mixed results, for a systematic review see (Scheuplein & van Harmelen, 2022). Tang et al. (2021) showed that high-quality friendships were associated with improved sympathetic nervous system reactivity to social rejection feedback at age 12 and reduced peer problems at age 16 in early institutionalized young people. In contrast, no support for friendship stress buffering was found in a small community sample of well-functioning adolescents with low to moderate CA (Fritz, Stretton, et al., 2020). Therefore, it remains unclear whether high-quality friendships aid mental health and well-being in young people with CA through dampening neurobiological stress responses.

To investigate friendship stress buffering in young people with CA, it is crucial to clearly quantify CA experiences, whilst accounting for the fact that different types of CA often co-occur (Brown et al., 2019; Dong et al., 2004). On the one hand, the cumulative-risk approach assumes that discrete forms of CA have additive, but not distinct, effects on neurocognitive functioning, with more CA being associated with stronger effects. Hence, this prevailing approach combines the number of distinct types of adverse experiences into a cumulative risk score (Evans et al., 2013). This approach has been highly influential in public policy and clinical practice (Lacey & Minnis, 2020). On the other hand, dimensional models of adversity differentiate between experiences of *threat/harshness* (involving harm or threat of harm to oneself and others), *deprivation* (involving absence of expected cognitive and social stimulation), and *unpredictability* (involving spatial-temporal variation in threat) to identify mechanisms linking these partially distinct experiences of CA with unique neurodevelopmental and psychopathological consequences (B. J. Ellis et al., 2009; Humphreys & Zeanah, 2015; McLaughlin et al., 2021; Sheridan & McLaughlin, 2014). In line with this

framework, Puetz et al. (2020) showed that different forms of childhood maltreatment (abuse, neglect, and their combination) were associated with differential neural processing of threat-related cues. Specifically, childhood abuse was associated with increased localized ventral amygdala reactivity to threat, whereas childhood neglect was associated with heightened reactivity in the dorsal amygdala and across spatially distributed frontoparietal brain networks. Notably, cumulative experiences of abuse and neglect were associated with hypoactivation in various higher- order cortical regions, in addition to the amygdala. Furthermore, a systematic review by McLaughlin, Weissman, et al. (2019) investigated differential associations between threat and deprivation experiences and neural development. To summarize their key findings, threat experiences were found to influence frontolimbic neural networks involved in threat detection and emotion regulation (amygdala and medial prefrontal cortex (mPFC)), salience processing (insula, ACC), and various forms of learning and memory (hippocampus). Deprivation experiences, on the other hand, were found to influence frontoparietal circuits (dorsolateral prefrontal cortex (dlPFC) and superior parietal cortex) contributing to working memory and cognitive control. However, these neural patterns were not consistently observed across studies, highlighting the need for more neuroimaging research to establish consistent and replicable associations between brain alterations and different dimensions of CA.

The main goal of this study was to investigate whether perceived friendship quality was related to reduced neural stress responses in a sample of adolescents and young adults (aged 16-26) with low to moderate CA. In addition, we examined relations between CA, friendship quality, and mental health and well-being. To challenge neural stress responses affected by CA, we utilized the Montreal Imaging Stress Task (MIST) (Dedovic et al., 2005). The MIST is a well-validate acute psychosocial stress paradigm combining the stress-eliciting effects of high cognitive demands (solving math problems under time pressure) with negative social feedback (on screen and verbally via the experimenter), see review by Noack et al. (2019). Previous studies that utilized the MIST reported stress-related activation in frontolimbic regions, including the hippocampus, amygdala, insula, mPFC, ACC, nucleus accumbens (NAc), and thalamus (Chung et al., 2016; Noack et al., 2019; Voges et al., 2022; Wheelock et al., 2016). Hence, we examined the neural correlates of friendship stress buffering in these regions of interest (ROIs).

Specifically, we examined three hypotheses. First, we hypothesized that more severe CA experiences would be associated with lower levels of perceived friendship quality (McCrory et al., 2022) and that reduced friendship quality would be associated with worse mental health functioning (Fritz, Stretton, et al., 2020; van Harmelen et al., 2016, 2021) (hypothesis 1). Second, we expected that acute psychosocial stress would increase state anxiety (Chung et al., 2016; Zschucke et al., 2015) and increase neural activity in our ROIs (Noack et al., 2019)

(hypothesis 2). Third, we expected that friendship quality would moderate the relationship between CA and neural stress responses. Specifically, we investigated whether higher friendship quality would be associated with reduced frontolimbic ROI reactivity to stress (Scheuplein & van Harmelen, 2022; Tang et al., 2021) (hypothesis 3). Finally, we examined these hypotheses with both a cumulative-risk approach and a dimensional approach. We expected that more CA experiences, or more severe threat experiences, would be associated with greater frontolimbic ROI reactivity to stress (McLaughlin, Weissman, et al., 2019).

## Method

### *Resilience after Individual Stress Exposure (RAISE) Study*

Data were drawn from the Resilience after Individual Stress Exposure (RAISE) study, a multilevel study of $N$ = 102 young people aged 16-26 (Moreno-López et al., 2021). All RAISE participants retrospectively self-reported a history of CA, which was defined as exposure to any adverse life event experienced within the family environment before the age of 16. This included childhood maltreatment (e.g., emotional, sexual, or physical abuse, emotional or physical neglect) or intrafamily adversity (e.g., marital distress or conflict, parental mental health problems or parental alcohol dependence, violence, or aggressive behavior) (Figure 1). Participants were recruited across Cambridgeshire, UK from the general population through flyers and via social media as well as from previous studies conducted at the Department of Psychiatry, University of Cambridge (NSPN 2400 Cohort; Kiddle et al. (2018)). The RAISE study has received funding from the Royal Society in January 2018, ethical approval from the National Research Ethics Service and the NRES Committee East of England-Cambridge Central (REC reference: 18/EE/0388, IRAS project ID: 241765) in February 2019, and commenced in August 2019.

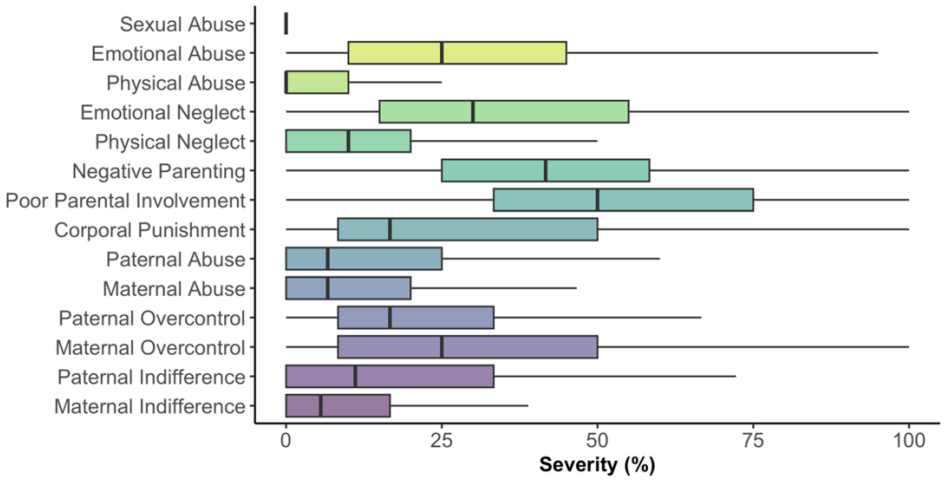

**Figure 1.** Severity of childhood adversities in the baseline sample. Severity (in percent; x-axis) of individual childhood adversities (y-axis) retrospectively self-reported by all *N* = 102 young people, who participated during the first assessment timepoint (T1) of the RAISE study. Each boxplot displays the median severity (solid vertical line) and interquartile range. Based on established cut-off scores for the CTQ (Bernstein et al., 1994), this baseline sample can be characterized reporting low to moderate levels of CA. Summary statistics are provided in the supplementary Table A.1.1.

***Participants***

This study utilized data from the first two RAISE assessment timepoints. At timepoint 1, participants completed online questionnaires (*N* = 102; "Baseline sample"). Timepoint 2 was completed on average 1 month later and consisted of an in-unit visit at Addenbrooke's Hospital in Cambridge, UK during which, among other measures, functional magnetic resonance imaging (fMRI) data was acquired (*n* = 62; "Neuroimaging sample"). At each timepoint, informed consent was obtained from the participant. A comprehensive description of the full study procedure as well as the inclusion and exclusion criteria has been previously published by Moreno-López et al. (2021). To summarize, individuals were eligible to participate if they were aged between 16-26 years, able to speak, write, and understand English, had a body mass index (BMI) between 18.5 and 29.9 kg/m$^2$, did not currently take medication (e.g., corticosteroids) likely to compromise the interpretation of our data, had no MRI contraindications, and self-reported CA experienced before the age of 16. All inclusion and exclusion criteria were assessed via telephone by a trained member of the study team to ensure that interested participants were eligible. Medication use and BMI were re-assessed by a trained research nurse at the day of scanning. Participants received a total of £150 upon completing all three study phases (please note that data from the third study phase was not included in the analysis for this study). If participants chose not to proceed after the first or second phase, they were partially reimbursed. The payment was distributed as follows: £10 for the initial completion of online questionnaires and three cognitive tasks, £100 for their attendance at Addenbrooke's Hospital, and £40 for completing the second set of online questionnaires. This study commenced in August 2019 and was terminated prematurely in March 2020, prompted by a University-wide closure of laboratory research activities and the redirection of clinical research facilities toward COVID-19 related studies. Hence, *n* = 42 participants who completed the baseline assessment could not be assessed at timepoint 2. However, key characteristics (e.g., age, gender, CA experiences, or friendship quality) are comparable between the neuroimaging sample and baseline and the participants who could not complete the study as a consequence of the COVID-19 pandemic (Table A.1.1).

### *Baseline Assessments (T1)*

At baseline, participants received an email containing an online link to remotely complete self-report questionnaires assessing past CA experiences as well as current (past two to four weeks) mental health, well-being, and friendship quality. Specifically, *CA* was assessed with the Short-Form of the Childhood Trauma Questionnaire (CTQ-SF; Bernstein et al. (2003)), the Measure of Parental Style Questionnaire (MOPS; Parker et al. (1997)), and the Alabama Parenting Questionnaire (APQ; Frick (1991)). *Mental health and well-being* (in the following referred to as psychosocial functioning) was assessed with the Mood and Feelings Questionnaire (MFQ; Angold & Costello (1987)), Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond (1978)), the Leyton Obsessional Inventory-Child Version (LOI-CV; Bamber et al. (2002)), the Behavioral Checklist (BCL; van Harmelen et al. (2017)), the Rosenberg Self-Esteem Scale (SES; Rosenberg (1965)), the Kessler Psychological Distress Scale (K10; Kessler et al. (2002)), the Warwick-Edinburgh Mental Well-Being Scale (WEMWBS; Tennant et al. (2007)), and the Drugs and Self Injury Questionnaire (DASI; Wilkinson et al. (2018)). *Friendship quality* was assessed with the Cambridge Friendship Questionnaire (CFQ; van Harmelen et al. (2017)). Across all questionnaires, higher scores reflect more severe CA experiences, better psychosocial functioning, and greater perceived friendship quality. A detailed description of all questionnaires is provided in the supplementary information (section B). Given that we recruited adolescents and young adults aged 16-26 ($M_{age}$ = 22.24 at baseline), we chose these measures to ensure that all questionnaires (incl. instructions and items) were accessible and age-appropriate for the entire sample (Demkowicz et al., 2020), which is also in line with similar approaches utilized in previous large-scale longitudinal cohort studies assessing young people aged 14-24 (Goodyer et al., 2010; Kiddle et al., 2018).

### *In-Unit Assessments (T2)*

During the in-unit visit, participants completed the vocabulary and matrix reasoning subtests of the *Wechsler Abbreviated Scale of Intelligence* (WASI-II; Wechsler (2018)) from which age-normed IQ scores were derived. IQ scores ranged from 78 to 138 ($M_{IQ}$ = 116.09, $SD$ = 10.18). Furthermore, the *Edinburgh Handedness Inventory* (EHI; Oldfield (1971)) indicated that 91% of participants preferred using the right hand for more complex manual tasks. Furthermore, state anxiety was assessed with the *State-Trait Anxiety Inventory* (STAI; Spielberger & Vagg (1984)) before and after participants completed the MIST in the MRI scanner.

#### *fMRI Stress Paradigm*

The *Montreal Imaging Stress Task (MIST)* is a well-validated and widely used acute psychosocial stress paradigm for fMRI (Berretz et al., 2021; Chung et al., 2016; Corr et al., 2021; Dedovic et al., 2005, 2009; Noack et al., 2019; Pruessner

et al., 2008). This computerized mental arithmetic task with an artificially induced failure component was presented as a block design across two imaging runs. Each run lasted 11 min and consisted of a stress, control, and rest condition (Figure 2). The order of these conditions was counterbalanced across participants to avoid order effects.

During the 5 min *stress condition* (Figure 2A), participants were asked to answer math problems of varying difficulty under time constraints whilst receiving trial-by-trial on screen performance feedback ("correct" in green, "error" in red, or "timeout" in yellow). To answer, participants were provided a button box and instructed to navigate left or right on a rotary-dial to the correct digit (between 0 and 9). In addition, a performance bar at the top of the screen continuously displayed the "average" performance of previous participants (artificially set to 80%) as well as the participant's current performance. Participants were instructed to attain or surpass the average performance of their peers. To induce a high failure rate, the participant's response time limit got adjusted throughout the task to enforce a range of approximately 20% to 45% correct responses (Dedovic et al., 2005). Specifically, participants were given 10% less time after three consecutive correct responses and 10% more time after three consecutive incorrect or timeout responses. To further induce psychosocial stress, participants were presented with a 5 sec on screen summary of their current performance and were reminded that their "performance should be close to or better than the average performance". This summary was presented at five timepoints during the stress condition. In addition, participants received scripted negative verbal feedback in between runs from a member of the study team saying: "Your performance is below average. In order for your data to be used, your performance should be close to or better than the average performance. Please try as hard as you can next round".

During the 5 min *control condition* (Figure 2B), participants answered math problems of the same difficulty level and received trial-by-trial performance feedback ("correct" in green, "error" in red). However, no time constraints were enforced, the performance bar (including the "average" peer performance) was not displayed, and participants were instructed that their performance would not be recorded.

During the 1 min *rest condition* (Figure 2C), participants were presented with the empty task interface and asked to keep their eyes open and not press buttons until the next math problem would appear.

The MIST took approximately 35 min to complete including approximately 5 min of practice outside the MRI scanner to familiarize participants with each condition. For this study, we used an adapted version of the MIST originally

programmed by Borchert (2019) for Millisecond Software, LLC (openly available at: https://www.millisecond.com/download/library/montrealstresstest).

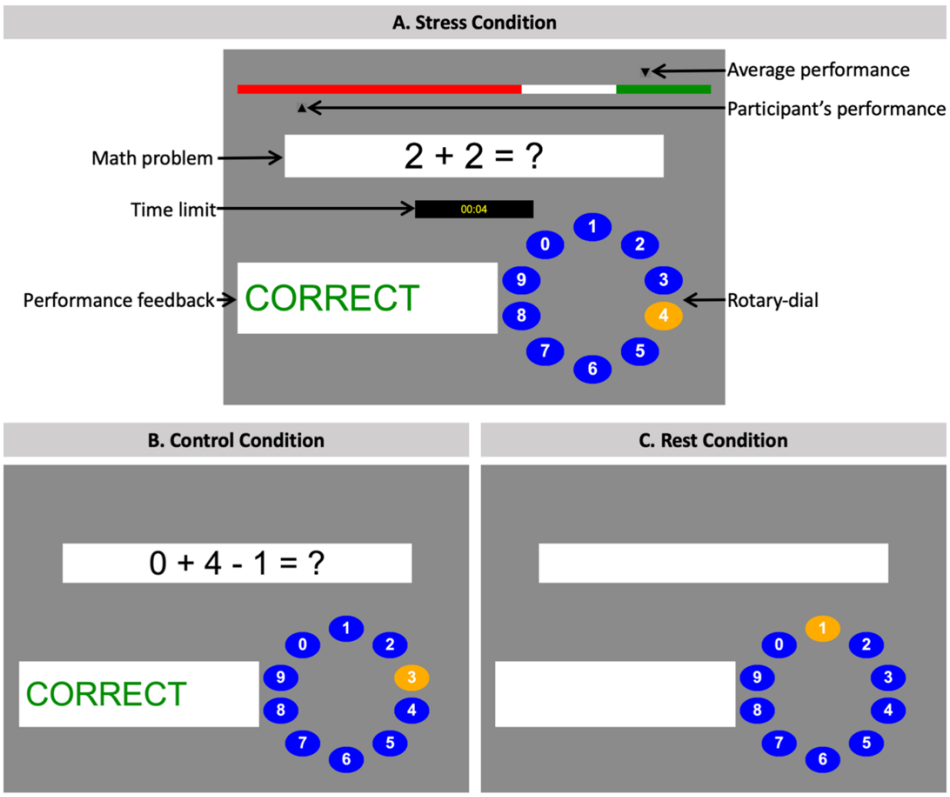


**Figure 2.** Graphical user interfaces of the Montreal Imaging Stress Task. **(A)** Stress condition: from top to bottom, the figure shows the performance bar displaying the average performance of previous participants (artificially set to 80%) as well as the participant's current performance. Below, participants were presented with math problems of varying difficulty whilst being shown the titrated time limit, they had to provide a response. A response was submitted via the rotary-dial (answer choices between 0 and 9). Finally, participants received trial-by-trial on screen performance feedback ("correct" in green, "error" in red, or "timeout" in yellow). **(B)** Control condition: participants answered math problems of the same difficulty level and received trial-by-trial performance feedback ("correct", "error"). However, no time constraints were enforced, the performance bar was not displayed, and participants were instructed that their performance would not be recorded. **(C)** Rest condition: participants were presented with the empty task interface and asked to keep their eyes open and not press buttons until the next math problem would appear.

### *Imaging Procedures*

*fMRI Data Acquisition*

fMRI was conducted on a Siemens 3T Magnetom Prisma Fit whole body MRI scanner (Siemens Healthcare GmbH, Erlangen, Germany) with a 32-channel head coil. Blood oxygen level-dependent (BOLD) data were collected using a T2*-weighted transversal echo planar imaging (EPI) sequence with interleaved slice acquisition, covering the entire brain (repetition time (TR) = 2000ms, echo time (TE) = 30ms, flip angle = 78°, number of slices = 34, slice thickness = 3mm, slice gap = 0.3mm, voxel size = 3 x 3 x 3mm$^3$, field of view (FOV) = 192 x 192mm$^2$, in-plane resolution = 64 x 64). To obtain a 3D structural scan, high-resolution sagittal T1-weighted images were acquired using a magnetization prepared-rapid gradient echo (MPRAGE) sequence (TR = 2000ms, TE = 2.98ms, , flip angle = 9°, number of slices = 176, slice thickness = 1mm, slice gap = 0.5mm, voxel size = 1 x 1 x 1mm$^3$, FOV = 256 x 256mm$^2$, in-plane resolution = 256 x 256).

*fMRI Preprocessing and Data Analysis*

Preprocessing of the imaging data was performed using SPM12 (https://www.fil.ion.ucl.ac.uk/spm/) implemented in MATLAB (version R2020a; MathWorks) following standard procedures. To summarize, images were realigned to the mean image of the scan run using a 6-parameter rigid body spatial transformation, spatially normalized to the standard stereotactic space of the Montreal Neurological Institute (MNI) template, resampled to 3 mm isotropic voxels, and smoothed with an 8mm full-width at half-maximum (FWHM) Gaussian kernel. In addition, framewise displacement (FD) was computed based on the head motion parameter and used as quality checks (Power et al., 2012). As recommended by Schwarz et al. (2020), participants with a $M_{FD}$ > 0.5mm or more than 20% volumes with FD > 0.5mm in any of the two runs were excluded from subsequent analyses. Based on this rule and through visual inspection, $n$ = 2 participants were excluded, leaving a total neuroimaging sample of $n$ = 60 (Table A.1.1).

For the first-level analysis, we defined a general linear model (GLM) for each subject and each condition of the MIST (convolved with the canonical hemodynamic response function (HRF) of SPM12). Six head motion parameters from the realignment step were included as covariates. A high-pass filter with a cut-off frequency of 1/262 Hz and an autoregressive model of the first order were applied. To identify regions showing greater activation (i.e., greater mean BOLD signal) during the stress condition compared to the control condition, we computed stress > control first-level contrasts for each participant. This contrast allowed for investigating the effect of acute psychosocial stress on brain activation whilst controlling for activation changes induced by mental arithmetic.

For the second-level analysis, single-subject contrast maps were entered into random effects analyses (one-sample *t*-test). Based on our a-priori hypotheses, ROI analyses were performed using bilateral masks for the hippocampus, amygdala, insula, mPFC, ACC, NAc, and thalamus. ROI analyses were conducted using the pipeline implemented in the Wake Forest University (WFU) PickAtlas SPM12 toolbox (version 3.0.5; Maldjian et al. (2004), (2003); https://www.nitrc.org/projects/wfu_pickatlas/). Specifically, these ROIs were defined using the PickAtlas GUI and resliced to match smoothing. Given that the anatomical region of the mPFC is less well defined, this ROI mask was based on the anatomical location of both dorsal and ventral mPFC (including the ACC; Brodmann areas (BA): 9, 10, 11, 24, 25, 32) (Moreno-López et al., 2020; Passingham & Wise, 2012; van Harmelen et al., 2013). All other ROIs were based on the Automated Anatomical Labeling (AAL) atlas (Tzourio-Mazoyer et al., 2002). ROI results were familywise error (FWE) corrected ($p_{FWE} < .05$) using voxel-level statistics. For all ROIs that showed a significant main effect of task, we extracted individual beta weights by averaging across all activated voxels in the cluster containing the ROI peak (Tong et al., 2016). We applied no restriction for the minimum cluster size. To comprehensively examine neural activation outside our ROIs, we additionally conducted follow-up exploratory whole-brain analyses ($p_{FWE} < .05$) using the same stress > control contrast, k > 25 voxels.

### *Principal Component Analysis*

Principal component analyses (PCAs) with oblique rotation were used to explore differential dimensions of CA experiences and to capture the range of psychosocial outcomes in our sample. Specifically, we computed principal component (PC) scores, weighted by their explained variance for CA and psychosocial functioning, respectively. The PCA for CA revealed a two-component solution so that CA could be delineated along two dimensions resembling *threat* and *deprivation* experiences. Those dimensional scores were subsequently combined into a *cumulative CA index*, with a higher index indicating more severe CA (Figure 3A). Specifically, this PCA was conducted on standardized individual total scores of the Measure of Parental Style Questionnaire (MOPS; measure of maternal and paternal abuse, indifference, and overcontrol), the Short-Form of the Childhood Trauma Questionnaire (CTQ-SF; measure of physical abuse, emotional abuse, physical neglect, and emotional neglect), and the Alabama Parenting Questionnaire (APQ; measure of corporal punishment, poor parental involvement, and negative parenting). The PCA for psychosocial functioning revealed a three-component solution, and we summed the weighted PC1, PC2, and PC3 scores to compute a *cumulative psychosocial functioning index*, with a higher index indicating better mental health and well-being (Figure 3B). This PCA was conducted on standardized individual total scores of the Warwick-Edinburgh Mental Well-Being Scale (WEMWBS; measure of mental well-being), the Revised Children's Manifest Anxiety Scale (RCMAS; measure of physiological anxiety,

worry/oversensitivity, and social concerns/concentration), the Mood and Feelings Questionnaire (MFQ; measure of depressive symptoms); the Rosenberg Self-Esteem Scale (SES; measure of self-esteem); the Kessler Psychological Distress Scale (K10; measure of psychological distress); the Leyton Obsessional Inventory-Child Version (LOI-CV; measure of compulsions, obsessions, and cleanliness), and the Behavioral Checklist (BCL; measure of behavioral problems). Please note that all results hold when only using the weighted PC1 score for psychosocial functioning. A similar method has been employed by Anand et al. (2019) and a detailed description of our analyses as well as a summary of the PC scores and their associations can be found in our supplementary information (section E).

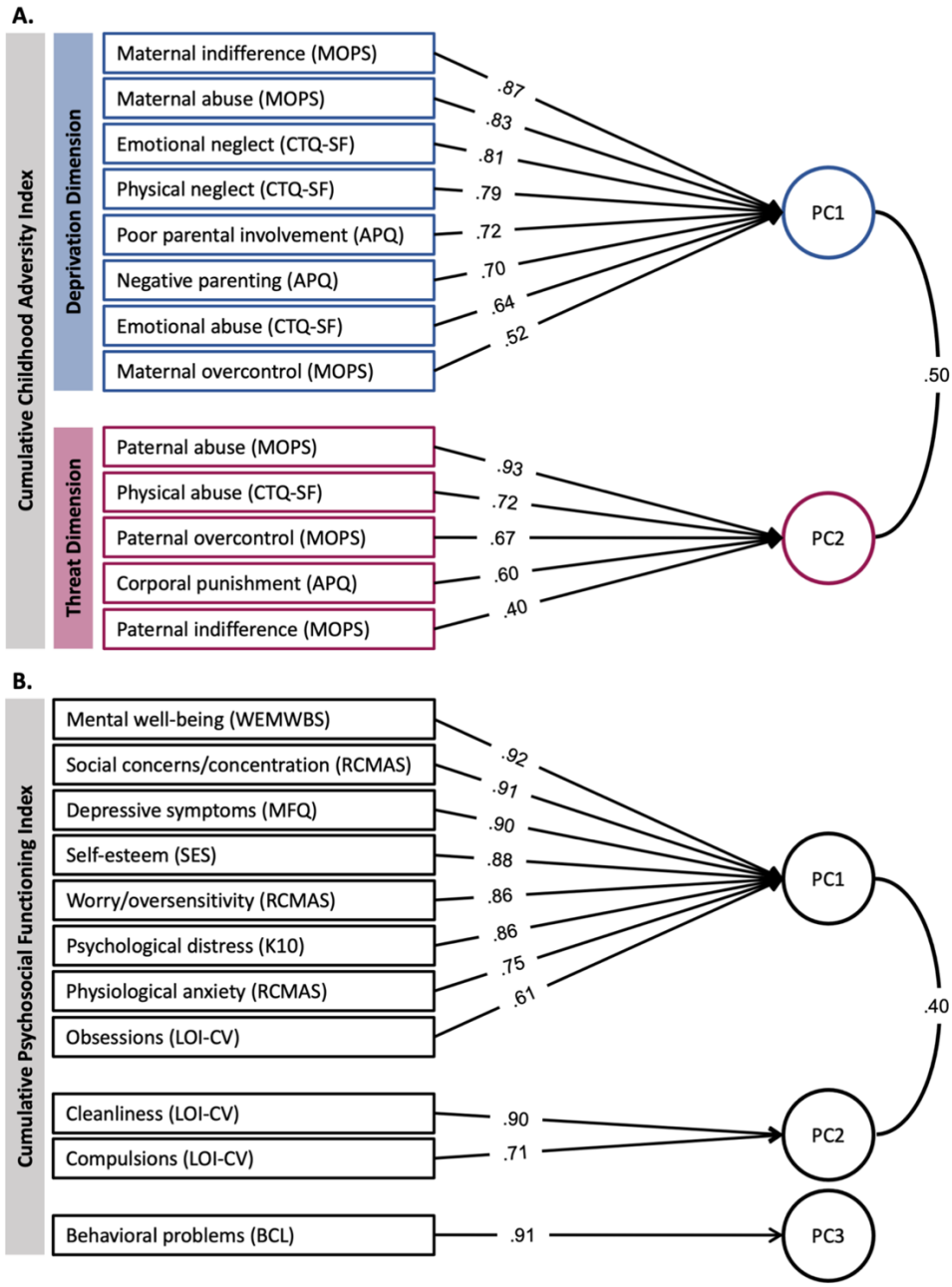


**Figure 3.** Principal component analysis loading matrices for childhood adversity and psychosocial functioning. Two PCAs with oblique rotation were conducted on individual scores of **(A)** CA measures and **(B)** psychosocial functioning measures. The PCA for CA resulted in two principal components (PCs) which were further divided into a *deprivation dimension* (blue; PC1 explaining 37% of variance) and a *threat dimension* (red; PC2 explaining 21% of variance). To account for the

contributions of both PCs, we weighted the scores for each PC by their explained variance and subsequently summed these scores to compute a single index of total severity experienced (*cumulative CA index*). The PCA for psychosocial functioning resulted in three PCs (PC1 explained 55% of variance; PC2 explained 15%; PC3 explained 10%). To compute a *cumulative psychosocial functioning index*, we summed the weighted PC1, PC2, and PC3 scores. Factor loadings of each measure are displayed on the arrows.

***Statistical Analyses***

First, we analyzed behavioral data collected at baseline (T1; $N = 102$). Specifically, we examined associations between friendship quality and CA through linear regression models. We ran separate models for the cumulative CA index (derived through summing the weighted PC1 and PC2 scores) as well as the weighted deprivation (PC1) and threat dimensions (PC2). In addition, we examined associations between friendship quality and the cumulative psychosocial functioning index (hypothesis 1). All models included age at the time of assessment and gender identity as covariates. To handle missing questionnaire data, we derived sum scores from scales with ≥15 items if 85% or more of the items were answered. For scales with less than 15 items, a sum score was only derived if 100% of the items were answered. This resulted in 2.45% of missing data.

Second, we analyzed data collected during the in-unit assessment (T2; $n = 60$). We used a paired *t*-test to examine individual mean differences in state anxiety before and after completing the MIST in the MRI scanner. Afterwards, we investigated overall task effects in our predefined ROIs (hypothesis 2) and then examined associations between CA and friendship quality on stress-induced significant ROI reactivity during stress (> control) trials of the MIST (hypothesis 3). We ran separate moderated multiple regression models for the cumulative CA index, the deprivation, and threat dimensions. All multiple regression models included age at the time of scanning and gender identity as covariates. As our stressor comprised of a timed arithmetic test, we further added IQ as a covariate to all models. Furthermore, friendship quality scores were mean centered to align the scaling of the predictor variables and thereby enhance interpretation of the multiple regression results (Iacobucci et al., 2016).

All statistical analyses outlined above were run in R version 3.6.3 (R Core Team, 2022). The PCAs were performed using the psych R package (version 2.2.9; Revelle (2022)) and mean imputations to replace missing values were performed using the mice R package (version 3.15.0; Van Buuren & Groothuis-Oudshoorn (2011)). Regression models were run using the stats R package (version 3.6.3). Partial Cohen's f-squared ($f_p^2$) and Cohen's d ($d$) effect size estimates are reported for all relevant tests. Significance was set at $p < .05$ throughout all analyses unless stated otherwise and all tests were Bonferroni corrected for multiple comparisons

(# of models tested). In addition, we used the Median Absolute Deviation (MAD) method (i.e., median plus or minus 3 times the MAD; Leys et al. (2013)) in combination with the Rosner's test (EnvStats R package version 2.7.0; Millard (2013)) to detect and exclude potential outliers. Moreover, to visualize significant interactions, we plotted model estimated marginal means using the sjPlot R package (version 2.8.14; Lüdecke (2023)) alongside 95% confidence intervals. Specifically, we explored how the relationship between CA and stress-induced ROI reactivity changed as a function of low and high friendship quality (-1*SD*, +1*SD*). For statistical power considerations, please refer to the supplementary information (section F).

## Results

### *Behavioral Results (T1; N = 102)*

First, we did not observe that participants with more severe retrospectively self-reported CA experiences self-reported lower friendship quality, $\beta$ = -0.19, SE = .01, $t_{97}$ = -1.89, $p$ = .062 (Figure 4A). Next, we observed that models specifying either deprivation or threat experiences as a predictor showed a better model fit compared to a model specifying cumulative CA (see supplementary information for full details). However, none of these dimensional models significantly predicted differences in friendship quality (deprivation experiences: $\beta$ = -0.16, SE = .01, $t_{97}$ = -1.57, $p$ = .120; threat experiences: $\beta$ = -0.17, SE = .01, $t_{97}$ = -1.65, $p$ = .103). Second, we found that greater subjectively perceived friendship quality was significantly related to better psychosocial functioning in young people with CA, $\beta$ = 0.44, SE = .02, $t_{97}$ = 4.87, $p$ < .001, $f^2_p$ = .245, $R^2_{adj}$ = .207 (Figure 4B). Finally, we observed no significant associations between CA (including cumulative index, threat, or deprivation experiences) and psychosocial functioning, $p$'s > .235. Please see our supplementary information for the full model output, descriptive statistics, and correlations between the study variables (sections G-H).

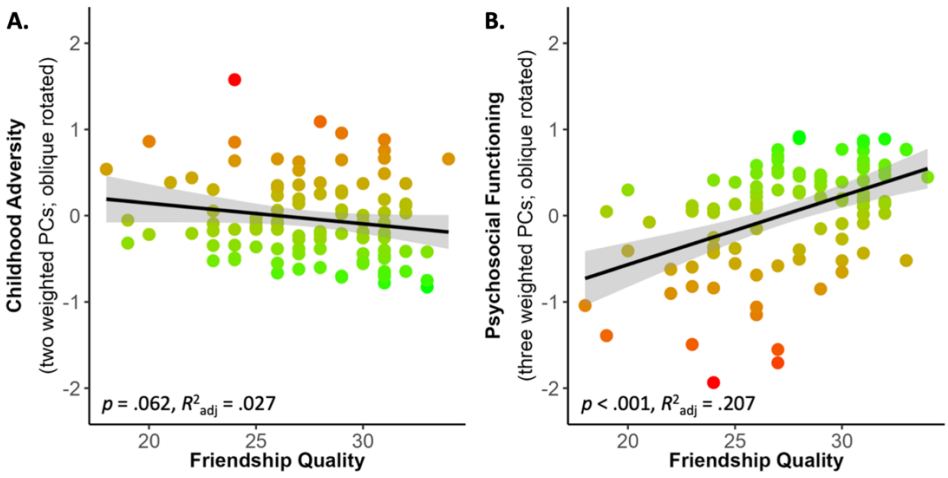

**Figure 4.** Associations between friendship quality and (A) childhood adversity and (B) psychosocial functioning**.** Participants with greater subjectively perceived friendship quality **(A)** did not retrospectively self-reported less negative CA ($p$ = .062) but **(B)** showed better psychosocial functioning ($p$ < .001). Index scores of CA comprise two weighted principal components (PCs) and index scores of psychosocial functioning comprise three weighted PCs, both oblique rotated. Both y-axes represent factor scores with $M$ = 0 and $SD$ = 1. Brighter shading (green) of individual data points represents (A) less severe CA and (B) better psychosocial functioning on each graph respectively. The black lines show the best-fitting linear regression lines, and the shaded regions around them represent the 95% confidence intervals.

***Neuroimaging Results (T2; n = 60)***

*State Anxiety Before and After Acute Psychosocial Stress*

We observed a significant increase in self-reported state anxiety upon completion of the MIST ($M_{before}$ = 29.19, $SD$ = 5.75; $M_{after}$ = 34.88, $SD$ = 11.57), $t_{57}$ = -4.33, $p$ < .001, $d$ = .568, suggesting that our task successfully induced subjective emotional stress (Figure 5).

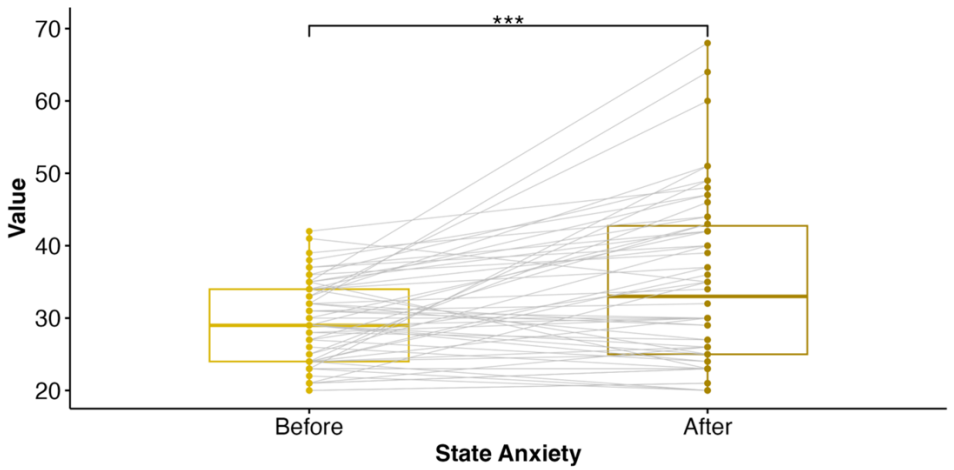


**Figure 5.** State anxiety increased after acute psychosocial stress. Participants exhibited greater self-reported state anxiety after completing the Montreal Imaging Stress Task (MIST) compared to state anxiety levels before the MIST ($p$ < .001). The boxplot displays the median (*Mdn*, solid vertical line) and interquartile range (IQR) before (*Mdn* = 29.00, IQR = 10) and after (*Mdn* = 33.00, IQR = 17.75) completing the MIST. The points represent individual datapoints with the grey lines connecting paired observations. *** $p$ < .001.

*Brain Responses to Acute Psychosocial Stress*

First, we investigated the main effect of stress (> control) in our predefined ROIs (hippocampus, amygdala, insula, mPFC (ACC), NAc, and thalamus) using familywise error (FWE) correction ($p_{FWE} < .05$) at the voxel level. This analysis identified significant activation in the left hippocampus ($t_{57} = 4.24$, $p_{FWE} = .007$; MNI coordinates: -27, -37, -4), bilateral insula ($t_{57} = 4.07$, $p_{FWE} = .020$; MNI coordinates: -42, 14, 5), left mPFC (ACC) ($t_{57} = 4.35$, $p_{FWE} = .043$; MNI coordinates: -3, 16, 38), right NAc ($t_{57} = 3.09$, $p_{FWE} = .016$; MNI coordinates: 15, 11, -10), and bilateral thalamus ($t_{57} = 5.22$, $p_{FWE} < .001$; MNI coordinates: 12, -10, 14). All significant task-related ROI clusters are summarized in Table 1 and visualized in Figure 6 below. Whole-brain analyses ($p_{FWE} < .05$) revealed no significant activation outside our predefined ROIs.

| | | MNI Coordinates | | | Cluster | | | $p_{FWE}$ |
|---|---|---|---|---|---|---|---|---|
| **Region** | **Side** | **x** | **y** | **z** | **Size** | ***t*** | ***z*** | **(Peak)** |
| Hippocampus | L | -27 | -37 | -4 | 8 | 4.24 | 3.93 | .007 |
| Insula | L | -42 | 14 | 5 | 2 | 4.07 | 3.80 | .020 |
| | L | -30 | 5 | 14 | 4 | 3.98 | 3.73 | .026 |
| | R | 45 | 11 | 2 | 1 | 3.97 | 3.71 | .027 |
| | L | -45 | -1 | 5 | 4 | 3.88 | 3.64 | .035 |
| | L | -33 | -16 | 8 | 1 | 3.76 | 3.54 | .048 |
| mPFC (ACC) | L | -3 | -16 | 38 | 1 | 4.35 | 4.03 | .043 |
| NAc | R | 15 | 11 | -10 | 4 | 3.09 | 2.96 | .016 |
| Thalamus | R | 12 | -10 | 14 | 195 | 5.22 | 4.70 | <.001 |
| | L | -9 | -19 | 14 | | 4.80 | 4.38 | .001 |
| | L | -18 | -22 | 14 | | 4.61 | 4.23 | .002 |

**Table 1.** ROIs activated during stress (> control) trials of the Montreal Imaging Stress Task. All reported statistics are significant at $p_{FWE} < .05$, voxel-level corrected for the ROI. All ROIs were bilaterally defined using the WFU PickAtlas Tool (version 3.0.5; Maldjian et al. (2003)) and based on the Automated Anatomical Labeling (AAL) atlas (Tzourio-Mazoyer et al., 2002). Given that the anatomical region of the mPFC is less well defined the ROI mask was based on the anatomical location of both dorsal and ventral mPFC (including the ACC; Brodmann areas (BA): 9, 10, 11, 24, 25, 32) (Moreno-López et al., 2020; Passingham & Wise, 2012; van Harmelen et al., 2013). ACC = anterior cingulate cortex; mPFC = medial prefrontal cortex; NAc = nucleus accumbens. L = left; R = right.

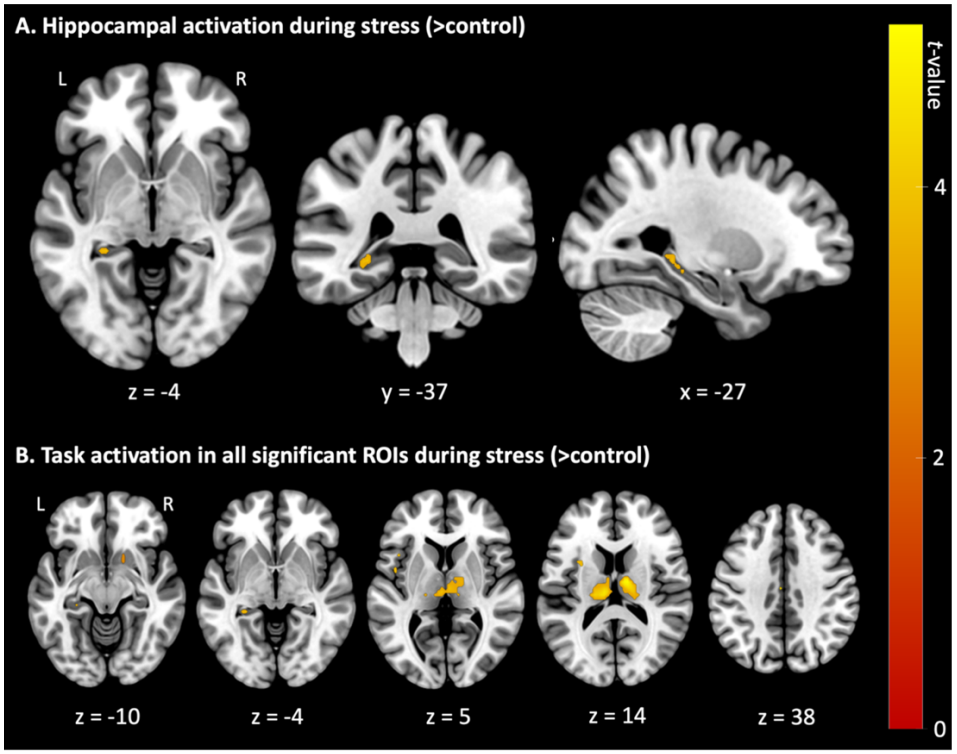


**Figure 6.** Overview of neural activation during acute psychosocial stress (> control). Displayed are *t*-values of neural activation ($p_{FWE} < .05$) during stress (> control) trials of the Montreal Imaging Stress Task. Results are presented **(A)** centered at the left hippocampus region of interest (MNI coordinates: x = -27, y = -37, z = -4) and **(B)** as axial slices with corresponding z-coordinates. L = left; R = right.

*Moderation Effect of Friendship Quality*

To examine whether perceived friendship quality was related to lower neural stress responses, we ran three separate linear regression models for each of our five predefined ROIs. Specifically, we examined the interaction between friendship quality and cumulative CA, deprivation, or threat experiences. These analyses revealed only a significant threat experiences x friendship quality interaction on left hippocampal reactivity, $\beta$ = -0.33, SE = .26, $t_{46}$ = -2.26, $p$ = .029, $f^2_p$ = .111, $R^2_{adj}$ = .142 (Figure 7). However, this effect did not survive correction for multiple comparisons ($p_{Bonf}$ = .145; corrected for five ROI comparisons). Age had a significant effect on left hippocampal reactivity across all analyses, with older participants showing increased left hippocampal reactivity. No other main effects or interactions were observed in any of our analyses ($p$'s > .050).

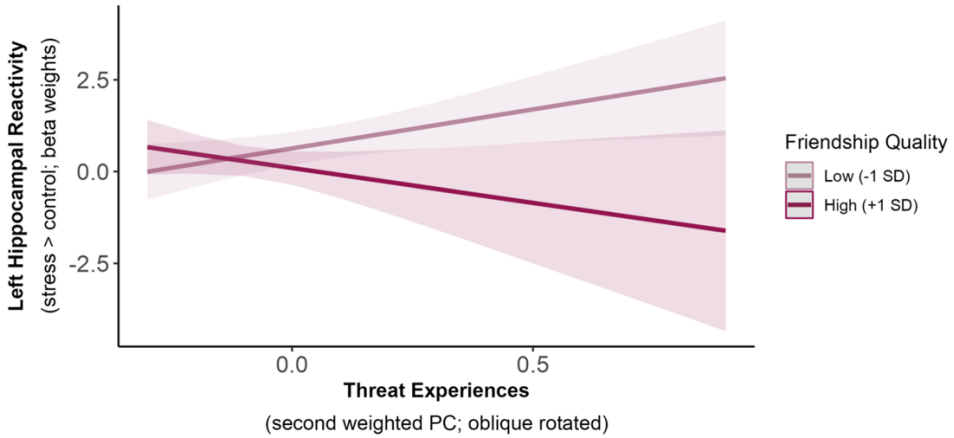


**Figure 7.** Exploratory average marginal effects in the interaction of threat experiences and friendship quality on left hippocampal reactivity to acute stress. Friendship quality had a weak moderating effect on the relationship between threat experiences and left hippocampal reactivity to acute stress ($p$ = .029), such that hippocampal reactivity increased with more negative threat experiences in participants reporting low friendship quality. However, this effect did not survive correction for multiple comparisons ($p_{Bonf}$ = .145; corrected for five ROI comparisons). The lines show the estimated marginal means of threat experiences (x-axis; second weighted PC) on left hippocampal reactivity (y-axis; beta weights) at different values of friendship quality (-1*SD* = 24.31; +1*SD* = 31.05) with a pointwise 95% confidence interval, derived from a multiple linear regression model.

## Discussion

In this study, we examined whether perceived friendship quality was related to better mental health and well-being ($N$ = 102) and lower neural stress responses using the Montreal Imaging Stress Task ($n$ = 62) in young people (aged 16-26) with low to moderate CA. In addition, we examined the relation between CA and friendship quality. A principal component analysis revealed two dimensions of CA resembling threat or deprivation like experiences. Hence, we investigated both cumulative and dimension specific effects of CA (Evans et al., 2013; Sheridan & McLaughlin, 2014). Contrary to the social transactional model of mental health vulnerability (McCrory et al., 2022), we found no support for social thinning after CA, meaning that the severity of CA (neither cumulative nor dimension specific) did not differentially impact friendship quality. Higher friendship quality, on the other hand, was strongly associated with better psychosocial functioning. Furthermore, we found that experimentally induced acute stress increased state anxiety and enhanced neural activity in five frontolimbic regions (left hippocampus, bilateral insula, left mPFC (ACC), right NAc, and bilateral thalamus). Finally, we found weak support that threat experiences interacted with

friendship quality to predict left hippocampal reactivity to acute stress. However, this effect did not survive correction for multiple comparisons. Therefore, future research is needed to examine whether friendships aid neural responses to acute stress in young people with childhood threat experiences.

Despite the prominence of research suggesting that CA can lead to impoverished social networks (Horan & Widom, 2015; Nevard et al., 2021; Sperry & Widom, 2013), we observed that neither conceptualization of CA (i.e., cumulative, deprivation, or threat) was associated with lower friendship quality. Our findings are in fact aligned with previous longitudinal studies by A. B. Miller et al. (2014) and van Harmelen et al. (2016) who showed that CA was not directly associated with poor friendships in healthy community samples. In addition, Fritz, Stretton, et al. (2020) even showed that CA predicted higher friendship quality at ages 14 and 18. The absence of social thinning in our sample may suggest that the risk of developing impoverished social networks is low for rather well-functioning young people with low to moderate CA. This assumption is further supported by our finding that neither conceptualization of CA was related to subsequent psychosocial functioning. Furthermore, we showed that higher friendship quality was strongly associated with better psychosocial functioning. This is in line with previous research showing that social support provided by friends, family, or significant others is related to better mental health and well-being in samples with CA (Jaffee, 2017; Lagdon et al., 2021; Salazar et al., 2011; van Harmelen et al., 2016, 2021; Vranceanu et al., 2007).

Next, we found that high-quality friendships aided left hippocampal reactivity to acute stress in young people with childhood threat experiences. While this interaction effect did not survive stringent correction for multiple comparisons, we recognize the value in cautiously aligning our uncorrected findings with previous research. For example, recent work by Tang et al. (2021) showed that low, but not high, levels of friendship quality facilitated blunted sympathetic nervous system reactivity to social rejection feedback, linking early institutionalization (i.e., severe deprivation experiences) with later-life peer problems. In contrast, Fritz, Stretton, et al. (2020) utilized a cumulative-risk approach to quantify CA and found that friendship support at ages 14 or 17 was not associated with neural responses to social rejection at age 18. Consequently, our results align with previous findings that have shown friendship stress buffering through dimensional, but not cumulative, approaches, despite some divergence regarding the specific dimensions investigated. However, it is worth noting that Tang et al. (2021) did not formally examine different dimensions of early experiences in their sample.

Given the established association between past threat experiences and hippocampal neurodevelopment, our uncorrected findings regarding friendship

stress buffering on hippocampal functioning are particularly interesting. The hippocampus is a subcortical region which develops mainly in the first two years of life, and is vital for learning, memory, spatial navigation, and emotional processing (Bird & Burgess, 2008; Phelps, 2004). The hippocampus also plays an important role in inhibiting HPA axis activity in response to elevated blood glucocorticoid levels (Lupien et al., 2009). Due to its dense innervation with glucocorticoid receptors, the hippocampus is particularly sensitive to chronic or repeated stress exposure. Both animal and human studies have shown that early onset and increased severity of CA, specifically threat exposure, was associated with structural and functional alterations in the hippocampus, which in turn was identified as a risk factor for later-life psychopathology (Y. Chen et al., 2008; Cohodes et al., 2021). For example, reductions in hippocampal volume were consistently observed in children and adolescents with past threat experiences, which partially mediated the relationship between threat exposure and internalizing (Weissman et al., 2020) and externalizing problems (Hanson, Nacewicz, et al., 2015), whilst also being associated with reduced friendship support (Malhi et al., 2020). Furthermore, hippocampal hyperreactivity to acute stress has been reported in young adults with cumulative CA (Seo et al., 2014) and middle-aged adults with emotional maltreatment (Leicht-Deobald et al., 2018). Interestingly, such hippocampal hyperreactivity to acute stress was associated with greater adverse health symptoms (Seo et al., 2014). In other words, reductions in hippocampal volume and functioning may act as a potential mechanism of stress vulnerability in young people with CA. In line with this claim, CA has been linked with a greater sensitivity towards peer rejection (van Harmelen et al., 2014) and a greater likelihood of experiencing interpersonal stress (Benedini et al., 2016; Handley et al., 2019; van Harmelen et al., 2016; Widom et al., 2014). Through this process, CA experiences are thought to reduce an individual's likelihood to form and maintain long-lasting, high-quality relationships (Labella et al., 2018; McLafferty et al., 2018). Again, an effect we did not observe in the current study, despite other studies reporting impoverished social networks in individuals with CA (Horan & Widom, 2015; Nevard et al., 2021; Sperry & Widom, 2013). Regardless, our findings, as well as those from other studies, consistently demonstrate a strong association between high-quality friendships and better mental health outcomes in young people with CA.

Our findings are considered in the context of important limitations. First, the current study design prohibits causal inferences. Future large-scale, longitudinal, prospective, and genetically sensitive studies are needed to draw conclusions about the causal impact of CA on neurocognitive and social functioning and the relationship to mental health vulnerability (Danese & Lewis, 2022; McCrory et al., 2022). Second, the data collection period of this study was cut short due to re-allocation of the clinical research facilities in Cambridge, UK during the COVID-19 pandemic, resulting in a small neuroimaging sample ($n$ = 62). Although a

retrospective power analysis confirmed that the sample size was sufficient to detect large effects, future research is needed to validate and extend our findings. Despite previous research indicating that stress can induce laterality changes in the hippocampus (Riem et al., 2015), we refrained from interpreting our laterality findings as these might be driven by our stringent significance threshold and reduced sample size. Third, the value of dimensional approaches for conceptualizing CA and identifying mechanisms shaping developmental outcomes is actively being debated (McLaughlin et al., 2021; Pollak & Smith, 2021). The current study suggests that continuously assessing the severity of different CA dimensions may be helpful for specifying putative neural mechanisms that potentially increase mental health vulnerability (McLaughlin, Weissman, et al., 2019; Puetz et al., 2020). In addition, future work should consider the developmental timing and chronicity of exposure to holistically understand the detrimental impact CA can have on the developing brain and consequently on neurocognitive and social functioning. Similarly, future work should account for differential friendship dimensions, such as intimacy, loyalty, frequency of engagement, or network size, as well as differences in stress paradigms with regards to type, intensity, and duration of acute stress, to gain a more nuanced mechanistic understanding about friendship stress buffering after CA. It is worth noting that our sample self-reported on average high levels of friendship quality suggesting a well-functioning group of young people. Nevertheless, current individual characteristics, such as mental health vulnerabilities, may have biased the reporting of friendship quality, in that relationships may be perceived as more negative (Baldwin & Degli Esposti, 2021; Colman et al., 2016). However, this concern seems negligible given that we successfully replicated previous longitudinal findings showing a strong link between high-quality friendships and better mental health in samples with low to moderate CA (van Harmelen et al., 2016, 2017, 2021). Furthermore, in our sample, we found no association between CA and psychosocial functioning, which is at odds with robust associations reported in the literature (Humphreys et al., 2016; McCrory et al., 2019; Shackman & Pollak, 2014; Sheikh et al., 2016). It is plausible that the remote assessment of CA and psychosocial functioning in our study may have introduced some limitations to the validity of these measures in our particular sample. However, remote psychosocial functioning assessments have demonstrated adequate psychometric properties (van Ballegooijen et al., 2016) and strong internal consistency (Brock et al., 2012) in previous studies. Furthermore, our remote assessment of the MFQ at T1 exhibited a moderate correlation with the MFQ assessment we conducted in the laboratory at T2 ($r = .69$, $p < .001$). Additionally, all our questionnaires demonstrated acceptable to excellent internal consistency at baseline, and we successfully replicated the above mentioned large-scale longitudinal findings (van Harmelen et al., 2017, 2021). Given these considerations, it is possible that the absence of a relationship between CA and psychosocial functioning in our sample can be attributed to the

fact that our sample consisted of relatively well-functioning young people who reported only low to moderate CA.

In conclusion, we showed that young people with more severe CA did not self-report lower friendship quality. However, higher friendship quality was strongly associated with better psychosocial functioning. We found only weak support that threat experiences interacted with friendship quality to predict left hippocampal reactivity to acute stress. However, this effect did not survive correction for multiple comparisons and therefore requires replication in larger ideally longitudinal samples. Hence, future research is needed to examine whether friendships aid neural responses to acute stress in young people with childhood threat experiences.

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**Data Availability Statement**
For all significant ROIs, group-level statistical maps displaying *t*-values of neural activation ($p_{FWE} < .05$) during stress (> control) trials of the MIST have been uploaded to NeuroVault (https://neurovault.org/collections/AXXZBAGI/).

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**Declaration of Competing Interests**
The authors have no competing interests to declare that are relevant to the content of this article. KI receives a stipend for editorial work from Elsevier.

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| Characteristics | Baseline Assessment (T1) | Attrition | In-Unit Assessment (T2) | Dropout Analysis | |
|---|---|---|---|---|---|
| | **Baseline Sample** ($N$ = 102) | **Dropouts** ($n$ = 42) | **Neuroimaging Sample** ($n$ = 60) | ***t*** | ***p*** |
| **Age** | 22.24 (2.76) | 21.76 (2.84) | 22.57 (2.68) | 1.46 | .148 |
| **Gender identity** | | | | -0.76 | .448 |
| Male | 36 (35.3) | 13 (31.0) | 23 (38.3) | | |
| Female | 66 (64.7) | 29 (69.0) | 37 (61.7) | | |
| **Race** | | | | -1.80 | .075 |
| Asian | 18 (17.7) | 11 (26.1) | 7 (11.7) | | |
| Black | 4 (3.9) | 2 (4.8) | 2 (3.3) | | |
| White | 69 (67.6) | 25 (59.5) | 44 (73.3) | | |
| Others | 11 (10.8) | 4 (9.6) | 7 (11.7) | | |
| **Highest education** | | | | .075 | .127 |
| GCSEs | 16 (15.8) | 9 (21.4) | 7 (11.7) | | |
| A-Levels | 29 (28.4) | 13 (31.0) | 16 (26.7) | | |
| Undergraduate degree | 39 (38.2) | 14 (33.3) | 25 (41.6) | | |

| | | | | | |
|---|---|---|---|---|---|
| Postgraduate degree | 18 (17.6) | 6 (14.3) | 12 (20.0) | | |
| **Housing** | | | | -0.04 | .967 |
| Parent(s) | 30 (29.4) | 12 (28.6) | 18 (30.0) | | |
| University housing | 33 (32.4) | 15 (35.6) | 18 (30.0) | | |
| Rented room | 8 (7.8) | 2 (4.8) | 6 (10.0) | | |
| Rented house or flat | 27 (26.5) | 13 (31.0) | 14 (23.3) | | |
| Owned a house or flat | 4 (3.9) | 0 (0) | 4 (6.7) | | |
| **Employment** | | | | 0.65 | .517 |
| Full-time | 26 (48.1) | 9 (45.0) | 17 (50.0) | | |
| Part-time | 20 (37.0) | 9 (45.0) | 11 (32.4) | | |
| Self-employed | 8 (14.8) | 2 (10.0) | 6 (17.6) | | |
| **Friendship quality** | 27.47 (3.55) | 27.17 (3.82) | 27.68 (3.37) | 0.72 | .473 |
| **Childhood adversity** | | | | | |
| Sexual abuse | 9.60 (22.10) | 13.25 (28.36) | 7.12 (16.41) | -1.36 | .178 |
| Emotional abuse | 30.15 (24.12) | 33.88 (26.13) | 27.67 (22.56) | -1.27 | .209 |
| Physical abuse | 7.82 (14.41) | 9.39 (15.62) | 6.75 (13.56) | -0.90 | .369 |

| | | | | | |
|---|---|---|---|---|---|
| Emotional neglect | 36.80 (25.75) | 34.25 (22.52 | 38.50 (27.74) | 0.81 | .422 |
| Physical neglect | 13.45 (15.24) | 14.15 (16.65 | 12.97 (14.30) | -0.38 | .705 |
| Negative parenting | 40.68 (23.31) | 42.89 (25.11 | 39.17 (22.09) | -0.79 | .434 |
| Poor parental involvement | 52.50 (24.55) | 53.33 (24.08) | 51.94 (25.04) | -0.28 | .783 |
| Corporal punishment | 28.28 (25.86) | 30.34 (26.73 | 26.94 (25.41 | -0.64 | .526 |
| Paternal abuse | 15.71 (23.86) | 17.30 (26.38) | 14.52 (21.95) | -0.57 | .571 |
| Maternal abuse | 14.13 (21.13) | 16.98 (25.00) | 12.09 (17.83) | -1.15 | .253 |
| Paternal overcontrol | 24.57 (25.39) | 25.79 (26.98) | 23.66 (24.35) | -0.41 | .683 |
| Maternal overcontrol | 32.35 (27.03) | 34.13 (27.04) | 31.11 (27.18) | -0.55 | .582 |
| Paternal indifference | 20.20 (26.33) | 21.56 (29.17 | 19.14 (24.12) | -0.45 | .657 |
| Maternal indifference | 13.29 (20.58) | 14.55 (21.39) | 13.41 (20.13) | -0.52 | .607 |

**Table A.1.1.** Characteristics of the baseline and neuroimaging sample (including dropout analysis). Age (in years), friendship quality, and severity of CAs (in percent) are reported as *M* (*SD*). All other characteristics are reported as n (%). A dropout analysis was performed using two sample t-tests to compare characteristics between the neuroimaging sample and the dropouts.

| Childhood Adversity | Baseline Assessment (T1) | Attrition | In-Unit Assessment (T2) |
|---|---|---|---|
| | **Baseline Sample** ($N$ = 102) | **Dropouts** ($n$ = 42) | **Neuroimaging Sample** ($n$ = 60) |
| **Deprivation dimension** | | | |
| Maternal indifference | 8.39 (3.70) | 8.62 (3.85) | 8.23 (3.62) |
| Maternal abuse | 7.12 (3.17) | 7.55 (3.75) | 6.81 (2.67) |
| Emotional neglect | 12.36 (5.15) | 11.85 (4.50) | 12.70 (5.55) |
| Physical neglect | 7.69 (3.05) | 7.83 (3.33) | 7.59 (2.86) |
| Poor parental involvement | 9.30 (2.95) | 9.40 (2.89) | 9.23 (3.00) |
| Negative parenting | 7.88 (2.80) | 8.15 (3.01) | 7.70 (2.65) |
| Emotional abuse | 11.03 (4.82) | 11.78 (5.23) | 10.53 (4.51) |
| Maternal overcontrol | 7.88 (3.24) | 8.10 (3.24) | 7.73 (3.26) |

| | | | |
|---|---|---|---|
| **Threat dimension** | | | |
| Paternal abuse | 7.36 (3.58) | 7.60 (3.96) | 7.18 (3.29) |
| Physical abuse | 6.56 (2.88) | 6.88 (3.12) | 6.35 (2.71) |
| Paternal overcontrol | 6.95 (3.05) | 7.10 (3.24) | 6.84 (2.92) |
| Corporal punishment | 6.39 (3.10) | 6.64 (3.21) | 6.23 (3.05) |
| Paternal indifference | 9.64 (4.74) | 9.88 (5.25) | 9.44 (4.34) |

**Table A.1.2.** Severity ratings of childhood adversity measures. Raw severity ratings of CA measures are reported as *M* (*SD*). Based on the principal component analyses for CA, each measure is sorted into a deprivation or threat dimension and ranked according to their factor loading. Based on established cut-off scores for the CTQ (Bernstein et al., 1994), the baseline sample can be characterized reporting low to moderate levels of CA. Specifically, emotional abuse severity ratings between 9-12 can be categorized as low to moderate trauma exposure. Physical abuse ratings between 5-7 can be categorized as none or minimal trauma exposure. Emotional neglect ratings between 10-14 can be categorized as low to moderate trauma exposure. And finally, physical neglect ratings between 5-7 can be categorized as none or minimal trauma exposure. Overall, CTQ scale severity ratings can be categorized into four groups representing none or minimal trauma exposure, low to moderate trauma exposure, moderate to severe trauma exposure, and severe to extreme trauma exposure.

**B. Baseline Assessments (T1)**

***B.1 Childhood Adversity***

During the baseline assessment (T1), participants completed three retrospective self-report questionnaires aimed at assessing different types of CA. Across all questionnaires, positive items were reverse coded so that higher scores reflect more severe experiences of CA.

*B.1.1 Short-Form of the Childhood Trauma Questionnaire (CTQ-SF)*

The CTQ-SF (Bernstein et al., 2003) is a 28-item screening measure for maltreatment experiences within the family environment during childhood or adolescence (up until age 18). On a 5-point Likert scale (1 = never true, 5 = very often true) participants responded to items such as "I didn't have enough to eat". The CTQ-SF comprises of five subscales (sexual, physical, and emotional abuse and physical and emotional neglect), which can be combined to estimate the total severity of childhood maltreatment experiences. In this sample, internal consistency was excellent for the total scale (Cronbach's α = .92) and acceptable to excellent for the five subscales (sexual abuse: α = .94; physical abuse: α = .81; emotional abuse: α = .85; physical neglect: α = .72; emotional neglect: α = .93). In our analyses, we utilized the four CTQ-SF subscales: physical abuse, emotional abuse, physical neglect, and emotional neglect. The sexual abuse subscale was excluded due to low prevalence (*Mdn* = 0, IQR = 0).

*B.1.2 Measure of Parental Style Questionnaire (MOPS)*

The MOPS (Parker et al., 1997) is a 30-item screening measure for perceived maternal and paternal parenting style experiences respectively. On a 4-point Likert scale (1 = not true at all, 4 = extremely true) participants responded to items such as "My father was physically violent or abusive to me". The MOPS comprises of six subscales (maternal and paternal abuse, -indifference, and -overcontrol), which can be combined to estimate the total severity of adverse maternal and paternal parenting style experiences. In this sample, internal consistency was excellent for the total maternal scale (α = .91) and paternal scale (α = .90) and acceptable to good for the six subscales (maternal abuse: α = .86, -indifference: α = .88; -overcontrol: α = .78; paternal abuse: α = .77; -indifference: α = .90; -overcontrol: α = .89). In our analyses, we utilized all six MOPS subscales: maternal and paternal abuse, -indifference, and -overcontrol.

*B.1.3 Alabama Parenting Questionnaire (APQ)*

The APQ (Frick, 1991) is a 42-item screening measure for past parenting experiences. On a 5-point Likert scale (1 = never true, 5 = very often true) participants responded to items such as "Your parents spank you with their hand when you have done something wrong". The APQ comprises of five subscales (corporal punishment, parental involvement, negative parenting, poor monitoring/supervision, and inconsistent discipline), which can be combined to

estimate the total severity of negative parenting experiences. For the current study, a modified 15-item version of the APQ was administered retaining all five subscales (guided by Elgar et al. (2007)). In this sample, internal consistency was poor for two subscales (poor monitoring/supervision: α = .51; inconsistent discipline: α = .57) which led us to exclude these scales from all analyses. Internal consistency was acceptable to good for the remaining three subscales (corporal punishment: α = .86; parental involvement: α = .77; negative parenting: α = .83) and good for the 9-item total scale (α = .85). Hence, in our analyses, we utilized the three APQ subscales: corporal punishment, parental involvement, and negative parenting.

### *B.2 Psychosocial Functioning*

During the baseline assessment (T1), participants also completed eight self-report questionnaires aimed at assessing psychosocial functioning over the past two to four weeks. Across all questionnaires, negative items were reverse coded so that higher scores reflect more healthy psychosocial functioning and reduced symptom frequency.

#### *B.2.1 Mood and Feelings Questionnaire (MFQ)*

The MFQ (Angold & Costello, 1987) is a 33-item screening measure for current depressive symptoms. On a 4-point Likert scale (1 = never, 4 = always) participants responded to items such as "I felt miserable or unhappy". In this sample, internal consistency was excellent for the total scale (α = .94), which was utilized in our analyses.

#### *B.2.2 Revised Children's Manifest Anxiety Scale (RCMAS)*

The RCMAS (Reynolds & Richmond, 1978) is a 28-item screening measure for current anxiety symptoms. On a 4-point Likert scale (1 = never, 4 = always) participants responded to items such as "I worried a lot of the time". The RCMAS comprises of three subscales (physiological anxiety, worry/oversensitivity, social concerns/concentration), which can be combined to estimate the total severity of anxiety symptoms. In this sample, internal consistency was excellent for the total scale (α = .94) and good for the three subscales (physiological anxiety: α = .80; worry/oversensitivity: α = .89; social concerns/concentration: α = .84). In our analyses, we utilized all three RCMAS subscales: physiological anxiety, worry/oversensitivity, and social concerns/concentration.

#### *B.2.3 Leyton Obsessional Inventory-Child Version (LOI-CV)*

The LOI-CV (Bamber et al., 2002) is a 20-item screening measure for current obsessive-compulsive symptoms. On a 4-point Likert scale (1 = never, 4 = always) participants responded to items such as "I worried about being clean enough". The LOI-CV comprises of three subscales (compulsions, obsessions, cleanliness), which can be combined to estimate the total severity of obsessive-compulsive

symptoms. In this sample, internal consistency was good for the total scale (α = .87) and acceptable to good for the three subscales (compulsions: α = .85; obsessions: α = .78; cleanliness: α = .83). In our analyses, we utilized all three LOI-CV subscales: compulsions, obsessions, and cleanliness.

*B.2.4 Behavioral Checklist (BCL)*
The BCL (van Harmelen et al., 2017) is an 11-item screening measure for current antisocial behavior symptoms. On a 4-point Likert scale (1 = never, 4 = always) participants responded to items such as "I stole things". In this sample, internal consistency was acceptable for the total scale (α = .72), which was utilized in our analyses.

*B.2.5 Rosenberg Self-Esteem Scale (SES)*
The SES (Rosenberg, 1965) is a 10-item screening measure for current self-esteem. On a 4-point Likert scale (1 = never, 4 = always) participants responded to items such as "At times, I thought I was no good at all". In this sample, internal consistency was acceptable for the total scale (α = .78), which was utilized in our analyses.

*B.2.6 Kessler Psychological Distress Scale (K10)*
The K10 (Kessler et al., 2002) is a 10-item screening measure for current psychological distress symptoms. On a 5-point Likert scale (1 = none of the time, 5 = all of the time) participants responded to items such as "How often did you feel nervous?". In this sample, internal consistency was excellent for the total scale (α = .91), which was utilized in our analyses.

*B.2.7 Warwick-Edinburgh Mental Well-Being Scale (WEMWBS)*
The WEMWBS (Tennant et al., 2007) is a 14-item screening measure for current mental well-being. On a 5-point Likert scale (1 = none of the time, 5 = all of the time) participants responded to items such as "I've been feeling optimistic about the future". In this sample, internal consistency was excellent for the total score (α = .93), which was utilized in our analyses.

*B.2.8 Drugs and Self Injury Questionnaire (DASI)*
The DASI (Wilkinson et al., 2018) is a 10-item screening measure for current risk-taking behavior related to smoking, alcohol, and drug use as well as non-suicidal self-injury (NSSI). On a 4-point Likert scale (1 = never, 4 = every day or nearly every day) participants responded to items such as "How often did you smoke a cigarette/s?". Due to weak correlations with the total score ($r$ < .30) both items assessing NSSI were excluded. In this sample, internal consistency was acceptable for the 8-item total scale (α = .71), which was utilized in our analyses.

### *B.3 Friendship Quality*

The Cambridge Friendship Questionnaire (CFQ; van Harmelen et al. (2017)) is an 8-item screening measure to assess the self-reported number, availability, and quality of friendships. During the baseline assessment (T1), participants responded to items such as "Do you feel that your friends understand you?". Higher scores reflect greater perceived friendship quality. An exploratory factor analysis was conducted on the 8-items of the CFQ revealing low factor loadings (< .40; Stevens (2001)) of item 6 ("Do people who aren't your friends laugh at you or tease you in a hurtful way?") which led to the exclusion of this item from all analyses. Please see below for a summary of the factor analysis (Table C.1). In this sample, internal consistency was acceptable for the 7-item total scale ($\alpha$ = .72). Across two different samples of young people with CA (van Harmelen et al., 2017, 2021), the CFQ has been successfully utilized to predict mental health functioning.

**C. Exploratory Factor Analysis on the Cambridge Friendship Questionnaire**

An exploratory factor analysis (FA) was conducted on the 8-items of the Cambridge Friendship Questionnaire (CFQ) with orthogonal rotation (varimax). The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO = . 80; "meritorious" according to Kaiser (1974)) and all KMO values for individual items were above the acceptable limit of .50. Bartlett's test of sphericity, $\chi^2_{28} = 269.77$, $p < .001$, indicated that correlations between items were sufficiently large for a FA. The scree plot and parallel analysis suggested retaining two factors. We performed a principal axes factor analysis using the psych R package (version 2.2.9; Revelle (2022)) with the maximum number of iterations for convergence set to 100. Due to rotated factor loadings of < .40 on both factors, item 6 ("Do people who aren't your friends laugh at you or tease you in a hurtful way?") was excluded from all analyses. Table C.1 below shows the factor loadings after rotation.

| | Item | FA1 | FA2 | *h2* | *u2* |
|---|---|---|---|---|---|
| 1. | Are you happy with the number of friends you've got at the moment? | **.77** | .01 | .59 | .41 |
| 2. | How often do you arrange to see friends other than at school, college or work? | **.70** | -.19 | .52 | .48 |
| 3. | Do you feel that your friends understand you? | **.74** | .28 | .62 | .38 |
| 4. | Can you confide in your friends? | **.70** | .14 | .51 | .49 |
| 5. | Do your friends ever laugh at you or tease you in a hurtful way? | -.03 | **.84** | .71 | .29 |
| 6. | Do people who aren't your friends laugh at you or tease you in a hurtful way? | .26 | .34 | .18 | .82 |
| 7. | Do you have arguments with your friends that upset you? | .01 | **.50** | .25 | .75 |
| 8. | Overall, how happy are you with your friendships? | **.80** | .10 | .65 | .35 |
| | Eigenvalues | 2.81 | 1.22 | | |
| | % of variance | 35.2 | 15.3 | | |

**Table C.1.** Summary of exploratory factor analysis results for the Cambridge Friendship Questionnaire ($N$ = 102). Factor loadings over .39 appear in bold. FA = varimax rotated factor loadings; h2 = communalities (proportion of common variance within a variable); u2 = uniqueness (proportion of unique variance for each variable).

## D. In-Unit Assessment (T2)

### *D.1 State Anxiety*

State anxiety was assessed with the State-Trait Anxiety Inventory (STAI; Spielberger & Vagg (1984)) before and after participants completed the MIST in the MRI scanner. As part of the STAI, participants responded to items such as "I feel nervous" on a 4-point Likert scale (1 = not at all, 4 = very much so). Positive items were reverse coded so that higher scores indicate greater state anxiety. In this sample, internal consistency ranged from good ($\alpha$ = .88) before scanning to excellent ($\alpha$ = .96) after scanning.

## E. Principal Component Analysis

### *E.1 Childhood Adversity*

Principal component analysis (PCA) was used to explore differential dimensions of CA experiences in our sample, which were subsequently combined into a cumulative CA index. Specifically, we computed weighted multi-modal composite scores for CA using a PCA with non-orthogonal (oblique) rotation on individual scores of the three APQ subscales (corporal punishment, parental involvement, and negative parenting), the four CTQ-SF subscales (physical abuse, emotional abuse, physical neglect, and emotional neglect), and the six MOPS subscales (maternal and paternal abuse, indifference, and overcontrol). Two of the APQ subscales (poor monitoring/supervision and inconsistent discipline) were excluded due to poor internal consistency ($\alpha$'s < .58) and the sexual abuse subscale of the CTQ-SF was excluded due to low prevalence of sexual abuse (*Mdn* = 0, IQR = 0). The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO = .85; "meritorious" according to Kaiser (1974)) and all KMO values for individual items were ≥ .70, which is well above the acceptable limit of .50. Bartlett's test of sphericity, $\chi^2_{78} = 722.86$, $p < .001$, indicated that correlations between items were sufficiently large for a PCA. The scree plot was slightly ambiguous and showed inflexions that would justify retaining both two and three components. Given the small sample size, and because the more parsimonious option is always preferred, we retained a two-component solution for the final analyses. The principal component (PC) scores and their associations are visualized in Figure 3A and further summarized in Table E.1. The figure shows that negative parenting (APQ), parental involvement (APQ), emotional abuse (CTQ-SF), emotional neglect (CTQ-SF), physical neglect (CTQ-SF), maternal indifference (MOPS), maternal overcontrol (MOPS), and maternal abuse (MOPS) all loaded onto PC1, which explained 37% of variance. Given that most items, except for maternal and emotional abuse, capture experiences involving an absence of expected inputs from the environment, we referred to PC1 as the *deprivation dimension* in all analyses. Furthermore, PC2 explained 21% of variance across the subscales: corporal punishment (APQ), physical abuse (CTQ-SF), paternal indifference (MOPS), paternal overcontrol (MOPS), and paternal abuse (MOPS). Given that most of these subscales capture experiences involving

harm or threat of harm, except for paternal indifference which only had a weak loading (.40), we referred to PC2 as the *threat dimension* in all analyses. To calculate a cumulative-risk score, we summed both dimensional scores. Specifically, to account for the contributions of both PCs, we weighted the scores for each PC by their explained variance and subsequently summed these scores to compute a single index of total severity experienced, which in all analyses we refer to as the *cumulative CA index*. A similar method has been employed by Anand et al. (2019).

| Items | PC1 | PC2 | *h2* | *u2* |
|---|---|---|---|---|
| Maternal indifference (MOPS) | **.87** | -.11 | .68 | .32 |
| Maternal abuse (MOPS) | **.83** | -.05 | .65 | .35 |
| Emotional neglect (CTQ-SF) | **.81** | .09 | .73 | .27 |
| Physical neglect (CTQ-SF) | **.79** | -.05 | .59 | .41 |
| Parental involvement (APQ) | **.72** | -.01 | .51 | .49 |
| Negative parenting (APQ) | **.70** | .05 | .53 | .47 |
| Emotional abuse (CTQ-SF) | **.64** | .30 | .68 | .32 |
| Maternal overcontrol (MOPS) | **.52** | .20 | .41 | .59 |
| Paternal abuse (MOPS) | -.11 | **.93** | .78 | .22 |
| Physical abuse (CTQ-SF) | .07 | **.72** | .56 | .44 |
| Paternal overcontrol (MOPS) | .08 | **.67** | .50 | .50 |
| Corporal punishment (APQ) | .27 | **.60** | .59 | .41 |
| Paternal indifference (MOPS) | .26 | **.40** | .32 | .68 |
| Eigenvalues | 4.82 | 2.73 | | |
| % of variance | 37.0 | 21.0 | | |

**Table E.1.** Summary of principal component analysis on childhood adversity measures ($N$ = 102). Factor loadings over .39 appear in bold. PC = oblique rotated principal component loadings; $h2$ = communalities (proportion of common variance within a variable); $u2$ = uniqueness (proportion of unique variance for each variable). APQ = Alabama Parenting Questionnaire; CTQ-SF = Short-Form of the Childhood Trauma Questionnaire; MOPS = Measure of Parental Style Questionnaire.

### *E.2 Psychosocial Functioning*

To capture the range of psychosocial outcomes, we computed weighted multi-modal composite scores for psychosocial functioning using a PCA with oblique rotation on individual total scores of the MFQ, the three RCMAS subscales (physiological anxiety, worry/oversensitivity, and social concerns/concentration), the three LOI-CV subscales (compulsions, obsessions, and cleanliness), the BCL, the SES, the K10, the WEMWBS, and the DASI. The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO = .91; "marvelous" according to Kaiser (1974)). All but one KMO values for

individual items were ≥ .78. Only the DASI had a KMO value of .44 ("unacceptable" according to Kaiser (1974)) which led to its exclusion from all analyses. Bartlett's test of sphericity, $\chi^2_{55} = 897.97$, $p < .001$, indicated that correlations between items were sufficiently large for a PCA. The scree plot showed inflexions that justified retaining three components. The PC scores and their associations are visualized in Figure 3B and further summarized in Table E.2. The figure shows that the total scores of the MFQ, SES, K10, and WEMWBS as well as the subscales: physiological anxiety (RCMAS), worry/oversensitivity (RCMAS), social concerns/concentration (RCMAS), and obsessions (LOI-CV) all loaded onto PC1, which explained 55% of variance. Furthermore, PC2 explained 15% of variance across the subscales: compulsions (LOI-CV), obsessions (LOI-CV), and cleanliness (LOI-CV) whereas BCL was the only scale loading onto PC3, which explained 10% of variance. To compute a *cumulative psychosocial functioning index*, we summed the weighted PC1, PC2, and PC3 scores.

| **Items** | **PC1** | **PC2** | **PC3** | ***h2*** | ***u2*** |
|---|---|---|---|---|---|
| Mental well-being (WEMWBS) | **.92** | -.13 | -.09 | .75 | .25 |
| Social concerns/concentration (RCMAS) | **.91** | -.07 | .06 | .80 | .20 |
| Depressive symptoms (MFQ) | **.90** | -.02 | .14 | .88 | .12 |
| Self-esteem (SES) | **.88** | -.01 | -.11 | .74 | .26 |
| Worry/oversensitivity (RCMAS) | **.86** | .07 | -.03 | .78 | .22 |
| Psychological distress (K10) | **.86** | .06 | .07 | .80 | .20 |
| Physiological anxiety (RMCAS) | **.75** | .14 | .29 | .84 | .16 |
| Obsessions (LOI-CV) | **.61** | **.44** | -.03 | .76 | .24 |
| Cleanliness (LOI-CV) | -.14 | **.90** | .20 | .81 | .20 |
| Compulsions (LOI-CV) | .32 | **.71** | -.32 | .77 | .23 |
| Behavioral problems (BCL) | .14 | .04 | **.91** | .92 | .08 |
| Eigenvalues | 6.00 | 1.69 | 1.15 | | |
| % of variance | 55.0 | 15.0 | 10.0 | | |

**Table E.2.** Summary of principal component analysis on psychosocial functioning measures ($N$ = 102). Factor loadings over .39 appear in bold. PC = oblique rotated principal component loadings; $h2$ = communalities (proportion of common variance within a variable); $u2$ = uniqueness (proportion of unique variance for each variable). MFQ = Mood and Feelings Questionnaire; RCMAS = Revised Children's Manifest Anxiety Scale; LOI-CV = Leyton Obsessional Inventory-Child Version; BCL = Behavioral Checklist; SES = Rosenberg Self-Esteem Scale; K10 = Kessler Psychological Distress Scale; WEMWBS = Warwick-Edinburgh Mental Well-Being Scale.

### F. Power Analysis

A power analysis was performed using G*Power (version 3.1.9.6; Faul et al. (2007)) to determine the minimum sample size required to examine associations

between CA (measured by weighted composite scores), friendship quality (measured by total CFQ scores), and stress-induced ROI reactivity. The following parameters were used to calculate the total sample size: effect size ($f^2$) = .40, α error probability = .05, power (1- β error probability) = .95, number of predictors = 5 (main predictors: CA, friendship support; covariates: age, gender, IQ). Results indicated that the required sample size to achieve 95% power for detecting a large effect was $N$ = 56 for linear regression analyses. Thus, our obtained neuroimaging sample of $n$ = 60 is adequate for the current research.

**G. Descriptive Statistics and Correlations between Study Variables**

| | Variable | *M* | *SD* | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|---|---|---|
| 1. | Age | 22.57 | 2.68 | - | | | | | |
| 2. | Gender | 1.62 | 0.49 | -.09 | - | | | | |
| 3. | Friendship quality | 27.68 | 3.37 | .03 | .07 | - | | | |
| 4. | Deprivation experiences | -0.04 | 0.31 | .06 | -.01 | -.22 | - | | |
| 5. | Threat experiences | -0.02 | 0.19 | .09 | .12 | -.21 | .51 | - | |
| 6. | Cumulative CA | -0.06 | 0.49 | .08 | .04 | -.25 | .94 | .77 | - |
| 7. | Psychosocial functioning | 0.03 | 0.60 | .09 | -.03 | .42 | .05 | .03 | .05 |

**Table G.1.** Descriptive statistics and correlations between study variables. $N$ = 102; Gender: 1 = male, 2 = female; CA = childhood adversity.

**H. Behavioral Results (T1; *N* = 102): Full Model Output**

| | *β* | SE | $t_{97}$ | *p* |
|---|---|---|---|---|
| Intercept | | 0.53 | 0.02 | .987 |
| Friendship quality | -0.19 | 0.01 | -1.89 | .062 |
| Age | 0.16 | 0.02 | 1.60 | .113 |
| Gender | 0.03 | 0.10 | 0.26 | .798 |

**Table H.1.** More severe cumulative childhood adversity was not associated with lower friendship quality. The cumulative CA index was derived through summing the weighted PC1 and PC2 scores. Age at assessment and gender identity were added as covariates. One outlier was excluded.

| | $\beta$ | SE | $t_{97}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.40 | -0.20 | .840 |
| Friendship quality | -0.16 | 0.01 | -1.57 | .120 |
| Age | 0.16 | 0.01 | 1.59 | .116 |
| Gender | 0.03 | 0.08 | 0.31 | .760 |

**Table H.2.** Deprivation experiences were not associated with friendship quality. The deprivation dimension was derived through the weighted PC1 scores. Age at assessment and gender identity were added as covariates. One outlier was excluded.

| | $\beta$ | SE | $t_{97}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.23 | 0.40 | .692 |
| Friendship quality | -0.17 | 0.01 | -1.65 | .103 |
| Age | 0.09 | 0.01 | 0.94 | .350 |
| Gender | 0.01 | 0.04 | 0.06 | .954 |

**Table H.3.** Threat experiences were not associated with friendship quality. The threat dimension was derived through the weighted PC2 scores. Age at assessment and gender identity were added as covariates. One outlier was excluded.

| | $\beta$ | SE | $t_{97}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.62 | -4.41 | <.001 |
| Friendship quality | **0.44** | **0.02** | **4.87** | **<.001** |
| Age | 0.08 | 0.02 | 0.87 | .386 |
| Gender | 0.13 | 0.12 | 1.48 | .141 |

**Table H.4.** Higher friendship quality was associated with improved psychosocial functioning. The cumulative psychosocial functioning index was derived through summing the weighted PC1, PC2, and PC3 scores. Age at assessment and gender identity were added as covariates. One outlier was excluded. $f^2_p$ = .245, $R^2_{adj}$ = .207.

| | $\beta$ | SE | $t_{96}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.43 | -1.61 | .111 |
| Psychosocial functioning | -0.09 | 0.08 | -0.86 | .393 |
| Age | 0.16 | 0.02 | 1.59 | .116 |
| Gender | 0.02 | 0.10 | 0.24 | .809 |

**Table H.5.** Cumulative childhood adversity was not associated with psychosocial functioning. The cumulative CA index was derived through summing the weighted PC1 and PC2 scores. The cumulative psychosocial functioning index was derived through summing the weighted PC1, PC2, and PC3 scores. Age at assessment and gender identity were added as covariates. Two outliers were excluded.

| | $\beta$ | SE | $t_{96}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.33 | -1.56 | .123 |
| Psychosocial functioning | -0.05 | 0.06 | -0.47 | .637 |
| Age | 0.16 | 0.01 | 1.53 | .130 |
| Gender | 0.02 | 0.08 | 0.24 | .808 |

**Table H.6.** Deprivation experiences were not associated with psychosocial functioning. The deprivation dimension was derived through the weighted PC1 scores. The cumulative psychosocial functioning index was derived through summing the weighted PC1, PC2, and PC3 scores. Age at assessment and gender identity were added as covariates. Two outliers were excluded.

| | $\beta$ | SE | $t_{96}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 0.18 | -1.04 | .301 |
| Psychosocial functioning | -0.12 | 0.03 | -1.19 | .236 |
| Age | 0.10 | 0.01 | 1.03 | .306 |
| Gender | 0.01 | 0.04 | 0.14 | .890 |

**Table H.7.** Threat experiences were not associated with psychosocial functioning. The threat dimension was derived through the weighted PC2 scores. The cumulative psychosocial functioning index was derived through summing the weighted PC1, PC2, and PC3 scores. Age at assessment and gender identity were added as covariates. Two outliers were excluded.

| | Dependent variable | Independent variable | Covariates |
|---|---|---|---|
| Model 1 | Friendship quality | Cumulative CA | Age, Gender |
| Model 2 | Friendship quality | Deprivation + Threat experiences | Age, Gender |
| Model 3 | Friendship quality | Deprivation experiences | Age, Gender |
| Model 4 | Friendship quality | Threat experiences | Age, Gender |

**Table H8.1.** Likelihood ratio tests. Models using different approaches to conceptualize CA whilst predicting friendship quality.

| | BIC | AIC |
|---|---|---|
| Model 1 | 560.15 | 547.07 |
| Model 2 | 564.58 | 548.89 |
| Model 3 | 561.25 | 548.18 |
| Model 4 | 561.00 | 547.92 |

**Table H8.2**

| | $\chi^2$ | $p$ |
|---|---|---|
| Model 1 vs. Model 2 | 0.18 | .671 |
| Model 1 vs. Model 3 | 1.10 | **<.001** |
| Model 1 vs. Model 4 | 0.86 | **<.001** |
| Model 3 vs. Model 4 | 0.25 | **<.001** |

**Table H8.3**

**I. Neuroimaging Results (T2; $n$ = 60): Full Model Output**

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.24 | 0.31 | .756 |
| Cumulative CA | 0.14 | 0.35 | 0.92 | .361 |
| Friendship quality | -0.11 | 0.06 | -0.72 | .474 |
| Age | 0.12 | 0.07 | 0.80 | .430 |
| Gender | -0.20 | 0.35 | -1.37 | .176 |
| IQ | -0.04 | 0.02 | -0.25 | .805 |
| Cumulative CA x Friendship quality | -0.15 | 0.10 | -1.04 | .306 |

**Table I.1.** Bilateral insula reactivity was not related to cumulative childhood adversity and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.88 | -0.67 | .509 |
| Cumulative CA | -0.08 | 0.45 | -0.51 | .611 |
| Friendship quality | -0.03 | 0.07 | -0.17 | .866 |
| Age | 0.26 | 0.09 | 1.71 | .095 |
| Gender | -0.09 | 0.46 | -0.65 | .519 |
| IQ | -0.01 | 0.02 | -0.07 | .947 |
| Cumulative CA x Friendship quality | -0.08 | 0.13 | -0.56 | .576 |

**Table I.2.** Left medial prefrontal cortex reactivity was not related to cumulative childhood adversity and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.54 | 1.19 | .241 |
| Cumulative CA | 0.15 | 0.39 | 0.99 | .327 |
| Friendship quality | 0.16 | 0.06 | 1.09 | .280 |
| Age | 0.07 | 0.08 | 0.46 | .648 |
| Gender | -0.13 | 0.40 | -0.92 | .364 |
| IQ | -0.18 | 0.02 | -1.16 | .252 |
| Cumulative CA x Friendship quality | -0.04 | 0.11 | -0.24 | .811 |

**Table I.3.** Right nucleus accumbens reactivity was not related to cumulative childhood adversity and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.20 | -0.80 | .428 |
| Cumulative CA | -0.04 | 0.34 | -0.28 | .778 |
| Friendship quality | -0.15 | 0.05 | -1.04 | .304 |
| Age | **0.42** | **0.07** | **2.96** | **.005** |
| Gender | -0.14 | 0.35 | -1.00 | .323 |
| IQ | -0.10 | 0.02 | -0.72 | .473 |
| Cumulative CA x Friendship quality | -0.10 | 0.10 | -0.71 | .483 |

**Table I.4.** Bilateral thalamus reactivity was not related to cumulative childhood adversity and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.85 | 0.41 | .681 |
| Cumulative CA | -0.002 | 0.29 | -0.02 | .984 |
| Friendship quality | -0.23 | 0.04 | -1.65 | .106 |
| Age | **0.31** | **0.06** | **2.09** | **.042** |
| Gender | -0.09 | 0.29 | -0.63 | .531 |
| IQ | -0.23 | 0.01 | -1.56 | .125 |
| Cumulative CA x Friendship quality | -0.22 | 0.08 | -1.62 | .112 |

**Table I.5.** Left hippocampus reactivity was not related to cumulative childhood adversity and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.24 | 0.26 | .800 |
| Deprivation experiences | 0.15 | 0.47 | 1.03 | .310 |
| Friendship quality | -0.10 | 0.05 | -0.70 | .488 |
| Age | 0.12 | 0.07 | 0.82 | .418 |
| Gender | -0.19 | 0.35 | -1.30 | .200 |
| IQ | -0.03 | 0.02 | -0.21 | .832 |
| Deprivation experiences x Friendship quality | -0.14 | 0.13 | -0.98 | .330 |

**Table I.6.** Bilateral insula reactivity was not related to deprivation experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.86 | -0.60 | .550 |
| Deprivation experiences | -0.12 | 0.60 | -0.84 | .404 |
| Friendship quality | -0.03 | 0.06 | -0.20 | .844 |
| Age | 0.26 | 0.08 | 1.73 | .091 |
| Gender | -0.10 | 0.45 | -0.71 | .481 |
| IQ | -0.02 | 0.02 | -0.14 | .893 |
| Deprivation experiences x Friendship quality | -0.11 | 0.16 | -0.78 | .441 |

**Table I.7.** Left medial prefrontal cortex reactivity was not related to deprivation experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.56 | 1.15 | .257 |
| Deprivation experiences | 0.09 | 0.54 | 0.58 | .563 |
| Friendship quality | 0.15 | 0.06 | 0.98 | .331 |
| Age | 0.08 | 0.08 | 0.53 | .598 |
| Gender | -0.12 | 0.40 | -0.83 | .413 |
| IQ | -0.19 | 0.02 | -1.20 | .237 |
| Deprivation experiences x Friendship quality | -0.04 | 0.14 | -0.25 | .802 |

**Table I.8.** Right nucleus accumbens reactivity was not related to deprivation experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.20 | -0.79 | .433 |
| Deprivation experiences | -0.08 | 0.46 | -0.56 | .579 |
| Friendship quality | -0.14 | 0.05 | -1.06 | .296 |
| Age | **0.43** | **0.07** | **2.99** | **.004** |
| Gender | -0.13 | 0.34 | -0.99 | .326 |
| IQ | -0.11 | 0.02 | -0.76 | .453 |
| Deprivation experiences x Friendship quality | -0.09 | 0.12 | -0.64 | .527 |

**Table I.9.** Bilateral thalamus reactivity was not related to deprivation experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.87 | 0.33 | .743 |
| Deprivation experiences | -0.08 | 0.40 | -0.59 | .561 |
| Friendship quality | -0.23 | 0.04 | -1.64 | .108 |
| Age | **0.31** | **0.06** | **2.16** | **.036** |
| Gender | -0.07 | 0.29 | -0.48 | .635 |
| IQ | -0.23 | 0.01 | -1.56 | .127 |
| Deprivation experiences x Friendship quality | -0.14 | 0.11 | -0.99 | .328 |

**Table I.10.** Left hippocampus reactivity was not related to deprivation experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.25 | 0.25 | .805 |
| Threat experiences | 0.03 | 0.97 | 0.21 | .839 |
| Friendship quality | -0.14 | 0.05 | -0.90 | .372 |
| Age | 0.14 | 0.07 | 0.92 | .363 |
| Gender | -0.18 | 0.36 | -1.24 | .220 |
| IQ | -0.05 | 0.02 | -0.32 | .752 |
| Threat experiences x Friendship quality | -0.13 | 0.34 | -0.80 | .426 |

**Table I.11.** Bilateral insula reactivity was not related to threat experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.88 | -0.74 | .461 |
| Threat experiences | 0.08 | 1.24 | 0.51 | .614 |
| Friendship quality | 0.04 | 0.07 | 0.28 | .782 |
| Age | 0.24 | 0.09 | 1.59 | .120 |
| Gender | -0.09 | 0.46 | -0.65 | .522 |
| IQ | 0.02 | 0.02 | 0.14 | .891 |
| Threat experiences x Friendship quality | 0.07 | 0.43 | 0.46 | .647 |

**Table I.12.** Left medial prefrontal cortex reactivity was not related to threat experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.49 | 1.28 | .205 |
| Threat experiences | 0.25 | 1.08 | 1.53 | .134 |
| Friendship quality | 0.21 | 0.06 | 1.35 | .185 |
| Age | 0.06 | 0.07 | 0.37 | .717 |
| Gender | -0.15 | 0.40 | -1.05 | .297 |
| IQ | -0.17 | 0.02 | -1.14 | .262 |
| Threat experiences x Friendship quality | 0.06 | 0.37 | 0.37 | .712 |

**Table I.13.** Right nucleus accumbens reactivity was not related to threat experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.20 | -0.84 | .407 |
| Threat experiences | 0.03 | 0.95 | 0.22 | .825 |
| Friendship quality | -0.13 | 0.05 | -0.86 | .395 |
| Age | **0.42** | **0.07** | **2.90** | **.006** |
| Gender | -0.14 | 0.35 | -1.02 | .315 |
| IQ | -0.09 | 0.02 | -0.64 | .529 |
| Threat experiences x Friendship quality | -0.06 | 0.33 | -0.42 | .679 |

**Table I.14.** Bilateral thalamus reactivity was not related to threat experiences and friendship quality.

| | $\beta$ | SE | $t_{46}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.77 | 0.46 | .648 |
| Threat experiences | 0.05 | 0.76 | 0.31 | .756 |
| Friendship quality | -0.26 | 0.04 | -1.84 | .072 |
| Age | **0.30** | **0.05** | **2.16** | **.036** |
| Gender | -0.11 | 0.28 | -0.80 | .431 |
| IQ | -0.23 | 0.01 | -1.63 | .110 |
| Threat experiences x Friendship quality | **-0.33** | **0.26** | **-2.26** | **.029** |

**Table I.15.** Left hippocampus reactivity was related to threat experiences and friendship quality. This effect did not survive correction for multiple comparisons ($p_{Bonf}$ = .145; corrected for five ROI comparisons).

**J. Exploratory Analyses (T2; *n* = 60): Full Model Output**

| | ***β*** | **SE** | $t_{48}$ | ***p*** |
|---|---|---|---|---|
| Intercept | | 2.17 | 0.24 | .809 |
| Cumulative CA | 0.16 | 0.33 | 1.11 | .273 |
| Age | 0.12 | 0.07 | 0.79 | .433 |
| Gender | -0.19 | 0.34 | -1.27 | .212 |
| IQ | -0.03 | 0.02 | -0.17 | .866 |

**Table J.1.** Bilateral insula reactivity was not related to cumulative childhood adversity.

| | ***β*** | **SE** | $t_{48}$ | ***p*** |
|---|---|---|---|---|
| Intercept | | 2.76 | -0.77 | .444 |
| Cumulative CA | -0.07 | 0.42 | -0.52 | .605 |
| Age | 0.26 | 0.08 | 1.78 | .082 |
| Gender | -0.08 | 0.44 | -0.58 | .567 |
| IQ | -0.002 | 0.02 | -0.02 | .988 |

**Table J.2.** Left medial prefrontal cortex reactivity was not related to cumulative childhood.

| | ***β*** | **SE** | $t_{48}$ | ***p*** |
|---|---|---|---|---|
| Intercept | | 2.46 | 1.01 | .316 |
| Cumulative CA | 0.10 | 0.38 | 0.72 | .473 |
| Age | 0.10 | 0.07 | 0.65 | .521 |
| Gender | -0.11 | 0.39 | -0.80 | .426 |
| IQ | -0.17 | 0.02 | -1.11 | .274 |

**Table J.3.** Right nucleus accumbens reactivity was not related to cumulative childhood adversity.

| | ***β*** | **SE** | $t_{48}$ | ***p*** |
|---|---|---|---|---|
| Intercept | | 2.14 | -0.80 | .430 |
| Cumulative CA | -0.01 | 0.33 | -0.05 | .963 |
| Age | **0.41** | **0.06** | **2.92** | **.005** |
| Gender | -0.13 | 0.34 | -0.97 | .337 |
| IQ | -0.10 | 0.02 | -0.70 | .488 |

**Table J.4.** Bilateral thalamus reactivity was not related to cumulative childhood adversity.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.85 | 0.37 | .714 |
| Cumulative CA | 0.05 | 0.28 | 0.34 | .738 |
| Age | 0.29 | 0.06 | 1.95 | .057 |
| Gender | -0.06 | 0.29 | -0.45 | .656 |
| IQ | -0.21 | 0.01 | -1.42 | .161 |

**Table J.5.** Left hippocampus reactivity was not related to cumulative childhood adversity.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.17 | 0.20 | .756 |
| Deprivation experiences | 0.16 | 0.46 | 0.92 | .361 |
| Age | 0.12 | 0.07 | 0.80 | .430 |
| Gender | -0.17 | 0.34 | -1.37 | .176 |
| IQ | -0.02 | 0.02 | -1.56 | .125 |

**Table J.6.** Bilateral insula reactivity was not related to deprivation experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.88 | -0.67 | .509 |
| Deprivation experiences | -0.08 | 0.45 | -0.51 | .611 |
| Age | 0.26 | 0.09 | 1.71 | .095 |
| Gender | -0.09 | 0.46 | -0.65 | .519 |
| IQ | -0.23 | 0.01 | -1.56 | .125 |

**Table J.7.** Left medial prefrontal cortex reactivity was not related to deprivation experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.48 | 1.00 | .322 |
| Deprivation experiences | 0.05 | 0.52 | 0.37 | .716 |
| Age | 0.10 | 0.07 | 0.69 | .491 |
| Gender | -0.11 | 0.39 | -0.74 | .461 |
| IQ | -0.18 | 0.02 | -1.15 | .256 |

**Table J.8.** Right nucleus accumbens reactivity was not related to deprivation experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.14 | -0.77 | .445 |
| Deprivation experiences | -0.05 | 0.45 | -0.41 | .684 |
| Age | **0.41** | **0.06** | **2.96** | **.005** |
| Gender | -0.13 | 0.34 | -0.97 | .338 |
| IQ | -0.11 | 0.02 | -0.76 | .453 |

**Table J.9.** Bilateral thalamus reactivity was not related to deprivation experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.86 | 0.40 | .693 |
| Deprivation experiences | -0.05 | 0.39 | -0.34 | .738 |
| Age | **0.30** | **0.06** | **2.02** | **.049** |
| Gender | -0.06 | 0.29 | -0.41 | .688 |
| IQ | -0.23 | 0.01 | -1.53 | .134 |

**Table J.10.** Left hippocampus reactivity was not related to deprivation experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.19 | 0.31 | .755 |
| Threat experiences | 0.11 | 0.86 | 0.76 | .453 |
| Age | 0.12 | 0.07 | 0.81 | .425 |
| Gender | -0.18 | 0.35 | -1.28 | .208 |
| IQ | -0.04 | 0.02 | -0.26 | .793 |

**Table J.11.** Bilateral insula reactivity was not related to threat experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.77 | -0.76 | .451 |
| Threat experiences | 0.05 | 1.09 | 0.35 | .730 |
| Age | 0.25 | 0.08 | 1.67 | .101 |
| Gender | -0.10 | 0.45 | -0.69 | .496 |
| IQ | 0.01 | 0.02 | 0.09 | .925 |

**Table J.12.** Left medial prefrontal cortex reactivity was not related to threat experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.44 | 1.11 | .272 |
| Threat experiences | 0.17 | 0.96 | 1.18 | .242 |
| Age | 0.09 | 0.07 | 0.58 | .565 |
| Gender | -0.14 | 0.39 | -0.95 | .345 |
| IQ | -0.18 | 0.02 | -1.12 | .267 |

**Table J.13.** Right nucleus accumbens reactivity was not related to threat experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 2.13 | -0.76 | .451 |
| Threat experiences | -0.09 | 0.84 | 0.65 | .522 |
| Age | **0.40** | **0.06** | **2.84** | **.007** |
| Gender | -0.15 | 0.34 | -1.09 | .281 |
| IQ | -0.09 | 0.02 | -0.64 | .525 |

**Table J.14.** Bilateral thalamus reactivity was not related to threat experiences.

| | $\beta$ | SE | $t_{48}$ | $p$ |
|---|---|---|---|---|
| Intercept | | 1.81 | 0.48 | .635 |
| Threat experiences | 0.21 | 0.71 | 1.53 | .133 |
| Age | 0.26 | 0.05 | 1.84 | .072 |
| Gender | -0.10 | 0.29 | -0.71 | .479 |
| IQ | -0.20 | 0.01 | -1.38 | .175 |

**Table J.15.** Left hippocampus reactivity was not related to threat experiences.

# Chapter 5

## Friendship Buffering Effects on Mental Health Symptoms Before and During the COVID-19 Pandemic: A UK Longitudinal Study of Young People with Childhood Adversity

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**Abstract**
Young people with childhood adversity (CA) were at increased risk to experience mental health problems during the COVID-19 pandemic. Pre-pandemic research identified high-quality friendship support as a protective factor that can buffer against the emergence of mental health problems in young people with CA. This longitudinal study investigated friendship buffering effects on mental health symptoms before and at three timepoints during the pandemic in 102 young people (aged 16-26) with low to moderate CA. Multilevel analyses revealed a continuous increase in depression symptoms following the outbreak. Friendship quality was perceived as elevated during lockdowns and returned to pre-pandemic baseline levels during reopening. A stress-sensitizing effect of CA on social functioning was evident, as social thinning occurred following the outbreak. Bivariate latent change score modeling revealed that before and during the pandemic, young people with greater friendship quality self-reported lower depression symptoms and vice versa. Furthermore, sequential mediation analysis showed that high-quality friendships before the pandemic buffered depression symptoms during the pandemic through reducing perceived stress. These findings highlight the importance of fostering stable and supportive friendships in young people with CA and suggest that through reducing stress perceptions high-quality friendships can mitigate mental health problems during times of multidimensional stress.

*Keywords*: friendship stress buffering, mental health, COVID-19 pandemic, young people, childhood adversity

## Introduction

The coronavirus disease 2019 (COVID-19) outbreak constituted a global public health emergency that introduced numerous psychosocial challenges, such as social isolation, health concerns, widened social inequalities, and uncertainty about the future (Gruber et al., 2021). This time of multidimensional stress coincided with a global increase in depressive and anxiety disorders during early stages of the COVID-19 pandemic (Hampshire et al., 2021), with some studies identifying young people as being disproportionately affected (Pierce et al., 2020; Santomauro et al., 2021; Xiong et al., 2020). Adolescence and early adulthood are sensitive developmental periods for the emergence of mental health problems (McGrath et al., 2023; Solmi et al., 2022) and having a history of childhood adversity (CA; such as child abuse or neglect) is known to potentiate that vulnerability (Kessler et al., 2010; McLaughlin et al., 2012). Recent longitudinal findings suggest that the COVID-19 outbreak may have exacerbated mental health problems in young people with CA (Stinson et al., 2021). Therefore, investigating the impact of the COVID-19 pandemic on the mental health of young people with CA and identifying protective factors that can mitigate mental health problems is essential for informing targeted psychosocial interventions aimed at boosting resilience in vulnerable young people.

The global prevalence of exposure to CA is estimated at around 50% (Bellis et al., 2014; Green et al., 2010). This includes stressful experiences like abuse, neglect, bullying, or severe poverty, and generally represents a deviation from the "expectable" childhood environment (McLaughlin, 2016). Chronic and repeated exposure to psychosocial stressors require young people to adapt their neurobiological, psychological, and social functioning, ultimately increasing the risk for later-life mental health problems (Cicchetti & Valentino, 2006; Clark et al., 2010; Danese & McEwen, 2012; Kessler et al., 2010; Lupien et al., 2009). Stress is typically perceived when environmental demands outweigh an individual's ability to effectively cope with those demands (Lazarus & Folkman, 1984; Monroe, 2008). In turn, perceived stress is thought to influence the pathogenesis of mental health problems by eliciting negative affective states (e.g., feelings of depression and anxiety), which then exert direct effects on physiological processes or behavioral patterns that influence susceptibility to prolonged mental disorders (Cohen et al., 2007). For example, CA has been associated with hypersensitive threat processing on both a neurobiological (e.g., heightened activity of the hypothalamus-pituitary-adrenal (HPA) axis; (Hein & Monk, 2017; McCrory et al., 2011; Moreno-López et al., 2020) and psychosocial level (e.g., over-attribution of threat-related cues; V. Lee & Hoaken (2007)). This hypervigilance to threat-related cues may be adaptive in the short-term to support survival in dangerous and stressful environments (e.g., maltreatment), but can impair social functioning in the long-term through a compromised ability to negotiate interpersonal challenges (e.g., hostile attributional bias) (McCrory et al.,

2019). The social transactional model of mental health vulnerability (McCrory et al., 2022) posits that such neurocognitive adaptations following CA can inadvertently generate a social environment characterized by more stressful interpersonal experiences (i.e., stress generation; McCrory et al. (2019)) and fewer protective social relationships (i.e., social thinning; Nevard et al. (2021); Sheikh et al. (2016)), consequently increasing mental health problems.

The COVID-19 pandemic was marked by numerous stress-inducing experiences such as risk of serious illness or death. Longitudinal studies in young people without CA from diverse cultural contexts, consistently reported a link between pandemic-related stress exposure and increased levels of mental health problems, specifically depression and anxiety symptoms (Hawes et al., 2022; Kauhanen et al., 2023; Santomauro et al., 2021; Xiong et al., 2020). In addition, the pandemic led to various socio-economic restructuring (e.g., university closures, lack of access to private space), which predicted concurrent eating disorder psychopathology in young people, even after adjusting for baseline CA (Ioannidis et al., 2022). The stress sensitization hypothesis postulates that CA exposure is associated with a lower stress threshold in response to additional stressors encountered later in life, particularly during adolescence, which can give rise to mental health problems (Hammen, 2015; Hammen et al., 2000; La Rocque et al., 2014). In line with that hypothesis, studies conducted during the COVID-19 pandemic have observed that young people with more severe CA were more likely to report elevated depression and anxiety symptoms (Doom et al., 2021; Gotlib et al., 2020; Guo et al., 2020; Kalia et al., 2020; Stinson et al., 2021). Moreover, Gotlib et al. (2020) and Achterberg et al. (2021) identified perceived stress as a potential mechanism linking challenging pre-pandemic experiences, such as CA or psychopathology, with elevated internalizing and externalizing behavior during the first two months of the COVID-19 pandemic. However, the number of studies investigating pandemic-related mental health vulnerability in young people with CA is limited and requires further examination.

Although having a history of CA is associated with a higher risk of later-life mental health problems, a substantial proportion of individuals are able to maintain or regain mental health despite exposure to CA (Ioannidis et al., 2020; Kalisch et al., 2017). Research conducted before the COVID-19 pandemic has shown that social support, particularly perceived friendship support, is a potent stress buffer capable of protecting young people with CA against the emergence and progression of mental health problems (König et al., 2023; van Harmelen et al., 2016, 2021). The availability of social support has also proven to buffer against the emergence of mental health problems following later-life stress exposure such as natural disasters or terrorist attacks (Bonanno et al., 2007, 2011). Despite growing evidence that the COVID-19 pandemic had a disproportionate impact on the mental health of young people with CA, compared to those without CA (Gotlib

et al., 2020; Kalia et al., 2020), few studies have investigated social buffering effects on mental health symptoms during the pandemic in young people with CA (McLaughlin et al., 2022) and most studies lacked access to pre-pandemic baseline measures (Kauhanen et al., 2023). Studies involving young people without CA have shown that those with higher levels of perceived social support, particularly friendship support, reported lower levels of depression and anxiety symptoms during the COVID-19 pandemic (Bernasco et al., 2021; Grey et al., 2020; Houghton et al., 2022; Juvonen et al., 2022; Magson et al., 2021; Özmete & Pak, 2020). Furthermore, those who felt virtually more connected with their friends during national lockdowns also reported lower levels of depression and anxiety symptoms (W. E. Ellis et al., 2020; Magson et al., 2021; McKinlay et al., 2022). In fact, not being able to see their friends was the greatest concern of young people during the first pandemic-related lockdown in Australia (Magson et al., 2021). This concern was rated as most distressing over and above health concerns, disruptions to daily routines, and educational worries.

The Resilience after COVID-19 Threat (REACT) study offers the rare opportunity to investigate friendship buffering effects on mental health symptoms before and at three timepoints during the COVID-19 pandemic in 102 young people (aged 16-26) with retrospectively self-reported low to moderate CA (A. J. Smith et al., 2021). Specifically, young people were assessed pre-pandemic (August 2019 to March 2020), during the first national lockdown in the UK (April to May 2020), during phased reopening (July to August 2020), and leading up to and during the second lockdown (October to November 2020). Prior to the pandemic, we have investigated the same sample of young people with CA and observed an association between greater perceived friendship quality and better mental health (König et al., 2023). In addition, we found that in a representative MRI sub-sample ($n$ = 62), high-quality friendships may aid hippocampal stress responsivity in those with threat experiences. Building on these findings, we first investigated the impact of the COVID-19 pandemic on psychosocial functioning. Specifically, we hypothesized that in response to the COVID-19 outbreak, young people with CA would report an overall increase in depression and anxiety symptoms (Hawes et al., 2022; Kauhanen et al., 2023; Santomauro et al., 2021; Xiong et al., 2020; hypothesis 1.1) as well as a reduction in perceived friendship quality (Bernasco et al., 2021; Magson et al., 2021; hypothesis 1.2). In addition, we expected these trends to be exacerbated during lockdown periods given that research by Pedersen et al. (2022) has shown poorer mental health outcomes in response to lockdowns and improved outcomes related to reopening phases. Second, we investigated CA exposure as a risk factor for poorer psychosocial outcomes during the COVID-19 pandemic. Specifically, we hypothesized that during the COVID-19 pandemic more severe CA would be associated with worse depression and anxiety symptoms (Gotlib et al., 2020; Kalia et al., 2020; hypothesis 2.1) as well as lower levels of perceived friendship quality (McCrory et

al., 2022; Nevard et al., 2021; Sheikh et al., 2016; hypothesis 2.2). Third, we investigated whether any friendship buffering effect observed before the COVID-19 pandemic would also extend into the COVID-19 pandemic. Specifically, we hypothesized that higher friendship quality would be associated with lower depression and anxiety symptoms before and during the COVID-19 pandemic (Bernasco et al., 2021; Houghton et al., 2022; Juvonen et al., 2022; König et al., 2023; Magson et al., 2021; Özmete & Pak, 2020; hypothesis 3). Finally, we explored the role of perceived stress during the COVID-19 pandemic as a potential mechanism linking pre-pandemic friendship quality with mental health symptoms during the COVID-19 pandemic (Achterberg et al., 2021; Gotlib et al., 2020).

## Method

### *Study Description*

The REACT study (A. J. Smith et al., 2021) builds on the Resilience after Individual Stress Exposure (RAISE) study, a UK multilevel study of young people aged 16-26 with retrospectively self-reported CA. The RAISE study commenced in August 2019 and was terminated prematurely in March 2020 due to a pandemic-related university-wide closure of laboratory research activities (Moreno-López et al., 2021). For the REACT study, we contacted all RAISE participants ($N$ = 102, $M_{age}$ = 22.24, 64.7% female) who had previously provided consent at the *pre-pandemic baseline* to be recontacted for future studies. This study utilized data collected at four online assessment timepoints. The pre-pandemic baseline took place between August 2019 and March 2020. The first follow-up assessment occurred from April to May 2020, during the first national lockdown in the UK (*first lockdown*: $n$ = 79). The second follow-up assessment occurred from July to August 2020, a period of eased restrictions (*reopening*: $n$ = 77). The final follow-up assessment occurred from October to November 2020, a second phase of increased pandemic-related restrictions (*second lockdown*: $n$ = 73) (Figure 1). All participants provided informed consent for both the RAISE and REACT studies. Comprehensive study protocols for both the RAISE study (Moreno-López et al., 2021) and the REACT study (A. J. Smith et al., 2021) have been previously published. Participants were recruited across Cambridgeshire, UK from the general population through flyers and via social media as well as from previous studies conducted at the Department of Psychiatry, University of Cambridge (NSPN 2400 Cohort; Kiddle et al. (2018)). The RAISE study received funding from the Royal Society in January 2018 and ethical approval from the National Research Ethics Service and the NRES Committee East of England-Cambridge Central (REC reference: 18/EE/0388, IRAS project ID: 241765) in February 2019. The REACT study was approved to be funded by the same grants and received ethical approval from the Cambridge Psychology Research Ethics Committee (PRE.2020.037).

**Participants**

Individuals were eligible to participate if they were aged between 16-26 years, able to speak, write, and understand English, and self-reported CA before the age of 16. Eligibility criteria were assessed via telephone before the pre-pandemic baseline by a trained member of the study team. Participants received a total of £100 upon completion of all four study phases. Specifically, participants received £10 for the completion of the pre-pandemic baseline assessments and £30 for the completion of each follow-up assessment. A dropout analysis using two-sample *t*-tests compared characteristics between the second lockdown sample and the participants ($n$ = 29) who dropped out before that assessment timepoint, indicating that attrition was random and not influenced by specific sample characteristics (e.g., age, gender identity, CA experiences, or friendship quality; supplementary Table S1).

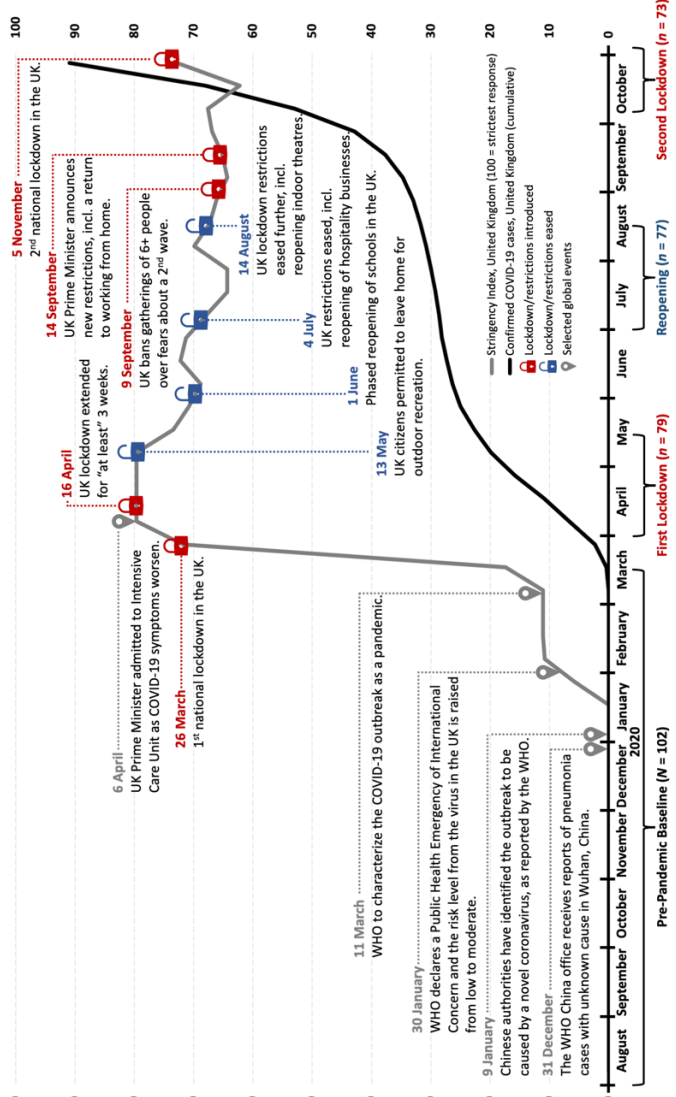


**Figure 1.** Study assessment timepoints and UK COVID-19 cases and restrictions from August 2019 to November 2020. The Stringency Index is a composite measure comprised of nine metrics including school and workplace closures, cancellation of public events, and travel bans. A higher Stringency Index indicates stricter UK government policies. Data related to the UK Stringency Index and confirmed COVID-19 cases has been retrieved from the Oxford COVID-19 Government Response Tracker (Hale et al., 2021). In addition, information about selected UK COVID-19 restrictions and developments has been retrieved from the (Institute for Government, 2022) and the British Foreign Policy Group (Aspinall, 2020).

### *Measures*

At all assessment timepoints, participants received an email with a secure online link to remotely complete self-report questionnaires. All questionnaires (incl. instructions and items) were carefully selected to ensure accessibility and age-appropriateness for our entire sample, which ranged in age from 16 to 26 (Demkowicz et al., 2021). Only the measures relevant for the current study are

reported below (supplementary Table S1), for a complete list of measures please see Moreno-López et al. (2021) and A. J. Smith et al. (2021).

*Mental Health*

The *Mood and Feelings Questionnaire* (MFQ; Angold & Costello (1987)) was used to assess current (i.e., past two weeks) depression symptoms. At each assessment timepoint, participants rated 33 items such as "I felt miserable or unhappy" on a 4-point Likert scale (1 = never, 4 = always). Positive items were reverse coded so that higher scores indicate more depression symptoms. Internal consistency for the total scale was excellent across all assessment timepoints (pre-pandemic baseline: α = .94, first lockdown: α = .93, reopening: α = .95, second lockdown: α = .95).

The *Revised Children's Manifest Anxiety Scale* (RCMAS; Reynolds & Richmond (1978)) was used to assess current (i.e., past two weeks) anxiety symptoms. At each assessment timepoint, participants rated items such as "I worried a lot of the time" on a 4-point Likert scale (1 = never, 4 = always). Positive items were reverse coded so that higher scores indicate more anxiety symptoms. This 28-item screening measure comprises three subscales (physiological anxiety, worry/oversensitivity, social concerns/concentration), which were combined to estimate the total severity of anxiety symptoms. Internal consistency for the total scale was excellent across all assessment timepoints (pre-pandemic baseline: α = .94, first lockdown: α = .95, reopening: α = .95, second lockdown: α = .95).

*Perceived Friendship Quality*

The *Cambridge Friendship Questionnaire* (CFQ; van Harmelen et al. (2017)) was used to assess the self-reported number, availability, and quality of current friendships. At each assessment timepoint, participants rated items such as "Do you feel that your friends understand you?". Negative items were reverse coded so that higher scores indicate greater perceived friendship quality. As previously detailed (König et al., 2023), an exploratory factor analysis conducted on the 8-items of the CFQ revealed low factor loading (< .40; Stevens (2001)) of item 6 ("Do people who aren't your friends laugh at you or tease you in a hurtful way?"), which therefore was excluded from all subsequent analyses. Internal consistency for the 7-item solution was acceptable across all assessment timepoints (pre-pandemic baseline: α = .75, first lockdown: α = .73, reopening: α = .68, second lockdown: α = .77).

*Perceived Stress*

The *Perceived Stress Scale* (PSS; Sheldon Cohen et al. (1983)) was used to assess current (i.e., past two weeks) levels of appraised stress, but was only assessed during the follow-up timepoints. At each follow-up assessment timepoint, participants rated 10 items such as "How often have you felt nervous and stressed"

on a 5-point Likert scale (1 = never, 5 = very often). Positive items were reverse coded so that higher scores indicate greater levels of perceived stress. Internal consistency for the total scale was excellent across all assessment timepoints (first lockdown: α = .88, reopening: α = .93, second lockdown: α = .86).

*Childhood Adversity*

At pre-pandemic baseline, different types of CA experiences were assessed using three retrospective self-report questionnaires: the Short-Form of the Childhood Trauma Questionnaire, the Measure of Parental Style Questionnaire, and the Alabama Parenting Questionnaire. Positive items on these questionnaires were reverse coded so that higher scores indicate more severe CA experiences. See below for details on how these scales were further processed to compute a cumulative CA index. Please note that this analytic procedure has been applied and presented to full detail in previous works (König et al., 2023). However, for completeness, we will provide a summary of its methodological details here.

The *Short-Form of the Childhood Trauma Questionnaire* (CTQ-SF; Bernstein et al. (2003)) was used to assess maltreatment experiences within the family environment during childhood or adolescence. Participants rated items such as "I didn't have enough to eat" on a 5-point Likert scale (1 = never true, 5 = very often true). This 28-item screening measure comprises five subscales (sexual, physical, and emotional abuse, and physical and emotional neglect), which can be combined to estimate a total severity score. Internal consistency was excellent for the total scale (Cronbach's α = .92) and acceptable to excellent for the four subscales (physical abuse: α = .81; emotional abuse: α = .85; physical neglect: α = .72; emotional neglect: α = .93). The sexual abuse subscale (α = .94) was excluded from all analyses due to too low prevalence (*Mdn* = 0, IQR = 0). Based on established cut-off scores for the CTQ (Bernstein et al., 1994), our baseline sample can be characterized reporting low to moderate levels of CA.

The *Measure of Parental Style Questionnaire* (MOPS; Parker et al. (1997)) was used to assess adverse maternal and paternal parenting style experiences. Participants rated items such as "My father was physically violent or abusive to me" on a 4-point Likert scale (1 = not true at all, 4 = extremely true). This 30-item screening measure comprises six subscales (maternal and paternal abuse, -indifference, and -overcontrol), which can be combined to estimate a total severity score. Internal consistency was excellent for the total maternal scale (α = .91) and paternal scale (α = .90) and acceptable to good for the six subscales (maternal abuse: α = .86, -indifference: α = .88; -overcontrol: α = .78; paternal abuse: α = .77; -indifference: α = .90; -overcontrol: α = .89).

The *Alabama Parenting Questionnaire* (APQ; Frick (1991)) was used to assess past adverse parenting experiences. Participants rated items such as "Your

parents spanked you with their hand when you have done something wrong" on a 5-point Likert scale (1 = never true, 5 = very often true). This 42-item screening measure comprises five subscales (corporal punishment, parental involvement, negative parenting, poor monitoring/supervision, and inconsistent discipline), which can be combined to estimate a total severity score. At pre-pandemic baseline, a modified 15-item version of the APQ was administered retaining all five subscales (guided by Elgar et al. (2007)). Internal consistency was poor for two subscales (poor monitoring/supervision: α = .51; inconsistent discipline: α = .57), which led to their exclusion from all analyses. Internal consistency was good for the 9-item total scale (α = .85) and acceptable to good for the remaining three subscales (corporal punishment: α = .86; parental involvement: α = .77; negative parenting: α = .83).

**Principal Component Analysis**

To compute a *cumulative CA index* (higher index indicating more severe CA experiences), a principal component analysis (PCA) with non-orthogonal (oblique) rotation was run on individual scores of the three APQ subscales, four CTQ-SF subscales, and six MOPS subscales. The PCA was run using the psych R package (version 2.3.3; Revelle (2022)) and mean imputations to replace missing values were performed using the mice R package (version 3.16.0; Van Buuren & Groothuis-Oudshoorn (2011)). The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis (KMO = .85; "meritorious" according to Kaiser (1974)) and all KMO values for individual items were ≥.70. Bartlett's test of sphericity, $\chi^2_{78} = 722.86$, $p < .001$, indicated that correlations between items were sufficiently large for a PCA. Examining the scree plot in the context of our relatively small sample size led us to retain a two-component solution. The principal component (PC) scores and their associations have been previously visualized and summarized by König et al. (2023). In summary, PC1 explained 37% of variance and is referred to as the *deprivation dimension* because most items capture experiences related to the absence of expected inputs from the environment. PC2 explained 21% of variance and is referred to as the *threat dimension* because most items capture experiences related to harm or threat of harm. To account for the contributions of both PCs, we weighted the scores for each PC by their explained variance and subsequently summed these scores to compute a single index of total severity experienced. This *cumulative CA* index was utilized in all subsequent analyses. Please note that dimensional effects of CA were not the focus of the current study and are therefore only reported in the supplementary information on an exploratory basis (Tables S10-S15).

**Statistical Analysis**

All analyses were performed in R (version 4.3.0; R Core Team (2022)). In case of missing questionnaire data, total scores were only derived if 100% of items were answered for scales with <15 items or if 85% of items were answered for scales

with ≥15 items. This resulted in an average of 1.96% of missing questionnaire data at pre-pandemic baseline, 1.27% during the first lockdown, 6.49% at reopening, and 1.37% during the second lockdown (supplementary Table S2). Outliers were detected and excluded based on the Rosner's test (EnvStats R package version 2.7.0; Millard (2013)). First, we examined whether the COVID-19 outbreak was associated with an increase in depression and anxiety symptoms (hypothesis 1.1) as well as a reduction in perceived friendship quality (hypothesis 1.2). Specifically, we examined whether these trends were exacerbated during lockdown periods. Second, we examined whether more severe CA exposure was associated with greater depression and anxiety symptoms (hypothesis 2.1) as well as lower levels of perceived friendships quality (hypothesis 2.2) before and during the COVID-19 pandemic. Third, we examined whether higher friendship quality would be associated with lower depression and anxiety symptoms before and during the COVID-19 pandemic (hypothesis 3). To accomplish this, we utilized linear mixed-effects models (lmerTest R package version 3.1.3; Kuznetsova et al. (2017)). In building our models, we started with a random intercept model including only the fixed effect of assessment timepoint. In step 2, we added fixed effects for CA or friendship support to determine their additional predictive value. In step 3, we added the interaction terms for assessment timepoint x CA or assessment timepoint x friendship support to account for potential differential impacts of CA or friendship support over time. In step 4, we added age at assessment timepoint and gender identity as covariates. Subject-level random intercepts were included for all models (Baayen et al., 2008) and reported coefficients were standardized using *z*-scores. Across all models, missing data was handled using maximum likelihood estimation allowing for the comparison of nested models using Akaike Information Criteria (AIC; Akaike (1974)). Model fit was assessed using both the AIC value with a lower value indicating better model fit as well as likelihood ratio tests with a non-significant difference ($p > .05$) resulting in the retention of the more parsimonious model. Please see our supplementary information for all model specifications and a summary of model fit indices (Tables S4-S26). Main effects of the best fitting models were inspected using omnibus Type III *F* tests with Satterthwaite's approximation for degrees of freedom. Post-hoc analyses were corrected for multiple comparisons using the false discovery rate (FDR) correction method (Benjamini & Hochberg, 1995). Given that mental health indicators were found to fluctuate depending on pandemic-related social distancing restrictions (Pedersen et al., 2022), we further explored the interrelationships between changes in friendship quality and mental health symptoms across all assessment timepoints. Specifically, based on the Kievit et al. (2018) tutorial, three exploratory bivariate latent change score (BLCS) models (lavaan R package version 0.6.16; Rosseel (2012)) were built to examine the interplay between perceived friendship quality and mental health symptoms from pre-pandemic baseline to first lockdown, first lockdown to reopening, and reopening to second lockdown (see supplementary section G for further

information; Figure S5). Because of our comparatively small sample size, we chose to analyze three distinct BLCS models instead of incorporating all relationships into a single model (Hertzog et al., 2006). Finally, we explored whether perceived stress during the first lockdown mediated the relationship between pre-pandemic levels of perceived friendship quality and mental health symptoms during reopening. Given that perceived stress was only assessed during follow-up and mediation has been proposed to represent a process that unfolds over time (O'Laughlin et al., 2018), we used a sequential mediation analysis (sem R package version 3.1.15; J. Fox (2006)) to capture the temporal sequence of the process of interest (Cain et al., 2018). To further explore the self-reported psychosocial experiences of young people with CA during the COVID-19 pandemic, we analyzed four items from the COVID-19 Adolescent Symptom and Psychological Experience Questionnaire (CASPE; Ladouceur, 2020) with findings detailed in the supplementary information (section J; Figures S9-S11).

Two post-hoc simulation-based power analyses were conducted. First, we used the mixedpower R package (version 0.1.0; Kumle et al. (2021)) to estimate power in our linear mixed-effects model examining the main effect of friendship quality on depression symptoms before and during the COVID-19 pandemic (Marginal $R^2$ = .178; Conditional $R^2$ = .668). Results of these Monte Carlo simulations indicated that a sample size of $N$ = 70 corresponds to more than 80% power for detecting the main effect. Hence, our sample sizes, ranging from $N$ = 102 before the COVID-19 pandemic to $N$ = 73 during the second lockdown, should provide at least 80% power to detect main effects. Second, to estimate sample size and power for our sequential mediation model, we ran Monte Carlo simulations via the Shiny App developed by Schoemann et al. (2017) (available at https://schoemanna.shinyapps.io/mc_power_med/). These simulations indicated that a sample of $N$ = 73 participants results in 80% power for detecting the indirect effect (ab path). Further details on these statistical power considerations are provided in the supplementary information (section I).

## Results

### *The Impact of the COVID-19 Pandemic on Mental Health Symptoms (Hypothesis 1.1)*

Compared to pre-pandemic baseline levels, depression symptoms significantly increased during the COVID-19 pandemic ($\beta$ = 0.06, SE = 0.03, 95% CI [0.01, 0.12], $p$ = .016). Specifically, depression symptoms were significantly elevated during the first lockdown ($\beta$ = 0.30, SE = 0.08, 95% CI [0.14, 0.45], $p$ < .001), the reopening ($\beta$ = 0.33, SE = 0.08, 95% CI [0.17, 0.49], $p$ < .001), and the second lockdown ($\beta$ = 0.18, SE = 0.08, 95% CI [0.02, 0.34], $p$ = .024) (Table 1, Figure 2A). Anxiety symptoms were significantly elevated during the first lockdown ($\beta$ = 0.20, SE = 0.08, 95% CI [0.04, 0.37], $p$ = .017), but returned to pre-pandemic baseline levels during reopening (β = 0.15, $p$ = .088), and the second lockdown ($\beta$

= 0.12, $p$ = .184) (Table 1, Figure 2B). No main effect of time was observed for anxiety symptoms across the COVID-19 pandemic ($\beta$ = 0.03, $p$ = .248). Therefore, the following analyses will focus on the effects related to depression symptoms. Findings related to anxiety symptoms are reported in the supplementary information (Tables S4-S5, S8, S14-S16, S18-S22, S26; Figures S3, S4, S6, S8), along with supplementary Table S3 displaying correlations between the main study variables across all assessment timepoints.

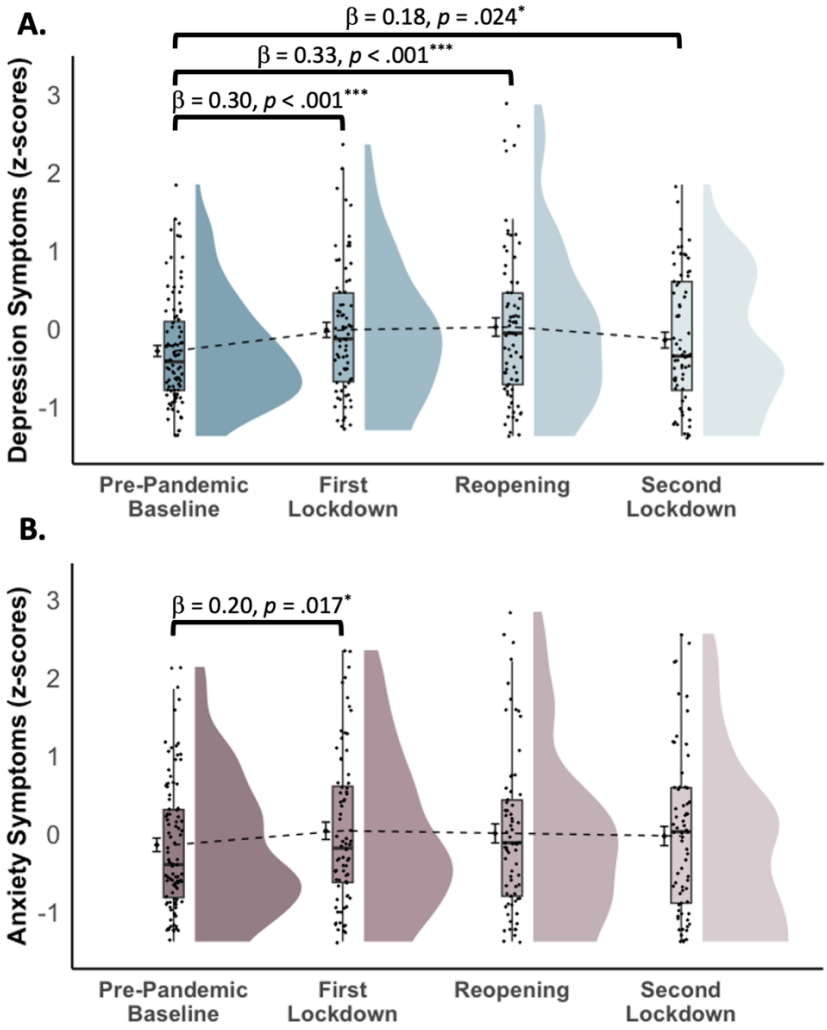


**Figure 2.** (A) depression and (B) anxiety symptoms before and during the COVID-19 pandemic. Compared to pre-pandemic baseline levels, participants self-reported (**A**) elevated depression symptoms during the first lockdown ($p$ < .001), the reopening ($p$ < .001), and the second lockdown ($p$ = .024) and (**B**) elevated anxiety symptoms during the first lockdown ($p$ = .017). The raincloud

plots (Allen et al., 2019) display standardized depression and anxiety scores (y-axis) across all assessment timepoints (x-axis). To emphasize the main effect of time, we first plotted the mean and 95% confidence intervals for each assessment timepoint and connected these with a dashed line. Second, we added box plots showing the median (solid vertical line) and interquartile range. The black dots represent individual raw datapoints. Third, we added violin plots to visualize the probability distribution. $\beta$ = standardized coefficient; $^{*}p < .05$, $^{***}p < .001$.

***The Impact of the COVID-19 Pandemic on Perceived Friendship Quality (Hypothesis 1.2)***

Compared to pre-pandemic baseline levels, our sample reported a significant increase in perceived friendship quality during the first lockdown ($\beta$ = 0.21, SE = 0.08, 95% CI [0.04, 0.38], $p$ = .014), a return to pre-pandemic baseline levels during reopening ($\beta$ = 0.07, $p$ = .436), and another significant increase during the second lockdown ($\beta$ = 0.18, SE = 0.09, 95% CI [0.01, 0.35], $p$ = .039) (Table 1, Figure 3, supplementary Tables S6, S7). No main effect of time was observed for friendship quality across the COVID-19 pandemic ($\beta$ = 0.04, $p$ = .136).

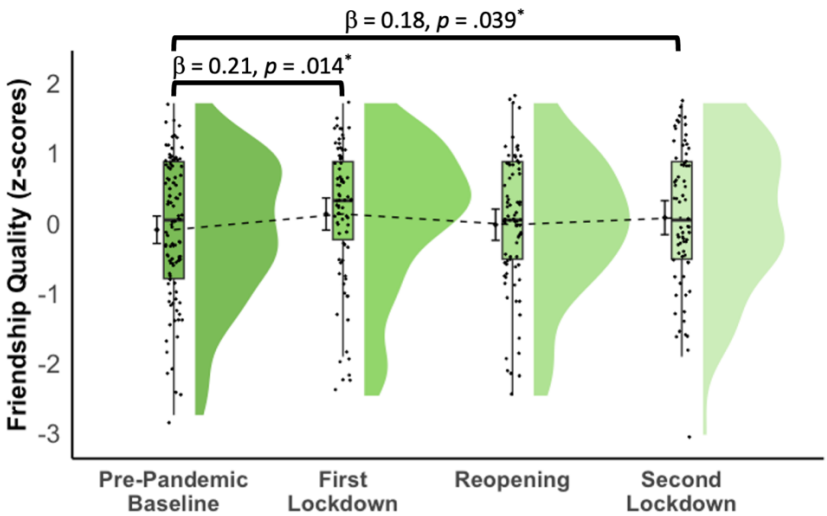


**Figure 3.** Perceived friendship quality before and during the COVID-19 pandemic. Compared to pre-pandemic baseline levels, participants self-reported elevated levels of perceived friendship quality during the first ($p$ = .014) and second lockdown ($p$ = .039). This raincloud plot displays standardized perceived friendship quality scores (y-axis) across all assessment timepoints (x-axis). To emphasize the main effect of time, we first plotted the mean and 95% confidence intervals for each assessment timepoint and connected these with a dashed line. Second, we added box plots showing the median (solid vertical line) and interquartile range. The black dots represent individual raw datapoints. Third, we

added violin plots to visualize the probability distribution. $\beta$ = standardized coefficient; $^{*}p < .05$.

| | *N* | *M (SD)* | *Mdn* | Min | Max | IQR | 95% CI |
|---|---|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | | | |
| Pre-pandemic baseline | 98 | 48.64 (10.51) | 46.50 | 33 | 87 | 13.00 | [46.53, 50.75] |
| First lockdown | 76 | 52.09 (11.39) | 51.50 | 34 | 84 | 15.25 | [49.49, 54.70] |
| Reopening | 74 | 53.05 (14.27) | 51.00 | 33 | 91 | 17.75 | [49.75, 56.36] |
| Second lockdown | 70 | 51.03 (12.65) | 47.00 | 33 | 92 | 18.50 | [48.01, 54.05] |
| **B. Anxiety Symptoms** | | | | | | | |
| Pre-pandemic baseline | 98 | 45.86 (12.23) | 42.00 | 28 | 78 | 17.50 | [43.41, 48.31] |
| First lockdown | 77 | 48.14 (13.74) | 45.00 | 28 | 81 | 17.00 | [45.03, 51.26] |
| Reopening | 71 | 48.08 (14.81) | 46.00 | 28 | 88 | 18.00 | [44.58, 51.59] |
| Second lockdown | 70 | 47.39 (14.44) | 48.00 | 28 | 84 | 21.00 | [43.94, 50.83] |
| **C. Friendship Quality** | | | | | | | |
| Pre-pandemic baseline | 102 | 27.47 (3.55) | 28.00 | 18 | 34 | 6.00 | [26.77, 28.17] |
| First lockdown | 78 | 28.29 (3.61) | 29.00 | 19 | 34 | 4.00 | [27.48, 29.11] |
| Reopening | 77 | 27.73 (3.49) | 28.00 | 19 | 34 | 5.00 | [26.94, 28.52] |
| Second lockdown | 72 | 28.04 (3.72) | 28.00 | 17 | 34 | 5.00 | [27.17, 28.92] |

**Table 1.** Descriptive statistics for (A) depression Symptoms, (B) anxiety symptoms, and (C) friendship quality before and during the COVID-19 pandemic. Descriptive statistics are provided for raw scores of the respective self-report questionnaire, following outlier removal. IQR = interquartile range, 95% CI = 95% confidence interval.

### *The Impact of Childhood Adversity on Depression Symptoms Before and During the COVID-19 Pandemic (Hypothesis 2.1)*

Depression symptoms before and during the pandemic were not related to CA. Specifically, compared to the baseline model which included only assessment timepoint as a main effect (AIC = 636.43, BIC = 658.93), adding CA as a predictor did not significantly improve model fit (AIC = 634.82, BIC = 661.06, $p > .05$) (see supplementary Table S8).

***The Impact of Childhood Adversity on Perceived Friendship Quality Before and During the COVID-19 Pandemic (Hypothesis 2.2)***

When controlling for all assessment timepoints, more severe CA was associated with lower friendship quality ($\beta$ = -0.42, SE = 0.18, 95% CI [-0.78, -0.07], $p$ = .020). FDR-corrected post-hoc analyses revealed that this relationship was present at each assessment timepoint during the COVID-19 pandemic ($ps_{FDR}$ = .022) but absent at pre-pandemic baseline ($p_{FDR}$ = .078) (Figure 4 and supplementary Tables S8-S9, Figure S1).

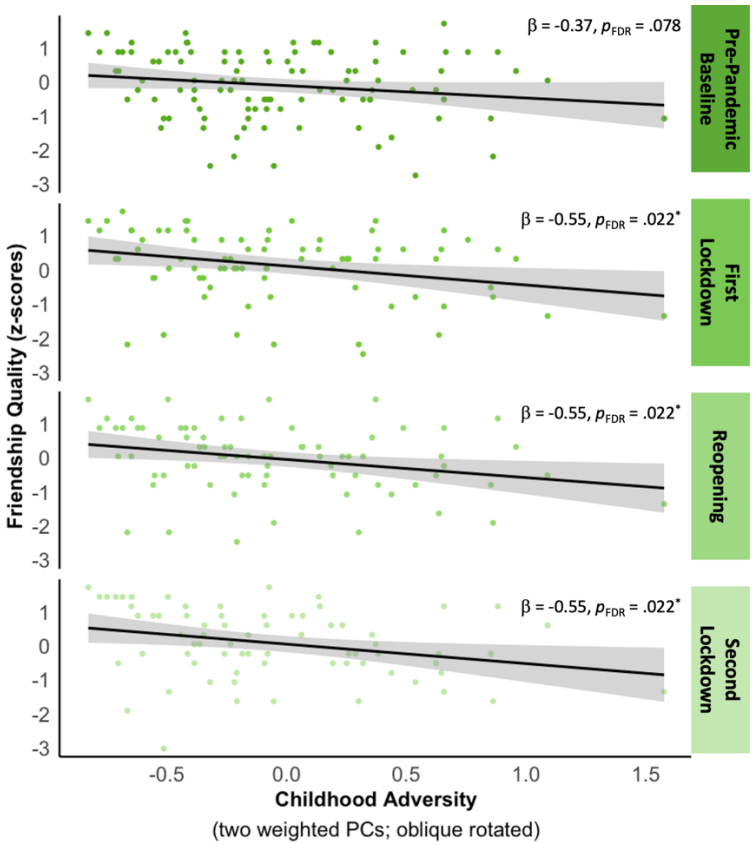


**Figure 4.** Childhood adversity effects on perceived friendship quality before and during the COVID-19 pandemic. Participants with more severe CA (x-axis) self-reported lower friendship quality (y-axis) at each assessment timepoint during the COVID-19 pandemic ($ps_{FDR}$ = .022) but not at pre-pandemic baseline ($p_{FDR}$ = .078). Index scores of CA comprise two weighted and oblique rotated principal components (PCs). Both axes represent standardized scores. The shading of individual data points corresponds to the four different assessment timepoints. The black lines show the best-fitting linear regression lines, and the shaded

regions represent the 95% confidence intervals. $\beta$ = standardized coefficient; $^{*}p_{FDR}$ < .05.

### *Friendship Effects on Depression Symptoms Before and During the COVID-19 Pandemic (Hypothesis 3)*

When controlling for all assessment timepoints, greater perceived friendship quality was associated with lower levels of depression symptoms ($\beta$ = -0.35, SE = 0.06, 95% CI [-0.48, -0.22], $p$ < .001). FDR-corrected post-hoc analyses confirmed that this negative relationship between friendship quality and depression symptoms was present at each assessment timepoint ($ps_{FDR}$ < .003) (Figure 5 and supplementary Tables S16-S17, Figure S2).

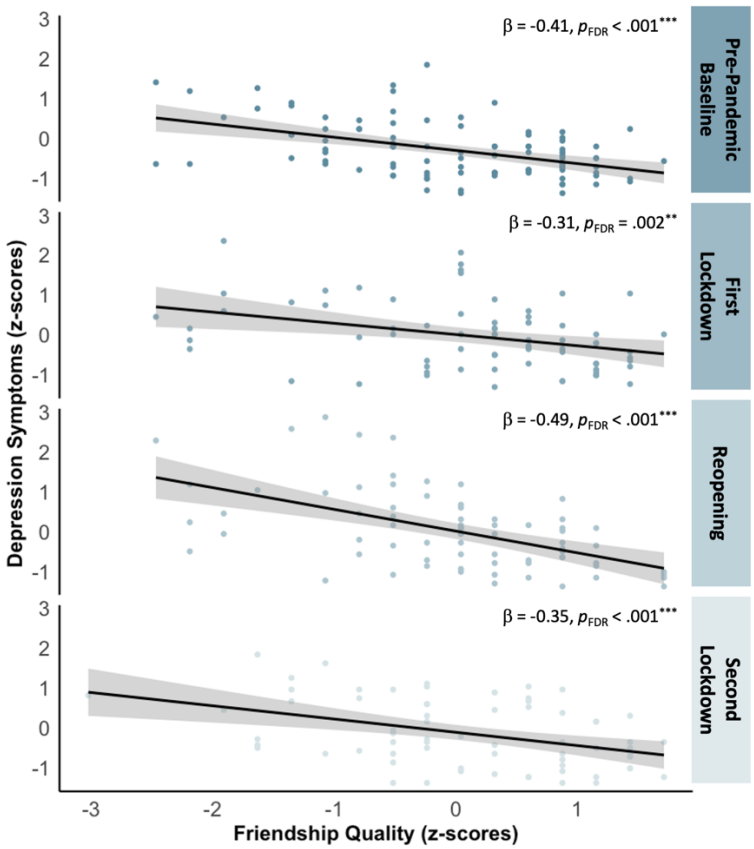


**Figure 5.** Friendship effects on depression symptoms before and during the COVID-19 pandemic. Participants with greater perceived friendship quality (x-axis) also self-reported lower levels of depression symptoms (y-axis) across all assessment timepoints ($ps_{FDR}$ < .003). Both axes represent standardized scores. The black lines show the best-fitting linear regression lines and the shaded regions

represent the 95% confidence intervals. $\beta$ = standardized coefficient; $^{**}p_{FDR} < .01$, $^{***}p_{FDR} < .001$.

### *Exploring the Interplay Between Perceived Friendship Quality and Depression Symptoms From Before to During the COVID-19 Pandemic*

We utilized BLCS modeling to explore the interrelationships between perceived friendship quality and depression symptoms from before to during the COVID-19 pandemic (Figure 6, supplementary Tables S19-S22). Specifically, we estimated the dynamics between both domains of interest from pre-pandemic baseline to first lockdown, first lockdown to reopening, and reopening to second lockdown.

*Pre-Pandemic Baseline to First Lockdown*

First, we observed a strong negative correlation between friendship quality and depression symptoms at pre-pandemic baseline (Est = -0.33, SE = 0.08, $z$ = -4.15, $p < .001$), indicating that individuals with greater friendship quality self-reported lower depression symptoms and vice versa (Figure 6A).

Second, greater friendship quality at pre-pandemic baseline was negatively associated with change in friendship quality between pre-pandemic baseline and the first lockdown (Est = -0.25, SE = 0.09, $z$ = -2.86, $p$ = .004). This indicates that those with already high friendship quality before the pandemic showed a slower increase in friendship quality when entering the first lockdown. Those with lower friendship quality instead reported a stronger increase in perceived quality when entering the first lockdown. However, greater friendship quality at pre-pandemic baseline was not associated with change in depression symptoms between both timepoints (Est = 0.06, $p$ = .512). Furthermore, greater depression symptoms at pre-pandemic baseline were neither associated with change in friendship quality (Est = 0.01, $p$ = .916) nor the change in depression symptoms (Est = -0.22, $p$ = .101).

Third, after accounting for these coupling and self-feedback pathways, we observed that changes in both friendship quality and depression symptoms were negatively correlated (Est = -0.15, SE = 0.06, $z$ = -2.72, $p$ = .007), suggesting that changes in both domains co-occur at the same time. In other words, a greater change in friendship quality co-occurred with a slower change in depression symptoms and vice versa.

*First Lockdown to Reopening*

First, we observed a strong negative correlation between friendship quality and depression symptoms during the first lockdown (Est = -0.31, SE = 0.10, $z$ = -2.99, $p$ = .003), indicating that individuals with greater friendship quality self-reported lower depression symptoms and vice versa (Figure 6B).

Second, greater friendship quality during the first lockdown was negatively associated with change in friendship quality between the first lockdown and reopening (Est = -0.31, SE = 0.10, $z$ = -3.08, $p$ = .002). This indicates that those with greater friendship quality showed a slower change in friendship quality between both timepoints. Moreover, greater friendship quality during the first lockdown was negatively associated with change in depression symptoms between both timepoints (Est = -0.16, SE = 0.07, $z$ = -2.30, $p$ = .022). This indicates that higher friendship quality during the first lockdown was associated with a slower change in depression symptoms. Furthermore, greater depression symptoms during the first lockdown were negatively associated with both change in depression symptoms (Est = -0.33, SE = 0.09, $z$ = -3.57, $p$ < .001) and change in friendship quality (Est = -0.17, SE = 0.08, $z$ = -2.08, $p$ = .037). This suggests that higher friendship quality during the first lockdown was associated with a slower increase in depression symptoms when entering the reopening period and that higher depressive symptoms during the first lockdown were associated with a slower increase in friendship quality. Such patterns of regression to the mean are often observed (Barnett et al., 2005).

Third, after accounting for these coupling and self-feedback pathways, we observed that changes in both friendship quality and depression symptoms were negatively correlated (Est = -0.13, SE = 0.04, $z$ = -2.99, $p$ = .003), suggesting that changes in both domains co-occur at the same time.

*Reopening to Second Lockdown*

First, we observed a strong negative correlation between friendship quality and depression symptoms during reopening (Est = -0.48, SE = 0.11, $z$ = -4.31, $p$ < .001), indicating that individuals with greater friendship quality self-reported lower depression symptoms and vice versa (Figure 6C).

Second, greater friendship quality during reopening was negatively associated with change in friendship quality between reopening and the second lockdown (Est = -0.25, SE = 0.06, $z$ = -3.87, $p$ < .001), indicating that those with greater friendship quality showed a slower change in friendship quality between both timepoints. However, greater friendship quality during reopening was not associated with change in depression symptoms between both timepoints (Est = 0.03, $p$ = .682). Furthermore, greater depression symptoms during reopening were negatively associated with change in depression symptoms (Est = -0.38, SE = 0.13, $z$ = -2.97, $p$ = .003), but not associated with change in friendship quality (Est = 0.03, $p$ = .764).

Third, after accounting for these coupling and self-feedback pathways, we observed that changes in both friendship quality and depression symptoms were not correlated (Est = -0.08, $p$ = .140).

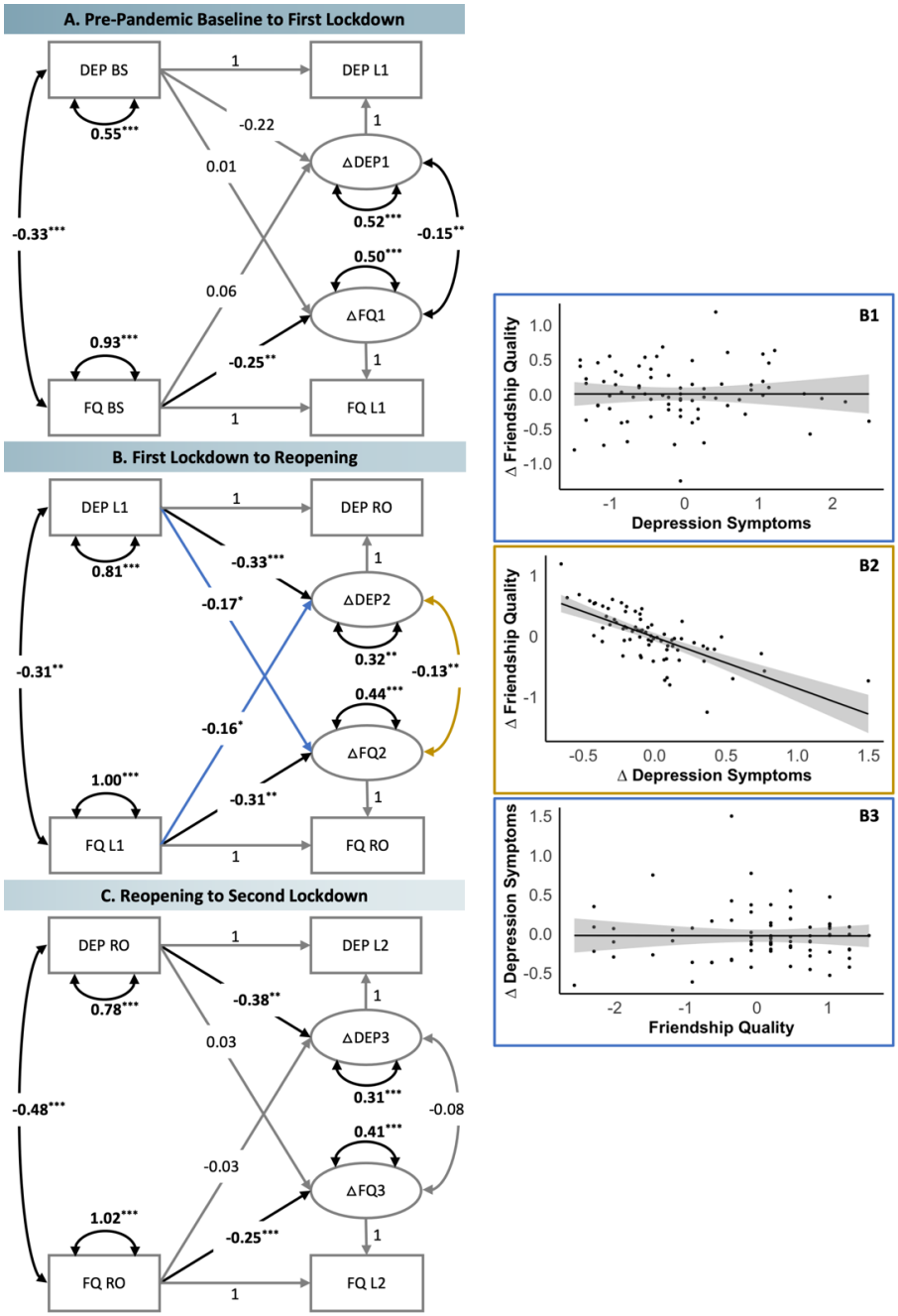


**Figure 6.** The interplay between perceived friendship quality and depression symptoms from before to during the COVID-19 pandemic. Each path shows standardized parameter estimates. FQ = friendship quality domain, DEP = depression symptom domain, BS = pre-pandemic baseline, L1 = first lockdown,

RO = reopening, L2 = second lockdown. Δ = latent change score, → = directed relationship, ↔ = undirected relationship. Path in black denote significant effects. B1 = Correlation between change in friendship quality from the first lockdown to reopening and depression symptoms at the first lockdown, B2 = Correlation between change in friendship quality and change in depression symptoms from the first lockdown to reopening, B3 = Correlation between change in depression symptoms from the first lockdown to reopening and friendship quality at the first lockdown. Comparisons between raw correlations (B1 & B3) and the model estimated coupling parameters indicate potential suppression effects. Refer to supplementary section G for guidance on interpreting these models. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

**Exploring Perceived Stress as a Potential Mechanism Linking Perceived Friendship Quality with Depression Symptoms**

A sequential mediation analysis revealed that perceived stress during the first lockdown fully mediated the relationship between friendship quality during pre-pandemic baseline and depression symptoms during reopening (indirect effect: $\beta$ = -0.13, SE = 0.05, 95% CI [-0.25, -0.05], $p$ = .010; Figure 7). This analysis controlled for gender identity because of a significant main effect on perceived stress across all pandemic assessment timepoints ($\beta$ = 0.61, SE = 0.20, 95% CI [0.21, 1.00], $p$ = .003). Specifically, females reported significantly greater levels of perceived stress during the COVID-19 pandemic than males (supplementary section H; Figure S7; Tables S23-S25).

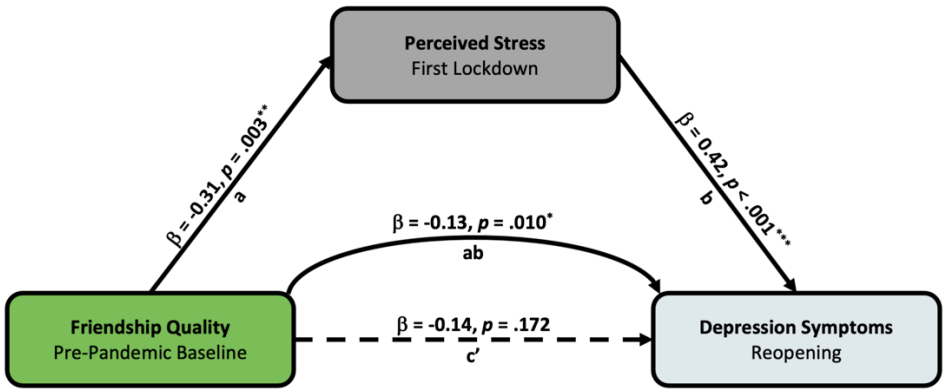


**Figure 7.** Perceived stress mediates the relationship between perceived friendship quality and depression symptoms. Path a shows the standardized regression coefficient of the relationship between friendship quality during pre-pandemic baseline and perceived stress during the first lockdown. Path b shows the standardized regression coefficient of the relationship between perceived stress during the first lockdown and depression symptoms during reopening, while controlling for gender identity. Paths ab (indirect effect) and c' (direct

effect) show the standardized regression coefficient of the relation between friendship quality during pre-pandemic baseline and depression symptoms during reopening without and while controlling for perceived stress during the first lockdown, respectively. Pre-pandemic baseline = August 2019 to March 2020 ($N$ = 97 after outlier removal); First lockdown = April to May 2020 ($n$ = 75 after outlier removal); Reopening = July to August 2020 ($n$ = 73 after outlier removal). Dashed line denotes non-significant effect. $\beta$ = standardized coefficient; $^{*}p < .05$, $^{***}p < .001$.

**Discussion**

In this study, we prospectively assessed friendship buffering effects on mental health symptoms from before to during the COVID-19 pandemic in 102 young people (aged 16-26) with low to moderate CA. Additionally, we explored the mediating role of perceived stress during the COVID-19 pandemic. First, we observed an overall increase in depression symptoms from before to during the COVID-19 pandemic. Anxiety symptoms were significantly elevated during the first lockdown but returned to pre-pandemic baseline levels afterwards. Perceived friendship quality increased during the first and second lockdown but returned to pre-pandemic baseline levels during reopening. Contrary to the stress sensitization hypothesis (Hammen et al., 2000), CA was not predictive of elevated depression symptoms following the COVID-19 outbreak. However, CA was associated with social thinning following the outbreak. Next, high-quality friendships were predictive of lower depression symptoms before and during the COVID-19 pandemic. In addition, we found that improvements in friendship quality co-occurred with reductions in depression symptoms between pre-pandemic baseline and reopening. Finally, we identified perceived stress during the first lockdown as a potential mechanism linking pre-pandemic baseline levels of perceived friendship quality with depression symptoms during reopening. Our findings highlight the importance of fostering stable and supportive friendships in young people with CA and suggest that through reducing stress perceptions high-quality friendships can mitigate mental health problems during times of multidimensional stress.

In line with global longitudinal and meta-analytic findings (Pierce et al., 2020; Racine et al., 2021; Robinson et al., 2022; Santomauro et al., 2021), we observed a continuous increase in depression symptoms following the COVID-19 outbreak. For example, a meta-analysis by Racine et al. (2021), which included 29 studies and 80,879 young people worldwide, found that the global prevalence of clinically elevated depression symptoms in young people increased throughout the first year of the COVID-19 pandemic. A different trend has been observed for anxiety symptoms, which peaked during the first lockdown and returned to pre-pandemic baseline levels afterwards. A similar trajectory has been reported by Fancourt et al. (2021) as well as Robinson et al. (2022) and may be related to a reduction in

perceived threats from uncertain physical and social environments following the first lockdown (Schweizer et al., 2023).

Surprisingly, perceived friendship quality increased during lockdowns compared to periods with less physical restrictions. This finding contrasts with trends observed in German and UK populations, where perceptions of social cohesion (i.e., social integration and stability) declined during pandemic-related lockdowns, particularly among vulnerable groups (Borkowska & Laurence, 2021; Silveira et al., 2022). However, qualitative findings by Larivière-Bastien et al. (2022) suggest that, despite maintaining virtual contact with peers during pandemic-related lockdowns, young people in Canada experienced a shift in their perspectives on in-person socialization and friendships. This shift, characterized by an increased awareness of the irreplaceable nature of friendships, may have triggered greater feelings of appreciation, particularly during lockdowns. Furthermore, the shift from face-to-face to predominantly online social interactions may have especially benefitted those with low-quality friendships, at least concerning access to support (Foulkes & Blakemore, 2021). Our exploratory bivariate latent change score models revealed that higher friendship quality at pre-pandemic baseline (or reopening) was negatively associated with changes in friendship quality between pre-pandemic baseline and the first lockdown (or between reopening and the second lockdown). This suggests that vulnerable young people with higher baseline friendship quality experienced slower changes in friendship quality during the lockdowns, whereas those with lower friendship quality experienced more rapid improvements. Relatedly, D. A. Cole et al. (2017) showed that young people with low-quality friendships may be more successful in receiving support online. One explanation could be that maladaptive social functioning, such as poor social skills, pose less of a risk for social rejection or relationship conflicts when navigating the online world (Breaux et al., 2023; Rodríguez-Domínguez et al., 2022). Furthermore, online social interactions have the advantage of not being geographically constrained, allowing young people with CA to more easily connect with individuals who share similar experiences (Ziebland & Wyke, 2012). Additionally, Wright & Wachs (2023) found that increased technology use for maintaining friendships predicted lower levels of self-isolation and higher friendship quality among young people in the U.S. during a pandemic-related lockdown. This buffering effect, observed around the same time as the first pandemic-related lockdown in the UK, contributes to the growing body of research highlighting the protective role of technology in sustaining relationship with significant others, such as friends, particularly when face-to-face contact is not possible (Juvonen et al., 2022). Having said that, the online world comes, unsurprisingly, with its own set of risks and challenges. For example, a systematic review by Daine et al. (2013) investigated the internet's influence on the risk of self-harm or suicide among vulnerable young people and found that up to 80% of those at risk had been exposed to suicide and self-harm-

related materials online. Additionally, while online forums can be perceived as supportive communities, their use can also expose vulnerable young people to cyberbullying and the normalization of self-harming behaviors (Daine et al., 2013). Relatedly, research by Lytle et al. (2017) explored risk and protective factors for suicidal behaviors in marginalized young people and found that greater perceived in-person social support was linked to reduced odds of experiencing bullying, an effect not observed for perceived online social support. Hence, future research is needed to carefully examine if and how online platforms can be safely harnessed to facilitate meaningful social interactions, especially for vulnerable young people.

Contrary to the stress sensitization hypothesis (Hammen, 2015; Hammen et al., 2000), CA did not exacerbate depression symptoms following the COVID-19 outbreak. This may be because our sample was rather well-functioning, reporting only low to moderate CA as well as on average high levels of pre-pandemic friendship quality (König et al., 2023).While stress sensitization did not predict internalizing problems, it may have affected externalizing behavior and hence social functioning, which would explain our observed social thinning effect following the COVID-19 outbreak. For example, in a sample of young people with severe childhood neglect experiences, Wade et al. (2019) showed that greater exposure to later-life stressors was predictive of more externalizing problems. Translational research is needed to explore how training specific psychosocial skills in young people with CA may foster protective, high-quality, and stable social relationships. One pragmatic and mechanistically informed target for intervention is self-regulation skills training. For example, G. E. Miller et al. (2015) showed that among low-income young people, better self-regulation (i.e., the capacity to regulate one's thoughts, feelings, and actions) was associated with more positive psychosocial outcomes such as reduced depressive symptoms, internalizing problems, substance use, and aggressive behavior. However, better self-regulation in these disadvantaged young people was also associated with accelerated epigenetic aging, highlighting potential unforeseen health costs. Relatedly, Fritz, Fried, et al. (2018) conducted a network analysis to investigate interrelations between empirically grounded protective factors in young people with and without CA. Compared to those without CA, young people with CA demonstrated predominantly antagonistic associations between protective factors. In other words, the degree to which protective factors hamper rather than enhance each other was significantly higher in young people with CA. For example, low expressive suppression (i.e., the conscious display of certain emotions) was associated with low friendship support in young people with CA, but with high friendship support in young people without CA. Hence, to appropriately tailor preventative interventions towards the needs of young people with CA, future research must investigate the dynamics between protective factors

and carefully consider potential health consequences (Méndez Leal & Silvers, 2021).

Next, we replicated recent longitudinal findings in young people without CA showing a link between high-quality friendships and better mental health following the first pandemic-related lockdown in the UK (Ashworth et al., 2022; Wiedemann et al., 2022). Specifically, we observed that high-quality friendship support was associated with lower depression symptoms before and during the COVID-19 pandemic. This study adds to a growing literature highlighting the mental health benefits of social support during the COVID-19 pandemic. For example, Choi et al. (2023) analyzed longitudinal data from 69,066 US adults (aged 18-88) and found that social support was associated with a 55% reduction in the odds of depression symptoms during the COVID-19 pandemic.

By utilizing bivariate latent change score modeling, we were able to further explore the dynamic interplay between friendship quality and depression symptoms before and during the COVID-19 pandemic. Specifically, we ran three models to capture the relations of interest between pre-pandemic baseline and first lockdown, first lockdown and reopening, and reopening and second lockdown. Across all models, young people with greater friendship quality self-reported lower depression symptoms and vice versa. This not only confirms our repeated cross-sectional and longitudinal findings, highlighting how high-quality friendships can protect against depression symptoms experienced before and during the COVID-19 pandemic (Bernasco et al., 2021; Gariépy et al., 2016; Sommerlad et al., 2021; van Harmelen et al., 2016), but also that greater depression symptoms can put young people at risk for experiencing poorer friendship support (Rosenquist et al., 2011). Furthermore, we found that improvements in friendship quality co-occurred with reductions in depression symptoms, which aligns with past pre-pandemic findings in a large sample of young people with CA (van Harmelen et al., 2021). This correlated change was observed between pre-pandemic baseline and reopening and may have disappeared afterwards due to a decreasing trend in depression symptoms. Together, these correlational findings align with the notion that mental health after stress exposure is dynamic and can fluctuate over time and that, at least to some extent, mental health is influenced by friendship support and vice versa (Ioannidis et al., 2020; A. S. Masten, 2014). Next, we observed that young people who entered the COVID-19 pandemic with high-quality friendships were better able to maintain that level of support, even during periods of increased physical distancing (Foulkes & Blakemore, 2021). In turn, these individuals might have been better equipped to deal with pandemic-related stressors as evident by lower depression symptoms. Furthermore, we found that following the COVID-19 outbreak, young people who reported higher depression symptoms were less likely to report reductions in their symptomatology over time. Interestingly, this

association was not observed between pre-pandemic baseline and first lockdown. This is opposite to Fancourt et al. (2021) longitudinal observations and suggests that, at least in our well-functioning sample, pre-existing depression symptomatology was not a risk factor for higher levels of depression symptoms during the first lockdown. Finally, cross-domain coupling effects emerged between the first lockdown and reopening. However, comparisons between raw correlations and the model estimated coupling parameters indicate potential suppression effects (Maassen & Bakker, 2001), which is why we refrain from interpreting these findings. Across all models, we observed significant individual differences in perceived friendship quality, depression symptoms, and their change between timepoints. This should be investigated in future studies as effects found at the group level may not generalize to the individual level (Foulkes & Blakemore, 2018).

Next, we observed that pre-pandemic friendship quality longitudinally buffered depression symptoms during reopening via lowering perceived stress during the first lockdown. This finding aligns with recent research proposing pandemic-related stress perception as a mechanism linking challenging pre-pandemic experiences, such as CA or psychopathology, with reduced mental health and well-being during the COVID-19 pandemic (Achterberg et al., 2021; Gotlib et al., 2020). In addition, we found that females reported higher levels of perceived stress during the COVID-19 pandemic compared to males. This aligns with prior reports indicating a global trend wherein females exhibited a greater increase in the prevalence and burden of mental health problems following the COVID-19 outbreak than males (Choi et al., 2023; Fancourt et al., 2021; Gotlib et al., 2020; Santomauro et al., 2021).

Several limitations should be noted. First, our sample of well-educated young people with low to moderate CA may not fully generalize to young people with more severe CA or to the broader UK population. For example, close to all participants were able to access the internet to not only complete the study but also to stay connected with friends via social media. Relatedly, our participants self-reported on average high-quality friendships before and during the COVID-19 pandemic, suggesting an overall well-functioning group of young people with CA. Second, to comply with UK physical distancing regulations, all data was necessarily derived from remotely collected self-reports. This may have unwillingly led to the exclusion of groups unable to engage remotely. Furthermore, relying solely on self-reports might have led participants to exhibit response tendencies influenced by social desirability or mood states, potentially inflating the relationship among variables (Jordan & Troth, 2020). Future research should carefully consider the role of stress exposure and employ diverse methods, such as investigating whether friendship support also buffers neurobiological stress responses in young people with CA (Scheuplein & van

Harmelen, 2022). Third, we present findings pertaining to one of the most prevalent mental health challenges during adolescence and early adulthood. However, future research needs to investigate whether the buffering effects extend to other mental health conditions such as bipolar disorder, psychosis, or suicidality. Finally, we utilized bivariate latent change score modeling to explore the dynamic interplay between friendship quality and depression symptoms before and during the COVID-19 pandemic. Future longitudinal studies with larger sample sizes and considerable assessment timepoints are needed to replicate these preliminary findings (Brandmaier et al., 2015).

The COVID-19 outbreak evidently exerted adverse effects on the mental health of young people who were already known to be at greater risk for the development of prolonged mental health conditions (McGrath et al., 2023; Solmi et al., 2022). Indeed, a UK national health survey reported that between 1995 and 2014 the prevalence of long-lasting mental health conditions increased up to sixfold across children, adolescents, and young adults (Pitchforth et al., 2019). While part of this trend may be attributed to an increased awareness and reduced stigma surrounding mental health, the growing burden of mental health problems faced by vulnerable young people and COVID-19 as a potential amplifier of these difficulties must be considered by mental health services. To appropriately inform these services a more nuanced understanding of risk and protective factors is needed. For instance, within the same sample, we recently demonstrated that assessing the severity of different CA dimensions aids in specifying neural mechanisms underlying mental health vulnerability (König et al., 2023). Furthermore, the experience of social relationships changed during the COVID-19 pandemic with individuals turning to more remote methods of communication (Ofcom, 2020). From a policy perspective, it will be important to critically investigate the effectiveness of online tools, such as social media, to buffer against negative mental health effects in vulnerable young people (Orben et al., 2020; Ruggeri et al., 2023).

In conclusion, we showed that young people with CA reported a significant increase in depression symptoms following the COVID-19 outbreak and that high-quality friendship support buffered these symptoms through reducing perceived stress. A history of CA in combination with exposure to pandemic-related stress was found to contribute to an attenuated social support network, consequently increasing the risk for mental health problems. Therefore, psychosocial interventions targeting stress (re)appraisals or aimed at fostering stable and supportive friendships could enhance resilience in young people with CA, especially during times of multidimensional stress.

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**Competing Interests**
The authors have no competing interests to declare that are relevant to the content of this article. KI receives a stipend for editorial work from Elsevier.

**Authors' Contributions (CRediT Statement)**
**M König**: Conceptualization, Methodology, Validation, Formal analysis, Data curation, Visualization, Writing - original draft. **AJS**: Investigation, Project administration. **LML**: Investigation, Project administration. **ED**: Investigation. **MD**: Investigation. **SO**: Investigation. **EMM**: Formal analysis. **TSP**: Supervision. **M Kaser**: Investigation. **KI**: Investigation. **ALvH**: Conceptualization, Investigation, Methodology, Supervision, Funding acquisition, Writing - review & editing. All authors read and approved the final manuscript.

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| Characteristics | Pre-Pandemic Baseline (*N* = 102) | First Lockdown (*n* = 79) | Reopening (*n* = 77) | Second Lockdown (*n* = 73) | Dropout Analysis | |
|---|---|---|---|---|---|---|
| | | | | | *t* | *p* |
| **Age** | 22.24 (2.76) | 22.80 (2.69) | 22.96 (2.69) | 23.40 (2.64) | 1.18 | .240 |
| **Gender identity** | | | | | 1.74 | .085 |
| Male | 36 (35.3) | 26 (32.9) | 25 (32.5) | 22 (30.1) | | |
| Female | 66 (64.7) | 53 (67.1) | 52 (67.5) | 51 (69.9) | | |
| **Race** | | | | | -0.29 | .772 |
| Asian | 18 (17.7) | 13 (16.5) | 13 (16.9) | 13 (17.8) | | |
| Black | 4 (3.9) | 2 (2.5) | 2 (2.6) | 2 (2.7) | | |
| White | 69 (67.6) | 57 (72.2) | 55 (71.4) | 52 (71.2) | | |
| Others | 11 (10.8) | 7 (8.8) | 7 (9.1) | 6 (8.3) | | |
| **Highest education** | | | | | 1.56 | .122 |
| GCSEs | 16 (15.8) | 10 (12.7) | 9 (11.7) | 8 (11.0) | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| A-Levels | 29 (28.4) | 23 (29.1) | 23 (29.9) | 21 (28.8) | | |
| Undergraduate degree | 39 (38.2) | 32 (40.5) | 32 (41.6) | 31 (42.5) | | |
| Postgraduate degree | 18 (17.6) | 14 (17.7) | 13 (16.9) | 13 (17.8) | | |
| **Housing** | | | | | | |
| Parent(s) | 30 (29.4) | 22 (27.8) | 21 (27.3) | 18 (24.7) | | |
| University housing | 33 (32.4) | 25 (31.6) | 25 (32.5) | 25 (34.2) | | |
| Rented room | 8 (7.8) | 7 (8.9) | 7 (9.1) | 7 (9.6) | | |
| Rented house or flat | 27 (26.5) | 21 (26.6) | 21 (27.3) | 20 (27.4) | | |
| Owned a house or flat | 4 (3.9) | 4 (5.1) | 3 (3.9) | 3 (4.1) | | |
| **Employment** | | | | | -0.03 | .976 |
| Full-time | 26 (48.1) | 24 (53.3) | 22 (51.2) | 20 (51.3) | | |
| Part-time | 20 (37.0) | 15 (33.3) | 15 (34.9) | 13 (33.3) | | |
| Self-employed | 8 (14.8) | 6 (13.3) | 6 (14.0) | 6 (15.4) | | |
| **Friendship quality** | 27.47 (3.55) | 28.29 (3.61) | 27.73 (3.49) | 28.04 (3.72) | -0.14 | .885 |

| | | | | | | |
|---|---|---|---|---|---|---|
| **Childhood adversity** | | | | | | |
| Sexual abuse | 9.60 (22.10) | 7.06 (4.65) | 6.99 (4.61) | 6.85 (4.42) | -0.26 | .793 |
| Emotional abuse | 30.15 (24.12) | 11.06 (4.91) | 10.95 (4.92) | 11.03 (4.96) | -0.01 | .993 |
| Physical abuse | 7.82 (14.41) | 6.54 (2.89) | 6.48 (2.82) | 6.48 (2.88) | -0.48 | .635 |
| Emotional neglect | 36.80 (25.75) | 12.58 (5.55) | 12.44 (5.55) | 12.44 (5.60) | 0.25 | .804 |
| Physical neglect | 13.45 (15.24) | 7.83 (3.27) | 7.74 (3.19) | 7.75 (3.20) | 0.34 | .734 |
| Negative parenting | 40.68 (23.31) | 7.92 (2.93) | 7.87 (2.94) | 7.82 (2.96) | -0.34 | .733 |
| Poor parental involvement | 52.50 (24.55) | 9.25 (3.12) | 9.16 (3.10) | 9.04 (3.02) | -1.45 | .149 |
| Corporal punishment | 28.28 (25.86) | 6.38 (3.20) | 6.36 (3.23) | 6.38 (3.21) | -0.06 | .956 |
| Paternal abuse | 15.71 (23.86) | 7.49 (3.54) | 7.47 (3.57) | 7.37 (3.55) | 0.06 | .951 |
| Maternal abuse | 14.13 (21.13) | 7.31 (3.28) | 7.25 (3.27) | 7.36 (3.33) | 1.21 | .228 |
| Paternal overcontrol | 24.57 (25.39) | 7.16 (3.21) | 7.05 (3.08) | 7.03 (3.13) | 0.41 | .685 |
| Maternal overcontrol | 32.35 (27.03) | 7.86 (3.38) | 7.91 (3.40) | 8.03 (3.42) | 0.71 | .476 |
| Paternal indifference | 20.20 (26.33) | 9.77 (4.82) | 9.65 (4.83) | 9.75 (4.94) | 0.37 | .714 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Maternal indifference | 13.29 (20.58) | 8.56 (3.98) | 8.44 (3.91) | 8.55 (3.99) | 0.67 | .503 |

**Table S1.** Sample characteristics and dropout analysis. Age (in years), friendship quality, and severity of CAs (%) are reported as *M* (*SD*). All other characteristics are reported as *n* (%). Compared to pre-pandemic baseline, sample characteristics did not significantly differ for any of the COVID-19 assessment timepoints. In addition, a dropout analysis was performed using two sample t-tests to compare characteristics between the second lockdown sample and all $n = 29$ participants that dropped out before then.

| | MFQ | RCMAS | CFQ | PSS | CTQ-SF | MOPS | APQ |
|---|---|---|---|---|---|---|---|
| **Pre-Pandemic Baseline (*N* = 102)** | 0 (0%) | 3 (2.94%) | 0 (0%) | 0 (0%) | 5 (4.90%) | 0 (0%) | 6 (5.88%) |
| **First Lockdown (*n* = 79)** | 0 (0%) | 1 (1.27%) | 1 (1.27%) | 0 (0%) | *NA* | *NA* | *NA* |
| **Reopening (*n* = 77)** | 0 (0%) | 5 (6.49%) | 0 (0%) | 0 (0%) | *NA* | *NA* | *NA* |
| **Second Lockdown (*n* = 73)** | 0 (0%) | 2 (2.74%) | 1 (1.37%) | 1 (1.37%) | *NA* | *NA* | *NA* |

**Table S2.** Missing questionnaire data. Missing questionnaire data is reported as *n* (%). MFQ = Mood and Feelings Questionnaire; RCMAS = Revised Children' s Manifest Anxiety Scale; CFQ = Cambridge Friendship Questionnaire; PSS = Perceived Stress Scale; CTQ-SF = Short-Form of the Childhood Trauma Questionnaire; MOPS = Measure of Parental Style Questionnaire; APQ = Alabama Parenting Questionnaire. NA = Questionnaire data was not assessed at this timepoint.

| | Variable | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. | 11. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. | Childhood adversity | - | | | | | | | | | | |
| 2. | Sex | -.02 | - | | | | | | | | | |
| 3. | Age (BL) | .07 | .03 | - | | | | | | | | |
| 4. | Age (L1) | .08 | .05 | .99 | - | | | | | | | |
| 5. | Age (RO) | .09 | .03 | .98 | .99 | - | | | | | | |
| 6. | Age (L2) | .08 | .03 | .99 | .99 | .98 | - | | | | | |
| 7. | Anxiety (BL) | .04 | .09 | .01 | .04 | .03 | .03 | - | | | | |
| 8. | Anxiety (L1) | .26 | .29 | .00 | .02 | .01 | .00 | .62 | - | | | |
| 9. | Anxiety (RO) | .23 | .16 | .05 | .06 | .08 | .05 | .48 | .70 | - | | |
| 10. | Anxiety (L2) | .28 | .36 | -.06 | -.04 | -.05 | -.04 | .66 | .80 | .68 | - | |
| 11. | Depression (BL) | .11 | -.03 | .03 | .07 | .06 | .06 | .85 | .52 | .42 | .58 | - |

| | Variable | 1. | 2. | 3. | 4. | 5. | 6. | 7. | 8. | 9. | 10. | 11. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12. | Depression (L1) | .28 | .19 | -.03 | .01 | .00 | -.01 | .60 | .90 | .67 | .80 | .60 |
| 13. | Depression (RO) | .24 | .08 | .02 | .03 | .04 | .02 | .43 | .71 | .91 | .69 | .46 |
| 14. | Depression (L2) | .23 | .18 | -.01 | .01 | .00 | .01 | .61 | .78 | .61 | .90 | .60 |
| 15. | Friendship quality (BL) | -.31 | .12 | .07 | .07 | .06 | .06 | -.53 | -.30 | -.27 | -.21 | -.54 |
| 16. | Friendship quality (L1) | -.35 | .29 | .14 | .15 | .13 | .13 | -.39 | -.32 | -.31 | -.26 | -.40 |
| 17. | Friendship quality (RO) | -.34 | .16 | .24 | .23 | .23 | .22 | -.45 | -.42 | -.54 | -.41 | -.46 |
| 18. | Friendship quality (L2) | -.38 | .05 | .22 | .22 | .22 | .21 | -.49 | -.44 | -.40 | -.48 | -.47 |
| 19. | Perceived stress (L1) | .22 | .38 | -.05 | -.02 | -.04 | -.05 | .53 | .77 | .54 | .62 | .32 |
| 20. | Perceived stress (RO) | .26 | .24 | -.09 | -.06 | -.07 | -.09 | .42 | .63 | .84 | .70 | .38 |
| 21. | Perceived stress (L1) | .26 | .38 | -.09 | -.06 | -.07 | -.08 | .44 | .65 | .61 | .81 | .33 |

| | Variable | 12. | 13. | 14. | 15. | 16. | 17. | 18. | 19. | 20. | 21. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 12. | Depression (L1) | - | | | | | | | | | |
| 13. | Depression (RO) | .77 | - | | | | | | | | |
| 14. | Depression (L2) | .86 | .73 | - | | | | | | | |
| 15. | Friendship quality (BL) | -.30 | -.25 | -.17 | - | | | | | | |
| 16. | Friendship quality (L1) | -.35 | -.41 | -.33 | .72 | - | | | | | |
| 17. | Friendship quality (RO) | -.44 | -.56 | -.39 | .68 | .77 | - | | | | |
| 18. | Friendship Quality (L2) | -.46 | -.44 | -.43 | .65 | .71 | .82 | - | | | |
| 19. | Perceived stress (L1) | .70 | .46 | .55 | -.31 | -.22 | -.33 | -.34 | - | | |
| 20. | Perceived stress (RO) | .67 | .82 | .63 | -.22 | -.30 | -.55 | -.46 | .61 | - | |
| 21. | Perceived stress (L2) | .70 | .62 | .76 | -.04 | -.15 | -.31 | -.34 | .62 | .70 | - |

**Table S3.** Correlations between main study variables across all assessment timepoints. BL = pre-pandemic baseline (August 2019 to March 2020; $N = 102$), L1 = first lockdown (April to May 2020; $n = 79$), RO = reopening (July to August 2020; $n = 77$), L2 = second lockdown (October to November 2020; $n = 73$).

**C. The Impact of the COVID-19 Pandemic on Mental Health Symptoms**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | |
| **1. Time** | **636.43** | **658.93** | | | |
| 2. Time + Age + Gender | 639.88 | 669.88 | 0.55 | 2 | .759 |
| **B. Anxiety Symptoms** | | | | | |
| **1. Time** | **692.16** | **714.62** | | | |
| 2. Time + Age + Gender | 694.72 | 724.66 | 1.44 | 2 | .486 |

**Table S4.** Model fit statistics for linear mixed-effects models predicting (A) depression and (B) anxiety symptoms. The best fitting models are highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| | A. Depression Symptoms | | | | |
|---|---|---|---|---|---|
| **Fixed Effects** | ***β*** | **SE** | **95% CI** | ***t*** | ***p*** |
| Intercept | -0.29 | 0.08 | [-0.46, -0.13] | -3.49 | **< .001** |
| First lockdown | 0.30 | 0.08 | [0.14, 0.45] | 3.77 | **< .001** |
| Reopening | 0.33 | 0.08 | [0.17, 0.49] | 4.15 | **< .001** |
| Second lockdown | 0.18 | 0.08 | [0.02, 0.34] | 2.27 | **.024** |
| | Marginal $R^2$ = .027; Conditional $R^2$ = . 649 | | | | |

| | B. Anxiety Symptoms | | | | |
|---|---|---|---|---|---|
| **Fixed Effects** | ***β*** | **SE** | **95% CI** | ***t*** | ***p*** |
| Intercept | -0.13 | 0.10 | [-0.32, 0.06] | -1.34 | .182 |
| First lockdown | 0.20 | 0.08 | [0.04, 0.37] | 2.41 | **.017** |
| Reopening | 0.15 | 0.09 | [-0.02, 0.32] | 1.71 | .088 |
| Second lockdown | 0.12 | 0.09 | [-0.06, 0.29] | 1.33 | .184 |
| | Marginal $R^2$ = .007; Conditional $R^2$ = .696 | | | | |

**Table S5.** Model estimates for the best fitting linear mixed-effects models predicting (A) depression and (B) anxiety symptoms. Two linear mixed-effects models predicting (A) depression and (B) anxiety symptomatology as outcomes. Assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) has been added as an independent variable. Random effects for participants have been included in both models. Pre-pandemic baseline = August 2019 to March 2020; First lockdown = April to May 2020; Reopening = July to August 2020; Second lockdown = October to November 2020. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

**D. The Impact of the COVID-19 Pandemic on Perceived Friendship Quality**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **1. Time** | **740.62** | **763.32** | | | |
| 2. Time + Age + Gender | 740.85 | 771.12 | 3.76 | 2 | .152 |

**Table S6.** Model fit statistics for linear mixed-effects models predicting perceived friendship quality. The best fitting model is highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.10 | 0.10 | [-0.29, 0.10] | -0.98 | .331 |
| First lockdown | 0.21 | 0.08 | [0.04, 0.38] | 2.47 | **.014** |
| Reopening | 0.07 | 0.09 | [-0.10, 0.23] | 0.78 | .436 |
| Second lockdown | 0.18 | 0.09 | [0.01, 0.35] | 2.08 | **.039** |
| | Marginal $R^2$ = .008; Conditional $R^2$ = .709 | | | | |

**Table S7.** Model estimates for the best fitting linear mixed-effects models predicting perceived friendship quality. One linear mixed-effects model predicting perceived friendship quality as the outcome. Assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) has been added as an independent variable. Random effects for participants have been included in both models. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

**E. The Impact of Childhood Adversity on Perceived Friendship Quality and Mental Health Symptoms Before and During the COVID-19 Pandemic**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Friendship Quality** | | | | | |
| 1. Time | 740.62 | 763.32 | | | |
| **2. Time + CA** | **737.21** | **763.70** | **5.40** | **1** | **.020** |
| 3. Time + CA + Time:CA | 742.32 | 780.15 | 0.89 | 3 | .827 |
| 4. Time + CA + Time:CA + Age + Gender | 740.91 | 786.32 | 5.41 | 2 | .067 |
| **B. Depression Symptoms** | | | | | |
| **1. Time** | **636.43** | **658.93** | | | |
| 2. Time + CA | 634.82 | 661.06 | 3.61 | 1 | .057 |
| 3. Time + CA + Time:CA | 636.43 | 673.92 | 4.39 | 3 | .223 |
| 4. Time + CA + Time:CA + Age + Gender | 639.37 | 684.37 | 1.06 | 2 | .590 |
| **C. Anxiety Symptoms** | | | | | |
| **1. Time** | 692.16 | 714.62 | | | |
| 2. Time + CA | 692.12 | 718.32 | 2.04 | 1 | .153 |
| 3. Time + CA + Time:CA | 690.86 | 728.29 | 7.26 | 3 | .064 |
| 4. Time + CA + Time:CA + Age + Gender | 693.02 | 737.94 | 1.84 | 2 | .399 |

**Table S8.** Model fit statistics for linear mixed-effects model predicting (A) perceived friendship quality, (B) depression symptoms, and (C) anxiety symptoms The best fitting model is highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion; CA = childhood adversity (cumulative).

| Fixed Effects | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.11 | 0.10 | [-0.30, 0.08] | -1.15 | .251 |
| CA | -0.42 | 0.18 | [-0.78, -0.07] | -2.36 | **.020** |
| First lockdown | 0.21 | 0.08 | [0.04, 0.38] | 2.49 | **.014** |
| Reopening | 0.07 | 0.09 | [-0.10, 0.23] | 0.78 | .436 |
| Second lockdown | 0.18 | 0.09 | [0.01, 0.35] | 2.07 | **.039** |
| | Marginal $R^2$ = .053; Conditional $R^2$ = .708 | | | | |

**Table S9.** Model estimates for the best fitting linear mixed-effects model predicting friendship quality. A linear mixed-effects model predicting friendship quality as the outcome. Childhood adversity and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. CA = childhood adversity (cumulative). $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

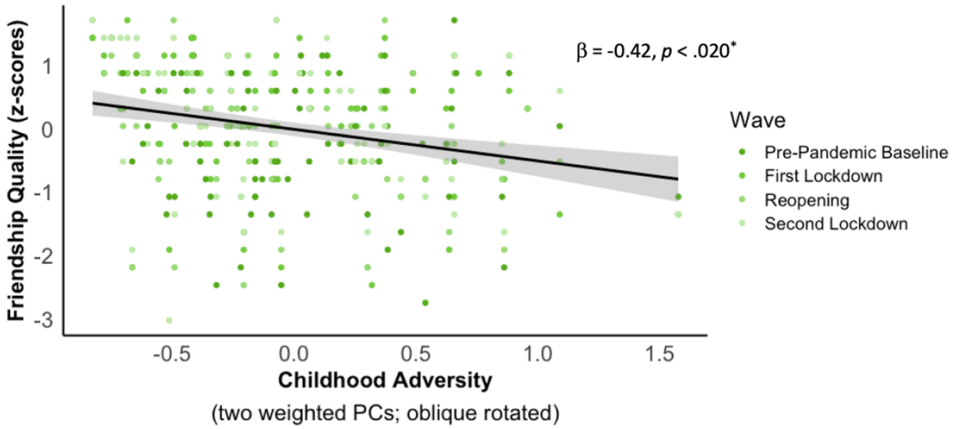



**Figure S1.** Childhood adversity effects on perceived friendship quality across all assessment timepoints. Participants with more severe CA (x-axis) self-reported lower friendship quality (y-axis) across all assessment timepoints. Index scores of CA comprise two weighted and oblique rotated principal components (PCs). Both axes represent standardized scores. The shading of individual data points represents the four different assessment timepoints. The black line shows the best-fitting linear regression line after controlling for all assessment timepoints and the shaded region represents the 95% confidence interval. $\beta$ = standardized coefficient; $^{*}p < .05$.

**Exploratory: Dimensional Effects of Childhood Adversity**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Deprivation Dimension** | | | | | |
| 1. Time | 740.62 | 763.32 | | | |
| **2. Time + Deprivation** | **738.41** | **764.90** | **4.20** | **1** | **.040** |
| 3. Time + Deprivation + Time:Deprivation | 742.86 | 780.70 | 1.55 | 3 | .671 |
| 4. Time + Deprivation + Time:Deprivation + Age + Gender | 741.59 | 786.99 | 5.28 | 2 | .071 |
| **B. Threat Dimension** | | | | | |
| **1. Time** | **740.62** | **763.32** | | | |
| 2. Time + Threat | 739.15 | 765.64 | 3.46 | 1 | .063 |
| 3. Time + Threat + Time:Threat | 745.04 | 782.88 | 0.12 | 3 | .990 |
| 4. Time + Threat + Time:Threat + Age + Gender | 744.64 | 790.05 | 4.39 | 2 | .111 |

**Table S10.** Model fit statistics for linear mixed-effects models predicting perceived friendship quality. The best fitting models are highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | $\beta$ | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.11 | 0.10 | [-0.30, 0.08] | -1.11 | .267 |
| Deprivation | -0.50 | 0.24 | [-0.97, -0.02] | -2.08 | **.040** |
| First lockdown | 0.21 | 0.09 | [0.04, 0.38] | 2.49 | **.014** |
| Reopening | 0.07 | 0.09 | [-0.10, 0.23] | 0.78 | .436 |
| Second lockdown | 0.18 | 0.09 | [0.01, 0.35] | 2.07 | **.040** |
| | | | Marginal $R^2$ = .043; Conditional $R^2$ = .708 | | |

**Table S11.** Deprivation dimension: model estimates for the best fitting linear mixed-effects model predicting perceived friendship quality A linear mixed-effects model predicting perceived friendship quality as the outcome. Deprivation experiences and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Deprivation Dimension** | | | | | |
| **1. Time** | **636.43** | **658.93** | | | |
| 2. Time + Deprivation | 636.61 | 662.85 | 1.82 | 1 | .177 |
| 3. Time + Deprivation + Time:Deprivation | 640.36 | 677.86 | 2.25 | 3 | .523 |
| 4. Time + Deprivation + Time:Deprivation + Age + Gender | 643.45 | 688.44 | 0.91 | 2 | .634 |
| **B. Threat Dimension** | | | | | |
| 1. Time | 636.43 | 658.93 | | | |
| **2. Time + Threat** | **633.90** | **660.15** | **4.53** | **1** | **.033** |
| 3. Time + Threat + Time:Threat | 633.44 | 670.94 | 6.46 | 3 | .091 |
| 4. Time + Threat + Time:Threat + Age + Gender | 636.61 | 681.60 | 0.83 | 2 | .660 |

**Table S12.** Model fit statistics for linear mixed-effects models predicting depression symptoms. The best fitting models are highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.28 | 0.08 | [-0.45, -0.12] | -3.45 | **< .001** |
| Threat | 0.76 | 0.35 | [0.06, 1.46] | 2.15 | **.034** |
| First lockdown | 0.30 | 0.08 | [0.14, 0.45] | 3.77 | **< .001** |
| Reopening | 0.33 | 0.08 | [0.17, 0.49] | 4.16 | **< .001** |
| Second lockdown | 0.18 | 0.08 | [0.02, 0.34] | 2.27 | **.024** |
| | Marginal $R^2$ = .006; Conditional $R^2$ = .651 | | | | |

**Table S13.** Threat dimension: model estimates for the best fitting linear mixed-effects model predicting depression symptoms. A linear mixed-effects model predicting depression symptoms as the outcome. Threat experiences and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Deprivation Dimension** | | | | | |
| **1. Time** | **692.16** | **714.62** | | | |
| 2. Time + Deprivation | 693.52 | 719.72 | 0.64 | 1 | .423 |
| 3. Time + Deprivation + Time:Deprivation | 694.33 | 731.76 | 5.19 | 3 | .159 |
| 4. Time + Deprivation + Time:Deprivation + Age + Gender | 696.59 | 741.51 | 1.74 | 2 | .419 |
| **B. Threat Dimension** | | | | | |
| 1. Time | 692.16 | 714.62 | | | |
| **2. Time + Threat** | **690.20** | **716.40** | **3.96** | **1** | **.047** |
| 3. Time + Threat + Time:Threat | 689.23 | 726.66 | 6.98 | 3 | .073 |
| 4. Time + Threat + Time:Threat + Age + Gender | 691.66 | 736.57 | 1.57 | 2 | .457 |

**Table S14.** Model fit statistics for linear mixed-effects models predicting anxiety symptoms. The best fitting models are highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | $\beta$ | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.12 | 0.09 | [-0.31, 0.07] | -1.27 | .205 |
| Threat | 0.84 | 0.42 | [0.01, 1.67] | 2.01 | **.048** |
| First lockdown | 0.20 | 0.08 | [0.04, 0.37] | 2.40 | **.017** |
| Reopening | 0.15 | 0.09 | [-0.02, 0.32] | 1.71 | .090 |
| Second lockdown | 0.12 | 0.09 | [-0.05, 0.29] | 1.34 | .183 |
| | Marginal $R^2$ = .039; Conditional $R^2$ = .697 | | | | |

**Table S15.** Threat dimension: model estimates for the best fitting linear mixed-effects model predicting anxiety symptoms. A linear mixed-effects model predicting anxiety symptoms as the outcome. Threat experiences and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

**F. Friendship Effects on Mental Health Symptoms Before and During the COVID-19 Pandemic**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | |
| 1. Time | 634.00 | 656.46 | | | |
| 2. Time + Friendship quality | 592.08 | 618.29 | 43.92 | 1 | < .001 |
| **3. Time + Friendship quality + Time:Friendship quality** | **589.09** | **626.52** | 9.00 | **3** | **.029** |
| 4. Time + Friendship quality + Time:Friendship quality + Age + Gender | 593.08 | 638.00 | 0.000 | 2 | .999 |
| **B. Anxiety Symptoms** | | | | | |
| 1. Time | 690.16 | 712.58 | | | |
| **2. Time + Friendship quality** | **640.07** | **666.23** | **52.09** | **1** | **< .001** |
| 3. Time + Friendship quality + Time:Friendship quality | 644.15 | 681.51 | 1.93 | 3 | .588 |
| 4. Time + Friendship quality + Time:Friendship quality + Age + Gender | 645.22 | 690.06 | 2.93 | 2 | .231 |

**Table S16.** Model fit statistics for linear mixed-effects models predicting (A) depression and (B) anxiety symptoms. The best fitting models are highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.31 | 0.07 | [-0.46, -0.16] | -4.11 | **< .001** |
| Friendship quality | -0.35 | 0.06 | [-0.48, -0.22] | -5.41 | **< .001** |
| First lockdown | 0.34 | 0.07 | [0.19, 0.48] | 4.51 | **< .001** |
| Reopening | 0.34 | 0.07 | [0.19, 0.49] | 4.55 | **< .001** |
| Second lockdown | 0.23 | 0.07 | [0.08, 0.38] | 2.99 | **.003** |
| First lockdown: Friendship quality | 0.11 | 0.08 | [-0.04, 0.26] | 1.44 | .152 |
| Reopening: Friendship quality | -0.10 | 0.08 | [-0.26, 0.06] | -1.27 | .207 |
| Second lockdown: Friendship quality | 0.09 | 0.08 | [-0.06, 0.25] | 1.18 | .238 |
| | | | Marginal $R^2$ = .189; Conditional $R^2$ = .681 | | |

**Table S17.** Model estimates for the best fitting linear mixed-effects model predicting depression symptoms. A linear mixed-effects model predicting depression symptomatology as the outcome. Perceived friendship quality and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

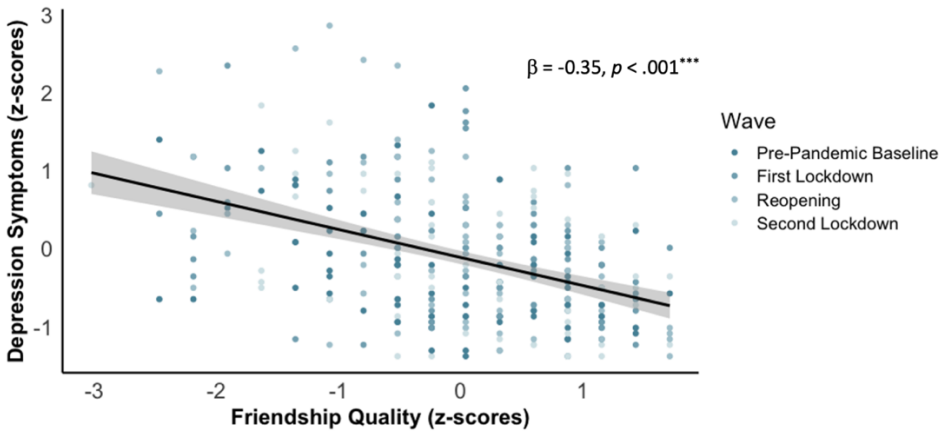



**Figure S2.** Friendship effects on depression symptoms across all assessment timepoints. Participants with higher friendship quality (x-axis) reported lower depression symptoms (y-axis) across all assessment timepoints. Both axes represent standardized scores. The shading of individual data points represents the four different assessment timepoints. The black line shows the best-fitting linear regression line after controlling for all assessment timepoints and the shaded region represents the 95% confidence interval. $\beta$ = standardized coefficient; $^{***}p < .001$.

| **Fixed Effects** | ***β*** | **SE** | **95% CI** | ***t*** | ***p*** |
|---|---|---|---|---|---|
| Intercept | -0.17 | 0.09 | [-0.34, -0.01] | -2.04 | **.043** |
| Friendship quality | -0.38 | 0.05 | [-0.48, -0.28] | -7.59 | **< .001** |
| First lockdown | 0.29 | 0.08 | [0.13, 0.44] | 3.56 | **< .001** |
| Reopening | 0.18 | 0.08 | [0.02, 0.34] | 2.26 | **.025** |
| Second lockdown | 0.18 | 0.08 | [0.02, 0.35] | 2.21 | **.028** |
| | | | Marginal $R^2$ = .178; Conditional $R^2$ = .714 | | |

**Table S18.** Model estimates for the best fitting linear mixed-effects model predicting anxiety symptoms. A linear mixed-effects model predicting anxiety symptomatology as the outcome. Friendship quality and assessment timepoint (dummy-coded: first lockdown, reopening, second lockdown, with pre-pandemic baseline as the reference group) have been added as independent variables. A random effect for participants has also been included in the model. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

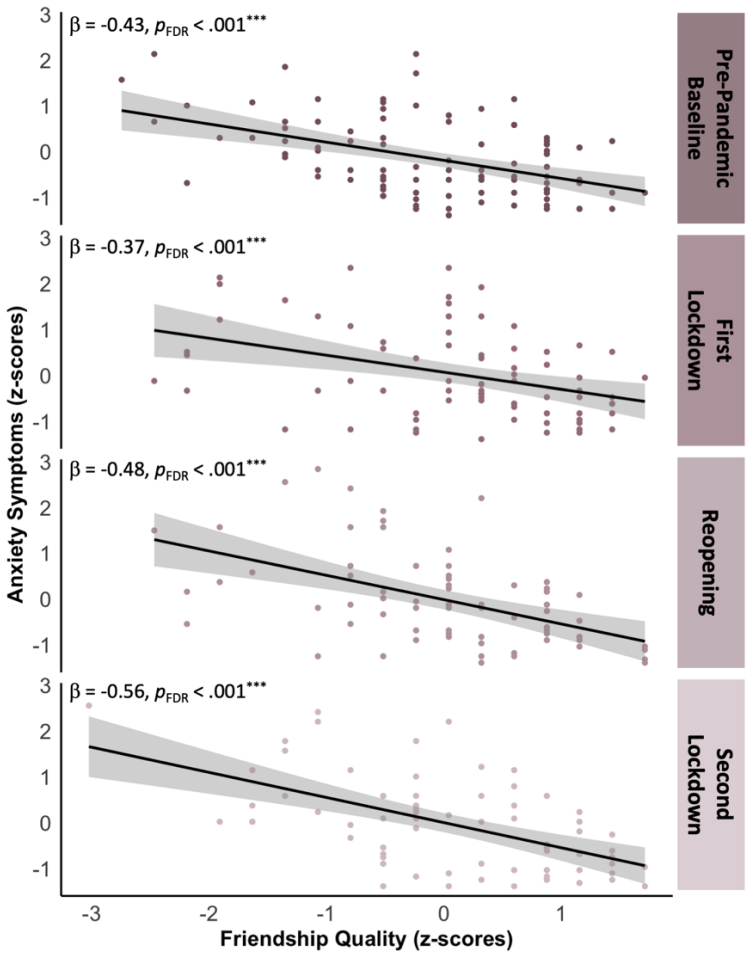



**Figure S3.** Friendship effects on anxiety symptoms before and during the COVID-19 pandemic. Participants with greater perceived friendship quality (x-axis) self-reported lower levels of anxiety symptoms (y-axis) across all assessment timepoints ($ps_{FDR}$ < .001). Both axes represent standardized score. The black lines show the best-fitting linear regression lines and the shaded regions around them represent the 95% confidence intervals. $\beta$ = standardized coefficient; $^{***}p_{FDR}$ < .001.

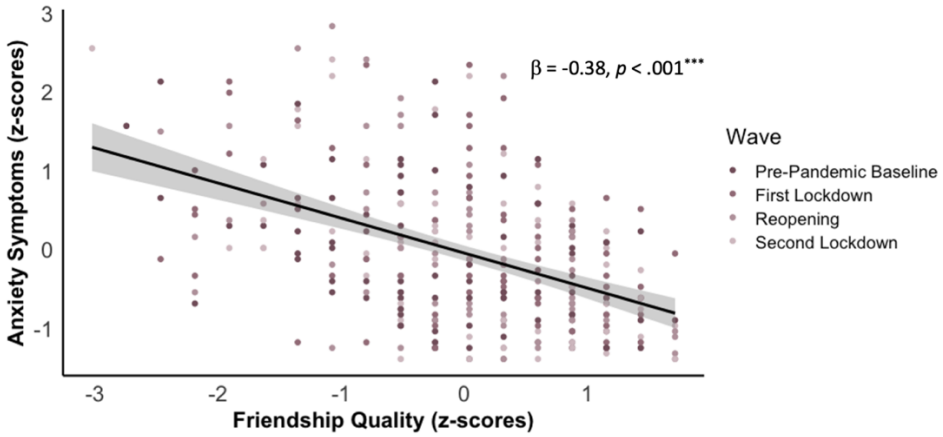



**Figure S4.** Friendship effects on anxiety symptoms across all assessment timepoints. Participants with higher friendship quality (x-axis) reported lower anxiety symptoms (y-axis) across all assessment timepoints. Both axes represent standardized scores. The shading of individual data points represents the four different assessment timepoints. The black line shows the best-fitting linear regression line after controlling for all assessment timepoints and the shaded region represents the 95% confidence interval. $\beta$ = standardized coefficient; $^{***}p <$ .001.

## G. Exploring the Interplay Between Perceived Friendship Quality and Mental Health Symptoms From Before to During the COVID-19 Pandemic

Bivariate latent change score modeling was utilized to explore the interrelationship between perceived friendship quality and mental health symptomatology from before to during the COVID-19 pandemic (see Figure G1 below). Specifically, five parameters of interest were investigated in each model. First, did perceived friendship quality reported at (A) pre-pandemic baseline, (B) first lockdown, or (C) reopening predict the degree of change in friendship quality (autoregressive parameter) and/or mental health symptoms (coupling parameter). Second, did mental health symptoms at (A) pre-pandemic baseline, (B) first lockdown, or (C) reopening predict the degree of change in mental health symptoms (autoregressive parameter) and/or friendship quality (coupling parameter)? Third, did changes in friendship quality and mental health symptoms co-occur across individuals (correlated change)?

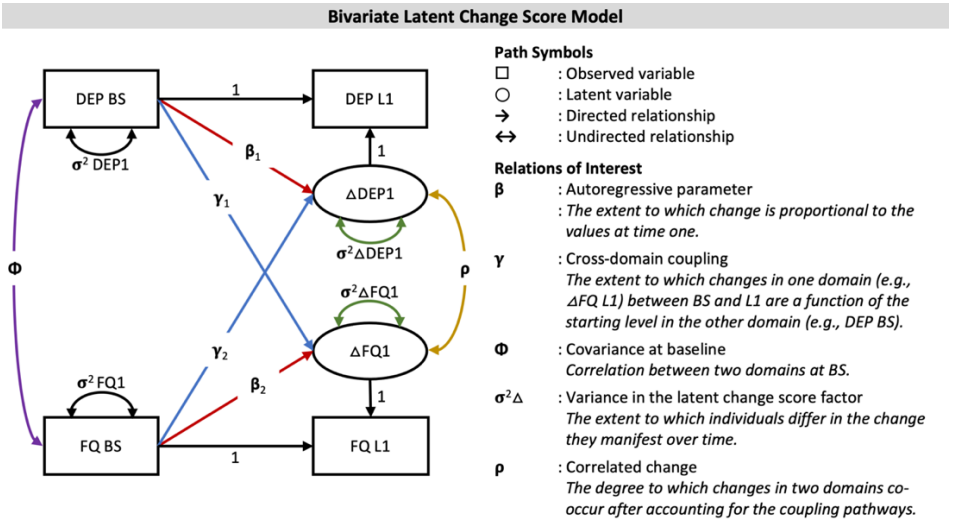



**Figure S5**. Example of a bivariate latent change score model assessing the interplay between perceived friendship quality and depression symptoms From Pre-Pandemic Baseline to First Lockdown. Means are omitted for visual clarity. This visualization and annotation is based on Kievit et al. (2018). DEP = depression symptom domain, FQ = perceived friendship quality domain, BS = pre-pandemic baseline, L1 = first lockdown.

| Domain | $\chi^2_{(6)}$ | *p* | RMSEA | CFI |
|---|---|---|---|---|
| **1. Pre-Pandemic Baseline to First Lockdown** | | | | |
| A. Depression symptoms | 114.29 | < .001 | < .001 | 1.00 |
| B. Anxiety symptoms | 121.20 | < .001 | < .001 | 1.00 |
| **2. First Lockdown to Reopening** | | | | |
| A. Depression symptoms | 144.96 | < .001 | < .001 | 1.00 |
| B. Anxiety symptoms | 138.56 | < .001 | < .001 | 1.00 |
| **3. Reopening to Second Lockdown** | | | | |
| A. Depression symptoms | 144.96 | < .001 | < .001 | 1.00 |
| B. Anxiety symptoms | 138.56 | < .001 | < .001 | 1.00 |

**Table S19.** Model fit statistics for bivariate latent change score models. Models exploring interrelationships between changes in perceived friendship quality and (A) depression and (B) anxiety symptoms from (1) pre-pandemic baseline to first lockdown, (2) first lockdown to reopening, and (3) reopening to second lockdown. RMSEA = root mean square error of approximation; CFI = comparative fit index.

| Domain | | Est | SE | *z* | *p* |
|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | |
| 1. | $DEP_{BS} \rightarrow \Delta DEP_1$ ($\beta_1$) | -0.22 | 0.13 | -1.64 | .101 |
| 2. | $DEP_{BS} \rightarrow \Delta FQ_1$ ($\gamma_1$) | 0.01 | 0.12 | 0.11 | .916 |
| 3. | $FQ_{BS} \rightarrow \Delta FQ_1$ ($\beta_2$) | -0.25 | 0.09 | -2.86 | **.004** |
| 4. | $FQ_{BS} \rightarrow \Delta DEP_1$ ($\gamma_2$) | 0.06 | 0.10 | 0.66 | .512 |
| 5. | $DEP_{BS} \leftrightarrow FQ_{BS}$ ($\phi$) | -0.33 | 0.08 | -4.15 | **< .001** |
| 6. | $\Delta DEP_1 \leftrightarrow \Delta FQ_1$ ($\rho$) | -0.15 | 0.06 | -2.72 | **.007** |
| 7. | $\sigma^2 DEP_1$ | 0.55 | 0.08 | 6.55 | **< .001** |
| 8. | $\sigma^2 FQ_1$ | 0.93 | 0.12 | 7.79 | **< .001** |
| 9. | $\sigma^2 \Delta DEP_1$ | 0.52 | 0.09 | 6.09 | **< .001** |
| 10. | $\sigma^2 \Delta FQ_1$ | 0.50 | 0.10 | 5.11 | **< .001** |
| **B. Anxiety Symptoms** | | | | | |
| 1. | $ANX_{BS} \rightarrow \Delta ANX_1$ ($\beta_1$) | -0.33 | 0.10 | -3.13 | **.002** |
| 2. | $ANX_{BS} \rightarrow \Delta FQ_1$ ($\gamma_1$) | 0.01 | 0.10 | 0.11 | .913 |
| 3. | $FQ_{BS} \rightarrow \Delta FQ_1$ ($\beta_2$) | -0.29 | 0.09 | -3.29 | **.001** |
| 4. | $FQ_{BS} \rightarrow \Delta ANX_1$ ($\gamma_2$) | -0.04 | 0.11 | -0.35 | .730 |
| 5. | $ANX_{BS} \leftrightarrow FQ_{BS}$ ($\phi$) | -0.43 | 0.10 | -4.24 | **< .001** |
| 6. | $\Delta ANX_1 \leftrightarrow \Delta FQ_1$ ($\rho$) | -0.11 | 0.05 | -2.19 | **.028** |
| 7. | $\sigma^2 ANX_1$ | 0.85 | 0.12 | 7.37 | **< .001** |
| 8. | $\sigma^2 FQ_1$ | 1.00 | 0.13 | 7.66 | **< .001** |
| 9. | $\sigma^2 \Delta ANX_1$ | 0.51 | 0.11 | 4.88 | **< .001** |
| 10. | $\sigma^2 \Delta FQ_1$ | 0.50 | 0.10 | 5.13 | **< .001** |

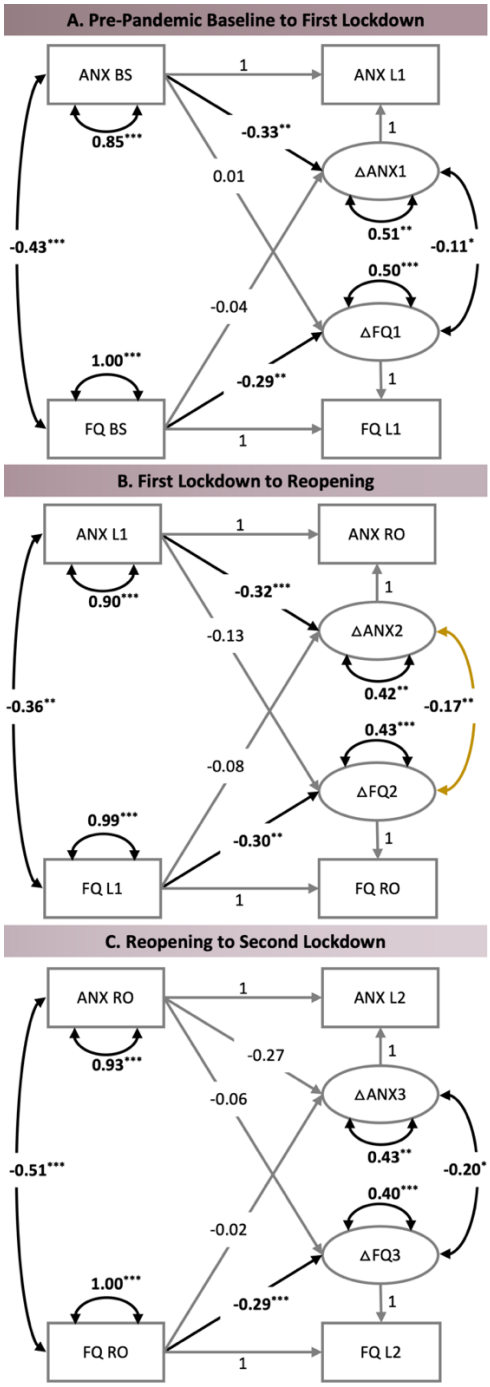
**Table S20.** Bivariate latent change score model output assessing the interplay between perceived friendship quality and (A) depression and (B) anxiety symptoms From Pre-Pandemic Baseline to First Lockdown. Est = standardized parameter estimates. FQ = perceived friendship quality domain, DEP = depression symptom domain, ANX = anxiety symptom domain, BS = pre-pandemic baseline. $\beta$ = autoregressive parameter, $\gamma$ = cross-domain coupling, $\phi$ = covariance at pre-pandemic baseline, $\rho$ = correlated change, $\sigma^2\Delta$ = variance in the latent change score, $\rightarrow$ = directed relationship, $\leftrightarrow$ = undirected relationship. Bold denotes significant effects.

| Domain | | Est | SE | *z* | *p* |
|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | |
| 1. | $DEP_{L1} \rightarrow \Delta DEP_1$ ($\beta_1$) | -0.33 | 0.09 | -3.57 | **< .001** |
| 2. | $DEP_{L1} \rightarrow \Delta FQ_1$ ($\gamma_1$) | -0.17 | 0.08 | -2.08 | **.037** |
| 3. | $FQ_{L1} \rightarrow \Delta FQ_1$ ($\beta_2$) | -0.31 | 0.10 | -3.08 | **.002** |
| 4. | $FQ_{L1} \rightarrow \Delta DEP_1$ ($\gamma_2$) | -0.16 | 0.07 | -2.30 | **.022** |
| 5. | $DEP_{L1} \leftrightarrow FQ_{L1}$ ($\phi$) | -0.31 | 0.10 | -2.99 | **.003** |
| 6. | $\Delta DEP_1 \leftrightarrow \Delta FQ_1$ ($\rho$) | -0.13 | 0.04 | -2.99 | **.003** |
| 7. | $\sigma^2 DEP_1$ | 0.81 | 0.13 | 6.11 | **< .001** |
| 8. | $\sigma^2 FQ_1$ | 1.00 | 0.17 | 5.88 | **< .001** |
| 9. | $\sigma^2 \Delta DEP_1$ | 0.32 | 0.11 | 2.92 | **.003** |
| 10. | $\sigma^2 \Delta FQ_1$ | 0.44 | 0.09 | 4.84 | **< .001** |
| **B. Anxiety Symptoms** | | | | | |
| 1. | $ANX_{L1} \rightarrow \Delta ANX_1$ ($\beta_1$) | -0.32 | 0.10 | -3.24 | **.001** |
| 2. | $ANX_{L1} \rightarrow \Delta FQ_1$ ($\gamma_1$) | -0.13 | 0.08 | -1.54 | .123 |
| 3. | $FQ_{L1} \rightarrow \Delta FQ_1$ ($\beta_2$) | -0.30 | 0.10 | -2.92 | **.003** |
| 4. | $FQ_{L1} \rightarrow \Delta ANX_1$ ($\gamma_2$) | -0.08 | 0.08 | -0.92 | .360 |
| 5. | $ANX_{L1} \leftrightarrow FQ_{L1}$ ($\phi$) | -0.36 | 0.11 | -3.39 | **.001** |
| 6. | $\Delta ANX_1 \leftrightarrow \Delta FQ_1$ ($\rho$) | -0.17 | 0.06 | -2.89 | **.004** |
| 7. | $\sigma^2 ANX_1$ | 0.90 | 0.13 | 6.99 | **< .001** |
| 8. | $\sigma^2 FQ_1$ | 0.99 | 0.17 | 4.97 | **< .001** |
| 9. | $\sigma^2 \Delta ANX_1$ | 0.42 | 0.13 | 3.23 | **.001** |
| 10. | $\sigma^2 \Delta FQ_1$ | 0.43 | 0.09 | 4.97 | **< .001** |

**Table S21.** Bivariate latent change score model output assessing the interplay between perceived friendship quality and (A) depression and (B) anxiety symptoms from first lockdown to reopening. Est = standardized parameter estimates. FQ = perceived friendship quality domain, DEP = depression symptom domain, ANX = anxiety symptom domain, L1 = first lockdown. $\beta$ = autoregressive parameter, $\gamma$ = cross-domain coupling, $\phi$ = covariance at first lockdown, $\rho$ = correlated change, $\sigma^2\Delta$ = variance in the latent change score, $\rightarrow$ = directed relationship, $\leftrightarrow$ = undirected relationship. Bold denotes significant effects.

| Domain | | Est | SE | *z* | *p* |
|---|---|---|---|---|---|
| **A. Depression Symptoms** | | | | | |
| 1. | $DEP_{RO} \rightarrow \Delta DEP_2$ ($\beta_1$) | -0.38 | 0.13 | -2.97 | **.003** |
| 2. | $DEP_{RO} \rightarrow \Delta FQ_2$ ($\gamma_1$) | 0.03 | 0.10 | 0.30 | .764 |
| 3. | $FQ_{RO} \rightarrow \Delta FQ_2$ ($\beta_2$) | -0.25 | 0.06 | -3.87 | **< .001** |
| 4. | $FQ_{RO} \rightarrow \Delta DEP_2$ ($\gamma_2$) | -0.03 | 0.07 | -0.41 | .682 |
| 5. | $DEP_{RO} \leftrightarrow FQ_{RO}$ ($\phi$) | -0.48 | 0.11 | -4.31 | **< .001** |
| 6. | $\Delta DEP_2 \leftrightarrow \Delta FQ_2$ ($\rho$) | -0.08 | 0.06 | -1.48 | .140 |
| 7. | $\sigma^2 DEP_2$ | 0.78 | 0.15 | 5.39 | **< .001** |
| 8. | $\sigma^2 FQ_2$ | 1.02 | 0.17 | 6.16 | **< .001** |
| 9. | $\sigma^2 \Delta DEP_2$ | 0.31 | 0.08 | 3.93 | **< .001** |
| 10. | $\sigma^2 \Delta FQ_2$ | 0.41 | 0.11 | 3.68 | **< .001** |
| **B. Anxiety Symptoms** | | | | | |
| 1. | $ANX_{RO} \rightarrow \Delta ANX_2$ ($\beta_1$) | -0.27 | 0.14 | -1.89 | .059 |
| 2. | $ANX_{RO} \rightarrow \Delta FQ_2$ ($\gamma_1$) | -0.06 | 0.12 | -0.47 | .642 |
| 3. | $FQ_{RO} \rightarrow \Delta FQ_2$ ($\beta_2$) | -0.29 | 0.07 | -4.09 | **< .001** |
| 4. | $FQ_{RO} \rightarrow \Delta ANX_2$ ($\gamma_2$) | -0.02 | 0.07 | -0.29 | .770 |
| 5. | $ANX_{RO} \leftrightarrow FQ_{RO}$ ($\phi$) | -0.51 | 0.12 | -4.35 | **< .001** |
| 6. | $\Delta ANX_2 \leftrightarrow \Delta FQ_2$ ($\rho$) | -0.20 | 0.09 | -2.35 | **.019** |
| 7. | $\sigma^2 ANX_2$ | 0.93 | 0.17 | 5.50 | **< .001** |
| 8. | $\sigma^2 FQ_2$ | 1.00 | 0.16 | 6.22 | **< .001** |
| 9. | $\sigma^2 \Delta ANX_2$ | 0.43 | 0.13 | 3.39 | **.001** |
| 10. | $\sigma^2 \Delta FQ_2$ | 0.40 | 0.10 | 3.94 | **< .001** |

**Table S22.** Bivariate latent change score model output assessing the interplay between perceived friendship quality and (A) depression and (B) anxiety symptoms from reopening to second lockdown. Est = standardized parameter estimates. FQ = perceived friendship quality domain, DEP = depression symptom domain, ANX = anxiety symptom domain, RO = reopening. $\beta$ = autoregressive parameter, $\gamma$ = cross-domain coupling, $\phi$ = covariance at reopening, $\rho$ = correlated change, $\sigma^2\Delta$ = variance in the latent change score, $\rightarrow$ = directed relationship, $\leftrightarrow$ = undirected relationship. Bold denotes significant effects.



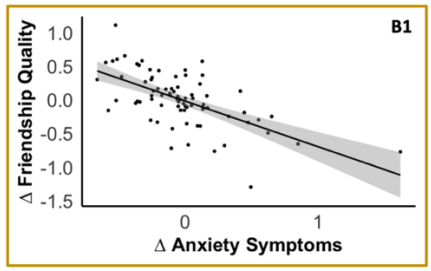




**Figure S6**. The interplay between perceived friendship quality and anxiety symptoms from before to during the COVID-19 pandemic. Each path shows standardized parameter estimates. FQ = friendship quality domain, ANX =

anxiety symptom domain, BS = pre-pandemic baseline, L1 = first lockdown, RO = reopening, L2 = second lockdown. Δ = latent change score, → = directed relationship, ↔ = undirected relationship. Path in black denote significant effects. B1 = Correlation between change in friendship quality and change in anxiety symptoms from the first lockdown to reopening. $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

**H. Exploring Perceived Stress as a Potential Mechanism Linking Perceived Friendship Quality with Mental Health Symptoms**

| Model | AIC | BIC | $\chi^2$ | *df* | *p* |
|---|---|---|---|---|---|
| 1. Time | 546.55 | 563.63 | | | |
| **2. Time + Age + Gender** | **541.80** | **565.71** | **8.75** | **2** | **.013** |

**Table S23.** Model fit statistics for linear mixed-effects models predicting perceived stress. The best fitting model is highlighted in bold. Random effects for participants have been included in all models. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Fixed Effects | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| Intercept | -0.39 | 0.17 | [-0.74, -0.05] | -2.28 | **.025** |
| Reopening | -0.07 | 0.09 | [-0.25, 0.12] | -0.73 | .467 |
| Second lockdown | -0.03 | 0.10 | [-0.23, 0.16] | -0.35 | .727 |
| Age | -0.04 | 0.09 | [-0.22, 0.15] | -0.43 | .667 |
| Gender | 0.61 | 0.20 | [0.21, 1.00] | 3.03 | **.003** |
| | Marginal $R^2$ = .083; Conditional $R^2$ = .656 | | | | |

**Table S24.** Model estimates for the best fitting linear mixed-effects models predicting perceived stress. One linear mixed-effects model predicting perceived stress as the outcome. Assessment timepoint (dummy-coded: reopening, second lockdown, with first lockdown as the reference group) has been added as an independent variable and age and gender identity have been added as covariates. Random effects for participants have also been included. $\beta$ = standardized coefficient; 95% CI = 95% confidence interval. Bold denotes significant effects.

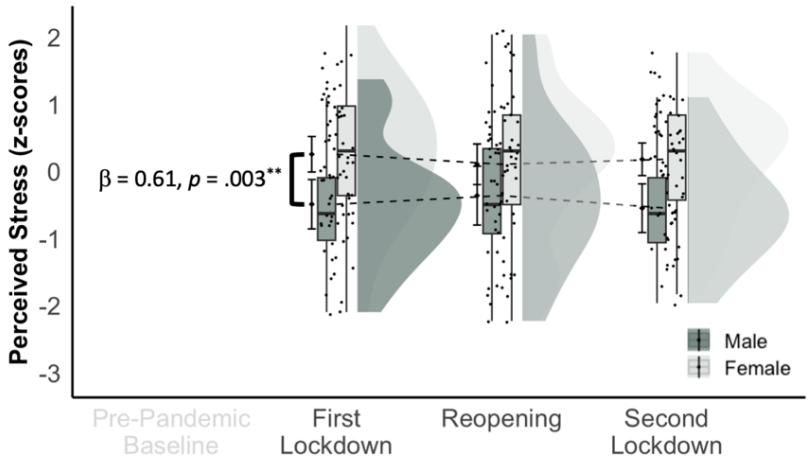



**Figure S7.** Perceived stress during the COVID-19 pandemic. Compared to the first lockdown, participants did not self-report changes in perceived stress. However, participants who identified as female reported significantly elevated levels of perceived stress across all assessment timepoints during the COVID-19 pandemic, compared to participants who identified as male ($p$ = .003). This raincloud plot displays standardized perceived stress scores (y-axis) across all assessment timepoints during the COVID-19 pandemic (x-axis). To emphasize the main effect of time, we first plotted the mean and 95% confidence intervals for each assessment timepoint and connected these with a dashed line. Second, we added box plots showing the median (solid vertical line) and interquartile range. The black dots represent individual raw datapoints. Third, we added violin plots to visualize the probability distribution. $^{**}p < .01$.

| | $\beta$ | SE | 95% CI | $z$ | $p$ |
|---|---|---|---|---|---|
| a path | -0.31 | 0.10 | [-0.50, -0.10] | -2.97 | **.003** |
| b path | 0.42 | 0.09 | [0.25, 0.59] | 4.87 | **< .001** |
| direct effect (c') | -0.14 | 0.10 | [-0.34, 0.06] | -1.37 | .172 |
| indirect effect (ab) | -0.13 | 0.05 | [-0.25, -0.05] | -2.57 | **.010** |

**Table S25.** Depression symptoms: parameter estimates mediation model. $\beta$ = standardized coefficient; 95% CI = 95% bootstrapped confidence interval. Bold denotes significant effects.

| | *β* | SE | 95% CI | *z* | *p* |
|---|---|---|---|---|---|
| a path | -0.36 | 0.10 | [-0.55, -0.14] | -3.43 | **< .001** |
| b path | 0.51 | 0.08 | [0.34, 0.67] | 6.15 | **< .001** |
| direct effect (c') | -0.14 | 0.09 | [-0.31, 0.05] | -1.54 | .123 |
| indirect effect (ab) | -0.18 | 0.06 | [-0.32, -0.08] | -2.99 | **.003** |

**Table S26.** Anxiety symptoms: parameter estimates mediation model. *β* = standardized coefficient; 95% CI = 95% bootstrapped confidence interval. Bold denotes significant effects.

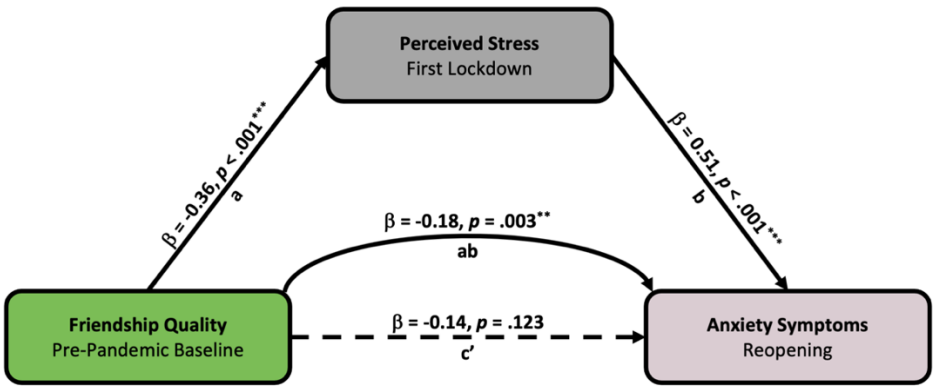



**Figure S8**. Perceived Stress Mediates the Relationship between Perceived Friendship Quality and Anxiety Symptoms. Path a shows the standardized regression coefficient of the relationship between perceived friendship quality during the pre-pandemic baseline and perceived stress during the first lockdown. Path b shows the standardized regression coefficient of the relationship between perceived stress during the first lockdown and anxiety symptoms during reopening, while controlling for gender identity. Paths ab (indirect effect) and c' (direct effect) show the standardized regression coefficient of the relation between friendship quality during the pre-pandemic baseline and anxiety symptoms during reopening without and while controlling for perceived stress during the first lockdown, respectively. Pre-pandemic baseline = August 2019 to March 2020 ($N$ = 100 after outlier removal); First lockdown = April to May 2020 ($n$ = 77 after outlier removal); Reopening = July to August 2020 ($n$ = 70 after outlier removal). $\beta$ = standardized coefficient. Dashed line denotes non-significant effect. $^{**}p < .01$, $^{***}p < .001$.

Please note that the mediation effects remained consistent when assessing depression or anxiety symptoms during the second lockdown. In other words, perceived stress during the first lockdown continued to fully mediate the relationship between pre-pandemic friendship quality and depression symptoms (indirect effect: $\beta$ = -0.14, SE = 0.05, 95% CI [-0.26, -0.05], $p$ = .009) or anxiety

symptoms (indirect effect: $\beta$ = -0.18, SE = 0.07, 95% CI [-0.34, -0.07], $p$ = .009) during the second lockdown.

**I. Monte Carlo Power Analyses**

Two post-hoc simulation-based power analyses were performed. First, we used the mixedpower R package (version 0.1.0; Kumle et al. (2021)) to estimate power in our linear mixed-effects model examining the main effect of friendship quality on depression symptoms before and during the COVID-19 pandemic (Marginal $R^2$ = .178; Conditional $R^2$ = .668). The following specifications were used to estimate power: fixed effect = 1 (main predictor: perceived friendship quality), simvar = subID (random effect for participants), steps = c(50, 60, 70) (sample sizes we estimated power for), critical_value = 2 (significance threshold for coefficients; $\alpha$ = .05), n_sim = 1000 (number of single simulations used to estimate power). Results of these Monte Carlo simulations indicated that a sample size of $N$ = 70 corresponds to more than 80% power for the main effect. Hence, our smallest available sample size (second lockdown with $n$ = 70 participants after outlier removal) is sufficient to ensure adequate power.

Second, to estimate sample size and power for our sequential mediation model, we followed the recommendations by Schoemann et al. (2017) and ran Monte Carlo simulations via the Shiny App (available at https://schoemanna.shinyapps.io/mc_power_med/; developed by Schoemann et al. (2017)). Standardized model parameters have been estimated based on the current dataset. Specifically, we set a = -0.32, b = 0.58, and c' = -0.32. Moreover, we set $N$ = 73 (reopening sample size after outlier removal), specified the total number of replications (# of Replications = 1,000), the number of draws for computing Monte Carlo confidence intervals (Monte Carlo Draws per Rep = 20,000), a random seed to ensure the exact replicability of the results, and the confidence levels of 95%. This analysis revealed that a sample of $N$ = 73 participants results in 80% power for the indirect effect (ab path).

**J. Exploratory: Psychosocial Experiences during the COVID-19 Pandemic**

Four items from the *COVID-19 Adolescent Symptom and Psychological Experience Questionnaire* (CASPE; Ladouceur (2020)) were selected to explore self-reported psychosocial experiences at each follow-up assessment timepoint. First, participants were asked about (A) "what event or change has been the most positive" and (B) "[…] most negative" (Figure J1). Second, participants were asked about "[…] how [they] stay connected with friends (Figure J2). Third, participants were asked about "how [they] are coping or dealing with the stress or anxiety related to the COVID-19 outbreak (Figure J3). Although utilized in previous studies (e.g., Porter et al. (2021)), the CASPE has not yet been validated.

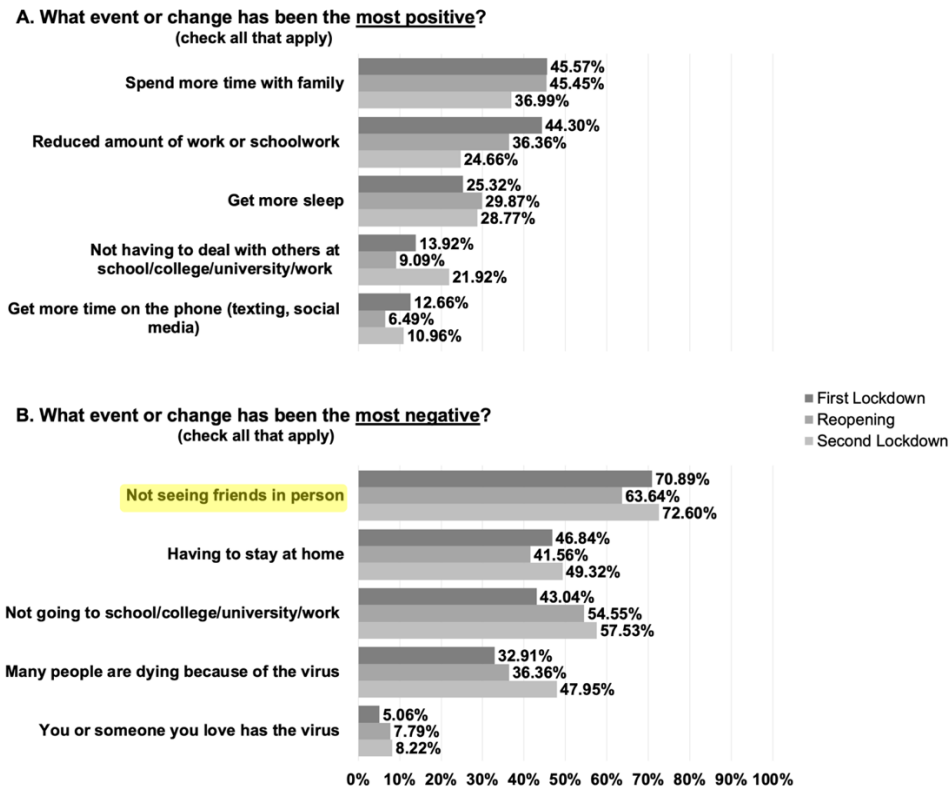



**Figure S9.** Notable (A) positive and (B) negative events or changes during the COVID-19 pandemic. At each assessment timepoint during the COVID-19 pandemic, participants reported (**A**) the most positive and (**B**) the most negative events or changes during the COVID-19 pandemic.

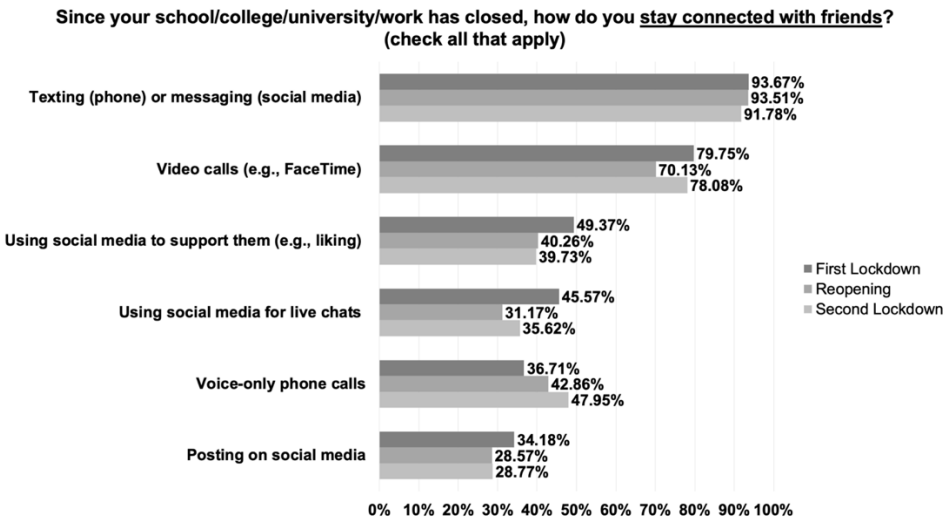



**Figure S10.** Tools used to maintain social connections with friends during the COVID-19 pandemic. At each assessment timepoint during the COVID-19 pandemic, participants reported all approaches they used to stay connected with friends.

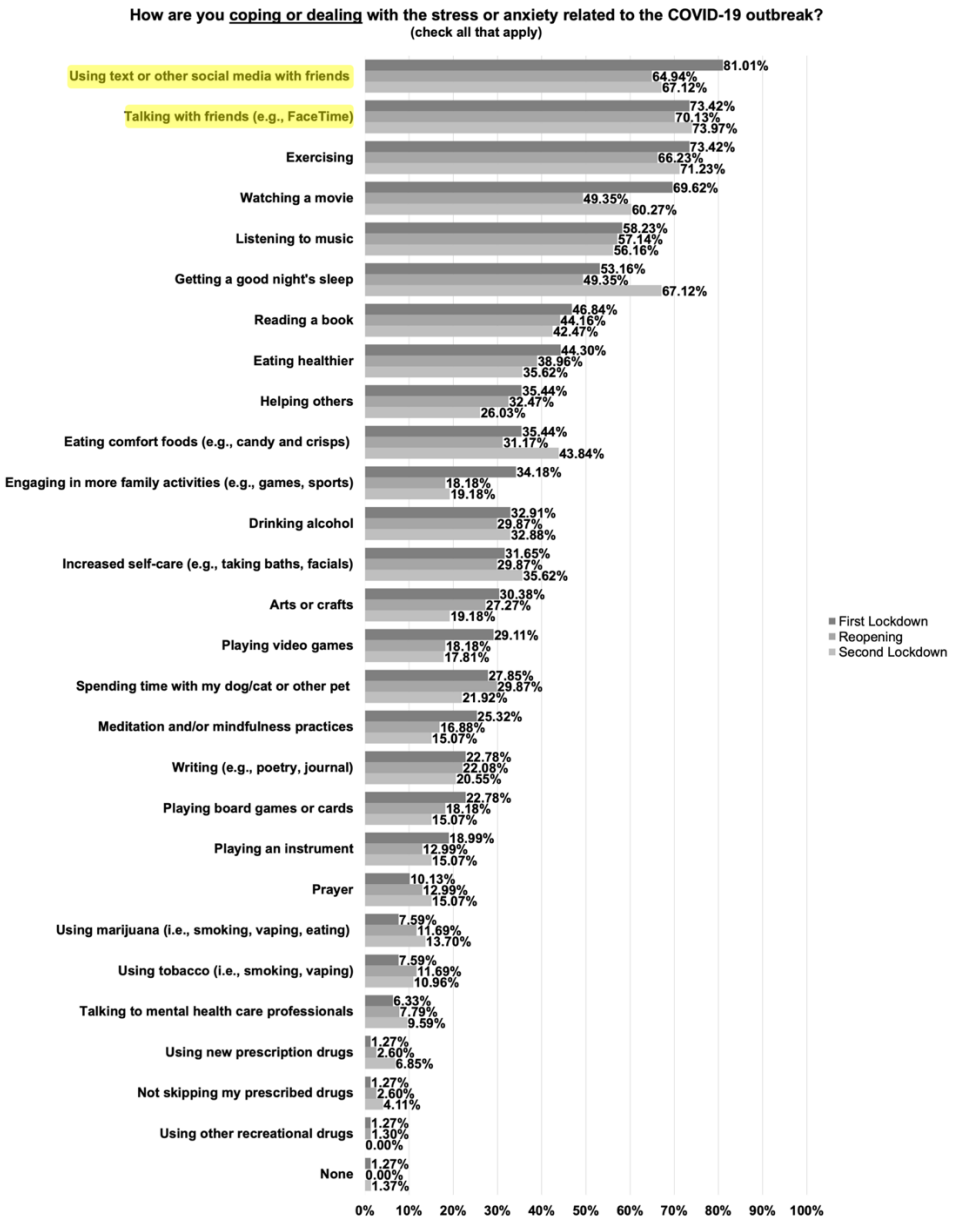



**Figure S11.** Coping strategies for dealing with pandemic-related stress or anxiety. At each assessment timepoint during the COVID-19 pandemic, participants reported approaches to deal with pandemic-related stress or anxiety.

# Chapter 6

## Friendship Buffering and Autobiographical Memory Specificity in Young People with Childhood Adversity

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Data and code available on *OSF*

**Abstract**

**Background:** Young people with childhood adversity (CA) tend to show altered autobiographical memory processing, such as reduced access to details of specific positive events, which may represent a potential mechanism through which CA increases mental health risk. Although friendship support is known to improve mental health outcomes in this population, the mechanisms underlying this protective relationship remain largely unknown.

**Objective:** This study aimed to investigate associations between perceived friendship support, valence-specific autobiographical memory recall, perceived stress, and depressive symptoms in young people with CA.

**Participants and Setting:** The study included 100 young people (aged 18-24 years) with low to moderate levels of CA, recruited from the general population across the Netherlands.

**Methods:** Hierarchical multiple regressions were conducted to examine relationships between friendship support, positive and negative autobiographical memory specificity, perceived stress, and depressive symptoms.

**Results:** Friendship support was not linked to positive or negative autobiographical memory specificity. However, it was associated with lower perceived stress ($\beta$ = -0.44, $p$ < .001) and fewer depressive symptoms ($\beta$ = -0.21, $p$ = .041). Autobiographical memory specificity showed no relationship with perceived stress or depressive symptoms. Furthermore, more severe CA ($\beta$ = 0.21, $p$ = .002) and higher perceived stress ($\beta$ = 0.56, $p$ < .001) were both associated with more depressive symptoms.

**Conclusions:** These findings point towards a model where friendship support exerts its protective mental health effects possibly through reducing perceived stress in young people with CA, rather than through influencing the specificity of positive or negative autobiographical memories.

*Keywords*: autobiographical memory specificity, friendship stress buffering, depressive symptoms, young people, childhood adversity

**Highlights**

- Young people with childhood adversity recalled autobiographical memories.
- Memory specificity was not associated with psychosocial functioning.
- More friendship support linked to less perceived stress and depressive symptoms.

## Introduction

Approximately half of all young people growing up worldwide are exposed to at least one form of childhood adversity (CA) (Bellis et al., 2014; McLaughlin, 2016). This includes often co-occurring experiences such as abuse or neglect, parental mental illness, bullying, growing up in severe poverty, or exposure to war (Brown et al., 2019; Dong et al., 2004). Chronic and repeated exposure to these toxic stressors requires young people to adapt their psychological, social, and cognitive functioning, and the strategies they employ may increase the risk for later-life mental health problems. Indeed, a large-scale epidemiological survey with more than 51,000 adults across 21 countries estimated that around one-third of all mental disorders worldwide are attributable to CA exposure (Kessler et al., 2010). Although these associations are well-documented, the mechanisms linking CA exposure to mental health problems as well as the protective factors that can mitigate these effects remain less understood. Hence, a better mechanistic understanding could provide crucial insights for developing targeted and effective prevention and intervention efforts for young people with CA.

Altered autobiographical memory processing following CA may be one potential pathway leading to mental health problems (McCrory et al., 2017). Autobiographical memory gradually develops from early childhood through young adulthood and is defined as the system that integrates specific personal experiences as well as perspectives, interpretations, and evaluations from both oneself and others to scaffold an overarching life narrative (Fivush, 2011). Young people with CA tend to show alterations in autobiographical memory processing, such as cognitive biases favoring negative memories, diminished richness of positive memories, and reduced specificity (i.e., generalized recall of single events) (Dalgleish & Werner-Seidler, 2014; McCrory et al., 2017; Puetz et al., 2021; Valentino et al., 2009). For example, when asked to complete the widely used Autobiographical Memory Task (AMT; J. M. Williams & Broadbent (1986)), in which the goal is to generate specific memories in response to differentially valenced cue words, maltreated adolescents (aged 10-14 years) showed reduced autobiographical memory specificity and increased amygdala activation in response to recalling negative compared to positive memories (McCrory et al., 2017). This tendency to recall memories in a generalized manner rather than as specific, detailed experiences may hold functional value in an adverse environment. For instance, by avoiding detailed recollections of traumatic or distressing events, individuals can shield themselves from overwhelmingly intense negative emotions often associated with such memories (J. M. Williams, 2006). At the same time, reduced autobiographical memory specificity is related to patterns of repetitive, negative thinking (rumination) which can increase vulnerability to mental health problems, particularly as individuals transition to a less adverse environment, such as school (McCrory & Viding, 2015; Valentino et al., 2009). Indeed, adversity-related alterations in autobiographical memory have

been linked to the onset, maintenance, and recurrence of depression (Dalgleish & Werner-Seidler, 2014; Hallford et al., 2022; McCrory et al., 2017; Valentino et al., 2009).

The neurocognitive social transactional model of psychiatric vulnerability (McCrory et al., 2022) argues that adversity-related cognitive alterations, such as reduced access to memories of specific events, may inadvertently generate more stressful experiences with peers (stress generation) or lead to an attenuation in the number and quality of friendships (social thinning), thus exacerbating mental health challenges. For example, reduced autobiographical memory specificity can affect social functioning by limiting the richness of autobiographical experiences available to navigate interpersonal challenges (Goddard et al., 1996), such as resolving conflicts with friends. In turn, this could perpetuate stress generation by prolonging conflicts and contribute to social thinning by jeopardizing the stability of friendships. Indeed, maltreated adolescents (aged 10-14 years at baseline) were found to exhibit reduced autobiographical memory specificity, aggregated across both positive and negative memories, which was associated with reduced prosocial behavior rated by the parent two years later (Puetz et al., 2021). Furthermore, among trauma-exposed adults with posttraumatic stress disorder (PTSD), Sutherland & Bryant (2008) observed a higher prevalence of reduced autobiographical memory specificity compared to adults without PTSD and found that in both groups memory recall was less specific for positive compared to negative events. In the same study, reduced autobiographical memory specificity, for both positive and negative memories, was strongly associated with deficits in real-life problem-solving abilities, particularly in resolving interpersonal challenges like friendship issues (Sutherland & Bryant, 2008). The existing evidence therefore indicates that, reduced autobiographical memory specificity seems to play a key role in disrupted social functioning and concurrently the maintenance of mental health problems in individuals with CA. It is therefore imperative to establish if and how autobiographical memory processing is associated with protective factors like friendship support to ultimately improve mental health outcomes in young people with CA.

Friendship support is a well-established protective factor that significantly enhances mental health in young people with CA (König et al., 2023, 2025; van Harmelen et al., 2016, 2017, 2021). Safe, stable, and reciprocal friendships become increasingly important during adolescence, a period that begins with puberty and ends with adult independence (Burnett Heyes et al., 2015; Crone & Dahl, 2012; Güroğlu, 2022). This critical developmental stage is marked by a heightened sensitivity to and need for peer interactions (Blakemore & Mills, 2014; Orben et al., 2020) as well as an increased vulnerability to the onset of mental health problems (Orben et al., 2022; Paus et al., 2008; Solmi et al., 2022). Therefore, friendship support may be particularly important for young people

with CA, given their elevated risk for mental health challenges. Indeed, greater perceived friendship quality was found to be associated with fewer depressive symptoms (König et al., 2025; van Harmelen et al., 2016) and increased adaptive mental health functioning (König et al., 2023; van Harmelen et al., 2017, 2021) in young people with CA. However, the mechanisms linking friendship support to mental health in young people with CA are largely unknown (e.g., Raposa et al., (2015); Scheuplein & van Harmelen (2022)).

Several studies highlight the interconnectedness between friendship support, valence-specific autobiographical memory recall, stress responsivity, and mental health in young people with and without CA. One potential pathway through which friendship support may exert its protective mental health effects is by influencing the specificity of positive autobiographical memories (Barry et al., 2019; Kensinger et al., 2016). This may subsequently reduce perceptions, reactions, and physiological responses to and after stress (Gunnar, 2017; R. M. Sullivan & Perry, 2015), thereby lowering the physiological burden of stress exposure and improving mental health functioning (Gotlib et al., 2020; Hammen et al., 2000; Hennessy et al., 2009; König et al., 2023). In line with this model, social interactions with friends may aid retrieval of emotionally salient memories, such as positive experiences (Güroğlu et al., 2008). Additionally, recalling positive autobiographical memories was found to lower cortisol levels and reduce negative affect following acute stress exposure in US college students (Speer & Delgado, 2017). In young people (aged 14 years) with CA, Askelund et al. (2019) found that more specific positive autobiographical memories were associated with lower morning cortisol and fewer negative self-cognitions during low mood over the course of one year. In the same study, positive memory specificity was related to fewer depressive symptoms mediated through fewer negative self-cognitions in response to recent stressful life events. A recent longitudinal study of young people (aged 16-26 years) with CA found that high-quality friendship support reduced perceived stress and subsequent depressive symptoms in response to the COVID-19 pandemic (König et al., 2025). In young people (aged 15-17 years) without CA, Barry et al. (2019) reported moderate associations between greater levels of perceived social support from friends and romantic partners and increased autobiographical memory specificity of both positive and negative events. Additionally, greater social support was positively associated with mental health functioning. However, no direct link between autobiographical memory specificity and mental health functioning was observed (Barry et al., 2019).

Building on this body of research, the current study examined whether greater perceived friendship support is associated with greater specificity of positive autobiographical memories in young people with CA as well as with lower perceived stress and fewer depressive symptoms. Hence, we analyzed cross-sectional data from the first 100 participants of the ongoing Towards Health and

Resilience in Volatile Environments (THRIVE) study. The THRIVE study is a longitudinal investigation of risk and protective factors affecting mental health in young people (aged 18-24 years) with retrospectively self-reported CA. To assess autobiographical memory specificity, we adapted the AMT and instructed participants to recall friendship memories evoked by four positive and four negative cue words. Specifically, we examined whether greater friendship support was associated with greater specificity of positive autobiographical friendship memories (hypothesis 1.1; Barry et al. (2019)), lower levels of perceived stress (hypothesis 1.2; König et al. (2025)), and fewer depressive symptoms (hypothesis 1.3; van Harmelen et al. (2016)). Next, we examined whether greater specificity of positive memories was associated with lower levels of perceived stress (hypothesis 2.1; Speer & Delgado (2017)) and fewer depressive symptoms (hypothesis 2.2; Askelund et al. (2019)). Finally, we aimed to replicate the association between lower levels of perceived stress and fewer depressive symptoms (hypothesis 3; Gotlib et al., (2020); Hammen et al., (2000); König et al., (2025)). To account for potential valence-specific effects, we analyzed associations for both positive and negative autobiographical friendship memories.

## Methods

### *Towards Health and Resilience in Volatile Environments (THRIVE) Study*

Cross-sectional data from the first 100 participants were drawn from the THRIVE study (see Table 1 for sample characteristics). This subset was selected based on the availability of complete assessments by the project deadline in June 2024. It was deemed sufficient for conducting robust preliminary statistical analyses and was chosen to provide initial insights while data collection for the full sample is ongoing. A post-hoc power analysis using G*Power 3.1 (Faul et al., 2007) further confirmed that the sample of $N = 100$ participants has 84% power to detect small to moderate main effects ($f^2 = .09$ at $\alpha = .05$, two-sided), which is consistent with effect sizes reported in related research (Puetz et al., 2021). The THRIVE study is an ongoing longitudinal study at Leiden University, the Netherlands, with a target sample size of 250 young people aged between 18 to 24 years with a retrospectively self-reported history of CA. CA was conceptualized as exposure to any adverse life event experienced within or outside the family environment before the age of 18. Participants were recruited across the Netherlands from the general population through flyer distribution at schools and universities, general practitioners' practices, shops, libraries, hospitals, out-patient care facilities, and social media. Individuals were eligible to participate if they were aged between 18 to 24 years, able to speak, write, and understand Dutch, and self-reported CA experiences before the age of 18. Due to the potentially stressful nature of the study protocol, individuals who had experienced severe depressive symptoms or suicidal thoughts within the past two weeks prior to the eligibility screening were excluded. Specifically, participants with a score above 14 on the 9-item Patient

Health Questionnaire (PHQ; Kroenke et al. (2001)) or a score greater than zero on question nine of the PHQ ("Thoughts that you would be better off dead, or of hurting yourself") were not included in the study. Eligibility criteria were assessed via telephone by a trained member of the study team. The THRIVE study received ethical approval from the Medical Ethics Committee Leiden The Hague Delft (NL80017.058.21) in July 2022 and commenced in October 2022.

| **Characteristics** | |
|---|---|
| **Age** | 21.23 (1.84) |
| **Gender identity** | |
| Male | 20% |
| Female | 79% |
| Non-binary | 1% |
| **Ethnic orientation** | |
| Asian | 3% |
| Black, African, or Caribbean | 2% |
| White | 84% |
| Other | 11% |
| **Highest education** | |
| HAVO (11 years of education) | 11% |
| VWO (12 years of education) | 54% |
| MBO (14 years of education) | 4% |
| HBO (15 years of education) | 7% |
| WO bachelor (17 years of education) | 19% |
| WO master (17+ years of education) | 4% |
| Other | 1% |
| **Maltreatment experiences** | |
| Childhood Trauma Questionnaire- Short Form (CTQ-SF) | |
| Sexual abuse | 5.12 (2.66) |
| Physical abuse | 6.62 (3.69) |
| Emotional abuse | 11.08 (5.62) |
| Physical neglect | 7.43 (3.12) |
| Emotional neglect | 11.39 (4.36) |
| **Friendship support** | |
| Multidimensional Scale of Perceived Social Support (MSPSS) | 23.69 (3.83) |
| McGill Friendship Questionnaire – Friendship Functions (MFQ-FF) | |
| Stimulating companionship | 35.94 (4.38) |
| Help | 33.54 (4.81) |
| Intimacy | 35.13 (5.07) |
| Reliable alliance | 37.87 (2.75) |

| | |
|---|---|
| Self-validation | 34.47 (5.28) |
| Emotional security | 35.17 (4.33) |
| **Perceived stress** | 15.95 (5.24) |
| **Depressive symptoms** | |
| Mood and Feelings Questionnaire (MFQ) | 10.62 (8.36) |
| Patient Health Questionnaire (PHQ-9) | 4.87 (3.61) |

**Table 1.** Sample characteristics (*N* = 100). Age is reported in years *M* (*SD*). Gender identity, ethnic orientation, and highest education are reported as %. Key features of the Dutch education system have been summarized by the (European Commission, 2024). Self-reported severity levels of maltreatment experiences, perceived friendship support, perceived stress, and depressive symptoms are presented as raw measurement characteristics *M (SD)*. Based on established cut-off scores for the original English Childhood Trauma Questionnaire (CTQ; Bernstein et al. (1994)), this sample can be characterized reporting low to moderate severity levels of maltreatment experiences. Specifically, the following cut-off scores can be applied to each scale: sexual abuse (low to moderate: 4-7), physical abuse (low to moderate: 5-9), emotional abuse (low to moderate: 5-12), physical neglect (low to moderate: 5-9), emotional neglect (low to moderate: 5-14). Please note that this study utilized the 24-item Dutch CTQ-SF with a 4-item sexual abuse subscale for which no published cut-off scores are currently available (Thombs et al., 2009).

### *Procedure*

This study utilized self-report data from the first 100 participants who completed the initial two assessment timepoints (on average 31 days apart) of the ongoing THRIVE longitudinal study. The measures relevant to the current study are described below. At each assessment timepoint, informed consent was obtained from participants, who were informed that they could withdraw from the study at any time without having to provide a reason and without any consequences.

During the first assessment timepoint (T1), eligible participants received a secure online link via email to remotely complete self-report questionnaires about past maltreatment experiences and currently perceived friendship support. These domains were assessed using the Dutch Childhood Trauma Questionnaire Short-Form (CTQ-SF; Thombs et al. (2009)), the Cambridge Friendship Questionnaire (CFQ; van Harmelen et al. (2017)), the McGill Friendship Questionnaire – Friendship Functions (MFQ-FF; Mendelson & Aboud (1999)), and the Multidimensional Scale of Perceived Social Support (MSPSS; Zimet et al. (1990)).

For the second assessment timepoint (T2), participants visited the Leiden University Medical Center in the Netherlands, on average one month after the completion of T1. During T2, participants provided saliva samples, mood ratings, and self-reports. These self-reports covered, among other measures, currently

perceived stress and depressive symptoms, using the Perceived Stress Scale (PSS; Sheldon Cohen et al. (1983)), the Mood and Feelings Questionnaire (MFQ; Angold & Costello (1987)), and the 9-item Patient Health Questionnaire (PHQ-9; Kroenke et al. (2001)). In addition, participants underwent magnetic resonance imaging (MRI) and completed a range of cognitive tasks both inside and outside the MRI environment. An adapted version of the Autobiographical Memory Task (AMT; J. M. Williams & Broadbent (1986)) was administered before scanning to assess autobiographical friendship memory processing. All T2 self-reports analyzed as part of this study were assessed after scanning.

Participants received €15 for the completion of T1 (approximately 48 minutes) and €70 for the completion of T2 (approximately 4 hours), adding up to a total of €85. This study was conducted in accordance with the principles of the Declaration of Helsinki (World Medical Association, 2013), the Medical Research Involving Human Subjects Act (WMO; The Central Committee on Research Involving Human Subjects (2018)), and the Leiden University code of ethics for research in the social and behavioral sciences involving human participants (Leiden University, 2018).

### *Measures*

*Maltreatment Experiences*

The *Dutch Childhood Trauma Questionnaire-Short Form* (CTQ-SF; Bernstein et al. (1994); Thombs et al. (2009)) was administered remotely at T1 to retrospectively assess self-reported maltreatment experiences within the family environment before the age of 18. Participants rated items such as "I believe that I was physically abused" on a 5-point Likert scale (1 = never true, 5 = very often true). The Dutch CTQ-SF consists of 24-items comprising five subscales (sexual, physical, emotional abuse and physical and emotional neglect), which were combined to calculate a standardized total severity *z*-score. Specifically, to compute this cumulative maltreatment index (higher index indicating more severe maltreatment experiences), mean imputations were performed to replace two missing responses, and positive items were reverse coded. Compared to the original English CTQ-SF (Bernstein et al., 2003), the Dutch CTQ-SF (Thombs et al., 2009) removed the item "I believe I was molested" due to translation ambiguity of the word molested. Internal consistency was excellent for the total scale (Cronbach's $\alpha = .94$) and acceptable to excellent for the five subscales (sexual abuse: $\alpha = .89$; physical abuse: $\alpha = .89$; emotional abuse: $\alpha = .91$; physical neglect: $\alpha = .69$; emotional neglect: $\alpha = .87$). To assess potential underreporting of maltreatment experiences, the CTQ-SF also includes a 3-item minimization/denial (MD)-scale. Participants who responded to MD-items such as "I had the perfect childhood" with "very often true" (a rating of 5 on the 5-point Likert scale) would be scored as 1. MD-scale ratings below 5 would be scored as 0. A total MD-score of 3 is thought to indicate strong underreporting of

maltreatment experiences. The prevalence of MD was 9% in our sample (MD total scores: 0 = 91%, 1 = 7%, 2 = 1%, 3 = 1%), which is lower compared to endorsements reported in both community and clinical samples (MacDonald et al., 2015, 2016).

*Friendship Support*

Currently perceived friendship support was assessed at T1 using three self-report questionnaires. The *Cambridge Friendship Questionnaire* (CFQ; van Harmelen et al. (2017)) was administered to assess the self-reported number, availability, and quality of current friendships. Participants rated items such as "Do you feel that your friends understand you?". Negative items were reverse coded so that higher scores indicate greater perceived friendship support. Internal consistency for the total scale was poor (Cronbach's $\alpha$ = .53), which led to its exclusion from all subsequent analyses.

The *McGill Friendship Questionnaire – Friendship Functions* (MFQ-FF; Mendelson & Aboud (1999)) was used to assess friendship support provided by a specific, self-selected friend. Participants rated items such as "[Name of friend] would make me feel better if I were worried" on a 9-point Likert scale (0 = never, 8 = always). The MFQ-FF consists of 30-items comprising six subscales (stimulating companionship, help, intimacy, reliable alliance, self-validation, emotional security), which can be combined to calculate a total friendship functioning score. Higher scores indicate greater perceived friendship support. Internal consistency was excellent for the total scale (Cronbach's $\alpha$ = .95) as well as acceptable to good for the six subscales (stimulating companionship: Cronbach's $\alpha$ = .79; help: Cronbach's $\alpha$ = .75; intimacy: Cronbach's $\alpha$ = .87; reliable alliance: Cronbach's $\alpha$ = .83; self-validation: Cronbach's $\alpha$ = .86; emotional security: Cronbach's $\alpha$ = .84).

The *Multidimensional Scale of Perceived Social Support* (MSPSS; Zimet et al. (1990)) was used to assess perceived social support from family, friends, and significant others. Specifically, this study only utilized the 4-items assessing perceived friendship support. Participants rated items such as "I can count on my friends when things go wrong" on a 7-point Likert scale (1 = very strongly disagree, 7 = very strongly agree) with higher scores indicating greater perceived friendship support. Internal consistency for the friendship subscale was excellent (Cronbach's $\alpha$ = .90).

To compute a single friendship support index (higher index indicating greater perceived friendship support), the standardized total *z*-scores of the MFQ-FF and MSPSS were averaged.

*Depressive Symptoms*
Current depressive symptoms (i.e., during the past two weeks) were assessed at T2 using two self-report questionnaires. The *Mood and Feelings Questionnaire* (MFQ; Angold & Costello (1987)) consists of 31-items such as "I felt miserable or unhappy", which were rated on a 3-point Likert scale (0 = not true, 3 = true). Higher scores indicate greater depressive symptoms. Internal consistency for the total scale was excellent (Cronbach's $\alpha$ = .90).

The *Patient Health Questionnaire* (PHQ-9; Kroenke et al. (2001)) consists of 9-items such as "Feeling down, depressed, or hopeless", which were rated on a 4-point Likert scale (0 = not at all, 3 = nearly every day). Higher scores indicate greater depressive symptoms. Internal consistency for the total scale was acceptable (Cronbach's $\alpha$ = .76).

To compute a single depressive symptoms index (higher index indicating greater depressive symptoms), the standardized total *z*-scores of the MFQ and PHQ-9 were averaged.

*Perceived Stress*
The *Perceived Stress Scale* (PSS; Sheldon Cohen et al. (1983)) was administered at T2 to assess levels of perceived stress during the past month. Participants rated 10 items such as "In the last month, how often have you felt nervous and stressed?" on a 5-point Likert scale (0 = never, 4 = very often). Positive items were reverse coded so that higher standardized total *z*-scores indicate greater levels of perceived stress during the past month. Internal consistency for the total scale was acceptable (Cronbach's $\alpha$ = .74).

*Autobiographical Friendship Memories*
The *Autobiographical Memory Task* (AMT; J. M. Williams & Broadbent (1986)) was adapted in a written, computerized format to assess specificity of autobiographical friendship memories. At T2, participants were asked to recall a memory of a situation or experience with a friend that a presented cue word reminded them of. Four positive and four negative Dutch cue words were presented in the following fixed order: gelukkig (happy), boos (angry), leuk (nice), jaloers (jealous), grappig (funny), gekwetst (hurt), gezellig (cozy), eenzaam (lonely). Additionally, participants were instructed to write about different memories in relation to each cue word and were informed that their friendship memories could be formed recently (e.g., last week) or years ago. While participants were instructed to recall real memories, there was no emphasize on a memory having to be specific. This minimal instruction approach has proven effective in enhancing the task's sensitivity to detect reduced memory specificity in non-clinical samples (Debeer et al., 2009). In response to each cue word,

participants were given two minutes to provide a friendship memory after which the task advanced automatically.

Two independent raters (RQ and EV), both native Dutch speakers, each scored a total of 800 responses based on a stringent scoring procedure, resulting in strong interrater reliability (kappa = .77). Any disagreements were resolved through discussion. A memory was scored as *specific* if it referred to a single event that has happened within a period of 24h, at a particular time and place. A memory was scored as *non-specific* if it pertained to a single event that unfolded over the course of more than 24h, at a particular time and place (extended); If it referred to a situation or experience that cannot be linked to a single event (categoric); If no response was provided or if a statement/general remark was given (omission); Or, if the response referred to an event previously reported (repeated). For each valence, the proportion of specific memories was calculated by dividing the number of specific memories by the total number of cue words. A higher proportion indicates a more specific recall of friendship memories for that valence. Results remained the same when the number of omissions was subtracted, as per Debeer et al. (2009) and Hitchcock et al. (2019). These confirmatory analyses are reported in the supplementary materials (Section 1). For ease of interpretation, all subsequent findings are reported without excluding the number of omissions. Wilcoxon signed-rank tests indicated no significant valence differences for specific autobiographical friendship memories, V = 1670.50, $p$ = .112, or generalized memories (extended and categoric combined), V = 1126, $p$ = .767. However, the number of omissions was significantly higher for negative cue words ($M$ = 0.16, $SD$ = 0.44) compared to positive cues ($M$ = 0.04, $SD$ = 0.20), V = 13, $p$ = .008. Descriptive statistics of AMT responses are presented in Table 2.

| | Cue Words | Specific | Extended | Categoric | Omission | Repeated |
|---|---|---|---|---|---|---|
| Positive | Happy | 69 (69.0) | 17 (17.0) | 14 (14.0) | 0 (0) | 0 (0) |
| | Nice | 67 (67.0) | 9 (9.0) | 23 (23.0) | 1 (1.0) | 0 (0) |
| | Funny | 81 (81.0) | 1 (1.0) | 15 (15.0) | 3 (3.0) | 0 (0) |
| | Cozy | 75 (75.0) | 4 (4.0) | 21 (21.0) | 0 (0) | 0 (0) |
| | ***M* (*SD*)** | **73 (6.32)** | **7.75 (6.99)** | **18.25 (4.43)** | **1 (1.41)** | **0 (0)** |
| Negative | Angry | 78 (78.0) | 6 (6.0) | 13 (13.0) | 3 (3.0) | 0 (0) |
| | Jealous | 54 (54.0) | 11 (11.0) | 31 (31.0) | 3 (3.0) | 1 (1.0) |
| | Hurt | 76 (76.0) | 5 (5.0) | 13 (13.0) | 2 (2.0) | 4 (4.0) |
| | Lonely | 62 (62.0) | 9 (9.0) | 20 (20.0) | 8 (8.0) | 1 (1.0) |
| | ***M* (*SD*)** | **67.5 (11.47)** | **7.75 (2.75)** | **19.25 (8.50)** | **4 (2.71)** | **1.5 (1.73)** |

**Table 2.** Percentage of responses by memory type in the autobiographical friendship memory task ($N = 100$). Characteristics are reported as *n* (%). Cue words were presented in Dutch in the following fixed order: gelukkig (happy), boos (angry), leuk (nice), jaloers (jealous), grappig (funny), gekwetst (hurt), gezellig (cozy), eenzaam (lonely). The number of omissions was significantly higher for negative cue words compared to positive cues ($p = .008$). No other significant valence differences were observed.

**Statistical Analysis**

All analyses were performed in R (version 4.3.0; R Core Team (2022)). Two outliers were detected using the Rosner's test (rosnerTest function of the EnvStats R package, version 2.7.0; Millard (2013)) in combination with the 3-sigma method (mean +/- three standard deviations). One outlier reported severe

maltreatment experiences, and one outlier reported severe depressive symptoms. Both outliers were excluded from subsequent analyses, resulting in a final sample size of $N = 98$. All analyses were conducted on standardized $z$-scores. Due to non-normality of residuals amongst our primary regression models (supplementary Table S2), we conducted robust hierarchical multiple regressions using Huber weights. The robust hierarchical multiple regressions approach was chosen to clarify the incremental contribution of the covariates, including maltreatment experiences, age, and gender identity. In step 1, the friendship support index, the autobiographical friendship memory specificity index, or the perceived stress index were entered to assess their direct effects on the outcome variable. In step 2, the cumulative maltreatment index, age, and gender identity were added to determine their additional predictive value. Models were compared using the Akaike Information Criteria (AIC; Akaike (1974)), with lower values indicating better model fit. Main effects of the best fitting models were inspected using two-sided robust Wald tests (model_parameters function of the parameters R package, version 0.21.7; Lüdecke et al. (2020)). Significance was set at $p < .05$ throughout all analyses and partial Cohen's $f$-squared ($f_p^2$) effect size estimates are reported for all relevant tests (cohens_f_squared function of the effectsize R package, version 0.8.8; Ben-Shachar et al. (2020). Model specifications and model fit indices are provided in the supplementary materials (Section 3) alongside Spearman's rank correlations with 95% bootstrap confidence intervals (Section 4). To perform these analyses, we used the cor_mat function of the rstatix R package (version 0.7.2; Kassambara (2023)), the corci function of the bootcorci R package (version 0.0.0.9000; Rousselet et al. (2019)), and the rlm function of the MASS R package (version 7.3.58.4; Venables & Ripley (2002)). Associations for both positive and negative autobiographical friendship memories were analyzed to account for potential valence specific effects and the false discovery rate (FDR) correction method (Benjamini & Hochberg, 1995) was used to correct for multiple comparisons. Mean imputation to replace two missing CTQ-SF values were performed using the mice R package (version 3.16.0; van Buuren & Groothuis-Oudshoorn (2011)).

## Results

### *Associations of Friendship Support with Autobiographical Friendship Memory Specificity, Perceived Stress, and Depressive Symptoms*

First, we examined whether greater friendship support was associated with greater specificity of positive autobiographical friendship memories (hypothesis 1.1). Contrary to our predictions, we observed no association between perceived levels of friendship support and specificity of either positive ($\beta = 0.15$, $p = .183$) or negative ($\beta = 0.02$, $p = .869$) autobiographical friendship memories. The inclusion of covariates (i.e., maltreatment experiences, age, and gender identity) did not improve model fit (Tables S3.1.2 and S3.1.4). Next, we examined whether

greater friendship support was associated with lower levels of perceived stress (hypothesis 1.2). In line with our predictions, greater levels of perceived friendship support were moderately associated with lower levels of perceived stress ($\beta$ = -0.44, SE = 0.12, 95% CI [-0.68, -0.21], $t_{96}$ = -3.74, $f_p^2$ = 0.16, $p$ < .001). The inclusion of covariates did not improve model fit (Table S3.2.2). Next, we examined whether greater friendship support was associated with fewer depressive symptoms (hypothesis 1.3). In line with our predictions, greater levels of perceived friendship support were weakly to moderately associated with fewer depressive symptoms ($\beta$ = -0.21, SE = 0.10, 95% CI [-0.42, -0.01], $t_{93}$ = -2.07, $f_p^2$ = 0.07, $p$ = .041). The inclusion of covariates improved model fit, revealing a small to moderate association between more severe maltreatment experiences and greater levels of depressive symptoms ($\beta$ = 0.25, SE = 0.09, 95% CI [0.07, 0.44], $t_{93}$ = 2.72, $f_p^2$ = 0.08, $p$ = .008) (Table S3.3.2).

***Associations of Autobiographical Friendship Memory Specificity with Perceived Stress and Depressive Symptoms***

Second, we examined whether specificity of positive autobiographical friendship memories was associated with lower levels of perceived stress (hypothesis 2.1). Contrary to our predictions, we observed no association between specificity and perceived stress, for neither positive ($\beta$ = 0.18, $p$ = .102) or negative ($\beta$ = 0.20, $p$ = .069) autobiographical friendship memories. The inclusion of covariates did not improve model fit (Tables S3.4.2 and S3.4.4). Next, we examined whether specificity of positive autobiographical friendship memories was associated with fewer depressive symptoms (hypothesis 2.2). Contrary to our predictions, specificity of positive autobiographical friendship memories was not associated with depressive symptoms ($\beta$ = 0.12, $p$ = .157). Greater specificity of negative autobiographical friendship memories was weakly associated with more depressive symptoms ($\beta$ = 0.17, SE = 0.08, 95% CI [0.01, 0.34], $t_{93}$ = 2.07, $f_p^2$ = 0.07, $p$ = .041), but this effect did not survive correction for multiple comparisons ($p_{FDR}$ = .082). For both models, the inclusion of covariates improved model fit, revealing a small to moderate association between more severe maltreatment experiences and greater levels of depressive symptoms (positive memory specificity: $\beta$ = 0.34, SE = 0.09, 95% CI [0.16, 0.51], $t_{93}$ = 3.87, $f_p^2$ = 0.12, $p_{FDR}$ < .001; and negative memory specificity: $\beta$ = 0.30, SE = 0.09, 95% CI [0.13, 0.48], $t_{93}$ = 3.44, $f_p^2$ = 0.11, $p_{FDR}$ < .001) (Tables 3.5.2 and 3.5.4).

***Association between Perceived Stress and Depressive Symptoms***

Finally, we examined whether lower levels of perceived stress were associated with fewer depressive symptoms (hypothesis 3). In line with our predictions, we observed a strong association between greater levels of perceived stress and more depressive symptoms ($\beta$ = 0.56, SE = 0.06, 95% CI [0.43, 0.68], $t_{93}$ = 8.87, $f_p^2$ = 0.77, $p$ < .001). The inclusion of covariates improved model fit and confirmed the previously reported moderate association between more severe maltreatment

experiences and more depressive symptoms ($\beta$ = 0.21, SE = 0.07, 95% CI [0.08, 0.35], $t_{93}$ = 3.20, $f_p^2$ = 0.15, $p$ = .002) (Table S3.6.2).

**Discussion**

This study examined whether greater perceived friendship support is associated with greater specificity of positive autobiographical friendship memories, lower perceived stress, and fewer depressive symptoms in 100 young people (aged 18-24 years) with low to moderate CA. In line with previous research, we found that more severe CA is associated with higher levels of depressive symptoms (K. Hughes et al., 2017) and that greater perceived friendship support is associated with both lower levels of perceived stress (König et al., 2025) and fewer depressive symptoms (van Harmelen et al., 2016). However, friendship support was not associated with the specificity of either positive or negative autobiographical friendship memories. Furthermore, we found no evidence that memory specificity for positive or negative events was related to perceived stress. We found only weak support that greater specificity of negative, but not positive, autobiographical friendship memories were associated with more depressive symptoms, but the effect did not survive correction for multiple comparisons. Lastly, in keeping with prior research, we found that greater levels of perceived stress are associated with more depressive symptoms (Gotlib et al., 2020; König et al., 2025). As such, our findings point towards a model where friendship support exerts its protective mental health effects possibly through reducing perceived stress in young people with CA, rather than through influencing the specificity of positive or negative autobiographical friendship memories. However, the cross-sectional nature of our data limits our ability to further investigate this potential stress-buffering pathway.

Contrary to previous findings, positive autobiographical memory specificity was not associated with lower perceived stress (Speer & Delgado, 2017), fewer depressive symptoms (Askelund et al., 2019), or greater friendship support (Barry et al., 2019). Sample and methodological differences may explain this discrepancy, as both Speer & Delgado (2017) and Barry et al. (2019) studied young people without CA and used different approaches to investigate key variables such as stress and social support. Speer & Delgado (2017) demonstrated that the active retrieval of specific positive autobiographical memories was an effective strategy to reduce psychological and physiological responses to acute stress, while Barry et al. (2019) showed that less specific autobiographical memory recall at baseline predicted reduced social support from friends and romantic partners both at baseline and after one year. In contrast, the current study assessed self-reported perceived stress over the past four weeks, rather than inducing acute stress using the socially evaluative cold pressor task (Schwabe et al., 2008; Speer & Delgado, 2017), and focused specifically on friendship support, rather than a combination of support from friends and romantic partners (Barry et al., 2019). Furthermore,

our study recruited a comparatively high-functioning, community sample of young people who, on average, retrospectively self-reported low to moderate CA, mild levels of depressive symptoms, and strong friendship support. Even low to moderate CA exposure may add additional layers of complexity to the association between autobiographical memory specificity and psychosocial functioning due to causing functional alterations across a range of neurocognitive systems (McCrory et al., 2022). For example, longitudinal data from Askelund et al. (2019) showed that positive autobiographical memory specificity was only indirectly associated with fewer depressive symptoms via reducing negative self-cognitions in response to recent stressful life events. Additionally, Puetz et al. (2021) reported that in a small sample of maltreated young people (aged 11-14 years), reduced autobiographical memory specificity predicted reduced prosocial behavior but not depressive symptoms, despite numerous studies establishing reduced autobiographical memory specificity as a cognitive marker of depression (Hallford et al., 2022; Liu et al., 2013). Thus, the relationship between adversity-related alterations in autobiographical memory specificity and psychosocial functioning appears to be complex, likely unfolds over time, and may involve intermediary factors, such as self-cognitions.

Having said that, we did not observe associations between the severity of CA and autobiographical memory specificity, despite previous research reporting reduced autobiographical memory specificity in maltreated young people (Barry, Lenaert, et al., 2018; McCrory et al., 2017; Valentino et al., 2009). However, several studies were unable to demonstrate consistent associations between trauma exposure and lower autobiographical memory specificity and instead suggest that lower specificity may be a function of comorbid affective disorders, such as depression, over and above any CA effects (Kuyken et al., 2006; Moore & Zoellner, 2007; J. M. G. Williams et al., 2007). For example, Kuyken et al. (2006) showed that young people with major depressive disorder (MDD) and no reported history of trauma produced less specific autobiographical memories during the Autobiographical Memory Task (J. M. Williams & Broadbent, 1986) compared to both never-depressed young people with no history of trauma and young people with MDD and a history of trauma. This suggests that reduced autobiographical memory specificity may develop through factors other than CA, for example through deficits in executive functioning (Dalgleish et al., 2007).

Next, we found that young people with CA who were more specific in recalling negative autobiographical friendship memories also self-reported greater depressive symptoms. While this valence-specific effect did not survive correction for multiple comparisons, it is worth noting that this uncorrected finding aligns with previous research. Negatively biased autobiographical memory processing, with faster access and a greater tendency to generate negative memories, is a defining feature of affective disorders such as depression (Dalgleish & Werner-

Seidler, 2014; Gotlib & Joormann, 2010). For instance, in a community sample of adult women (aged 31-41 years) with sexual abuse experiences, Burnside et al. (2004) found that those with major depression disorder were more specific in recalling negative autobiographical memories compared to those without the disorder. Additionally, neuroimaging studies involving clinically depressed adults (aged 18-55 years; K. D. Young et al. (2017)) and maltreated adolescents (aged 10-14 years; McCrory et al. (2017)) have both shown that recalling specific negative, compared to positive, autobiographical memories elicits stronger activation in areas of the brain implicated in salience processing. This suggests that negative autobiographical memories may hold greater salience for these individuals, thereby influencing their increased accessibility and specificity (Barry, Chiu, et al., 2018).

In line with recent longitudinal findings (König et al., 2025), we observed that young people with CA who self-reported greater friendship support also self-reported lower levels of perceived stress and fewer depressive symptoms. These findings add to a growing body of research emphasizing the importance of friendship support for mental health and well-being, especially in young people with CA (Fritz, de Graaff, et al., 2018; König et al., 2023, 2025; Scheuplein & van Harmelen, 2022; van Harmelen et al., 2016, 2017, 2021). According to social stress buffering models (Gunnar, 2017), the availability of a social partner is thought to mitigate psychological and physiological stress responses, thereby lowering the risk of mental health problems. A recent longitudinal study of young people (aged 16-26 years) with CA found that high-quality friendship support assessed prior to the COVID-19 pandemic buffered depressive symptoms during the pandemic through reducing perceived stress (König et al., 2025). Hence, strong friendship support may have protected our participants from experiencing severe depressive symptoms through reducing perceived stress. But again, due to the cross-sectional nature of this analysis, a comprehensive investigation of such a mechanistic, stress-buffering pathway is not possible.

In addition to our main study objectives, we observed that retrospectively self-reported CA is a potent risk factor for current psychosocial functioning. First, we found that young people with more severe maltreatment experiences self-reported greater depressive symptoms. This finding aligns with numerous studies highlighting the pervasive long-term negative mental health consequences of child maltreatment (Norman et al., 2012; Vachon et al., 2015). For example, meta-analytic evidence suggests that regardless of type, individuals with maltreatment experiences are 2.81 times more likely to develop depression in adulthood compared to those without such experiences (J. Nelson et al., 2017). Interestingly, emotional maltreatment has consistently shown the strongest associations with depressive symptoms and diagnosis (Humphreys et al., 2020), which aligns with our sample predominantly reporting emotional maltreatment experiences.

However, the study's small sample precluded our ability to further investigate specific associations between maltreatment type and depression vulnerability. Second, analyses reported in the supplementary materials revealed that more severe maltreatment experiences were moderately correlated with lower friendship quality. This association could be a sign of social thinning in vulnerable young people and has been reported in previous studies (König et al., 2025; McCrory et al., 2022; McLafferty et al., 2018; Nevard et al., 2021; Salzinger et al., 1993), outlining pragmatic targets for prevention and intervention efforts in the aftermath of CA.

The findings of the current study should be interpreted considering certain limitations. First, the observational study design prevents causal inferences. The neurocognitive social transactional model of psychiatric vulnerability (McCrory et al., 2022) proposes a dynamic interplay between stress adaptation, friendship support, and mental health vulnerability following CA, which ideally requires investigation through prospective longitudinal studies. Additionally, further research is needed to understand if and how adversity-related alterations in autobiographical memory processing are linked to these processes. Second, we did not use formal diagnostic procedures to assess mental health. Due to the potentially stressful nature of the study protocol, young people who recently experienced severe depressive symptoms or suicidal thoughts were not eligible to participate, likely resulting in a sample that may not fully represent the broader population of young people with more severe emotional distress. This could also explain the underrepresentation of young people with more severe CA. Third, compared to previous studies, it is possible that our adapted version of the AMT did not contain sufficient cue words for each valence to identify strong valence-specific effects, as most studies used twice the number of cue words we included (Hitchcock et al., 2019; van Vreeswijk & De Wilde, 2004). Additionally, most prior studies instructed participants to recall any memory associated with a given cue word, rather than memories specifically related to a friend. This shorter version was chosen due to time constraints on the day of testing. Finally, we used retrospective measures of CA to identify eligible participants. However, this approach may have introduced individuals with different risk trajectories for mental health problems compared to those identified using prospective measures (Baldwin et al., 2019).

This study adds to a growing literature highlighting the protective, stress-buffering role of friendships (König et al., 2023, 2025). However, a more nuanced mechanistic understanding is needed to inform preventative intervention efforts. For example, future studies could inspect friendship characteristics, such as stability, closeness, intimacy, or emotional security (Güroğlu, 2022). Furthermore, incorporating observational data and peer reports alongside self-reports can help mitigate potential limitations such as social desirability and recall

biases (Jordan & Troth, 2020). Once the longitudinal THRIVE study is concluded, it would be valuable to replicate the current findings and move beyond only using memory specificity as an index of altered autobiographical memory processing. For example, analyzing sensory-perceptual and contextual details could provide additional fine-grained insights into the associations between adversity-related alterations in autobiographical memory processing and psychosocial functioning (Hitchcock et al., 2022). To achieve this, natural language models offer a promising avenue to accurately and precisely code large amounts of text-based autobiographical memories (Mistica et al., 2024).

In conclusion, we observed that young people with CA who self-reported greater friendship support also reported lower levels of perceived stress and fewer depressive symptoms. Greater specificity when recalling negative autobiographical friendship memories was only weakly associated with more depressive symptoms, but did not survive correction for multiple comparisons and therefore requires exploration in larger longitudinal samples. Finally, lower levels of perceived stress were strongly associated with fewer depressive symptoms. Our findings suggest that friendship support may exert its protective mental health effects through reducing perceived stress in young people with CA, rather than through influencing the specificity of positive or negative autobiographical friendship memories.

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**Data Availability Statement**
Upon publication, all R scripts and pre-processed, anonymized data will be made available on DataverseNL. To facilitate a double anonymized peer review process, R scripts and anonymized data are temporarily available on OSF: https://osf.io/7tnhz/?view_only=6b2837343b1e42e1b30cac8a12e8d97e. Raw AMT data is not provided to safeguard the anonymity of our study participants.

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**Conflicts of Interest**
The authors declare no conflict of interest.

**Institutional Review Board Statement**
The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Medical Ethics Committee Leiden The Hague Delft (NL80017.058.21) in July 2022.

**Informed Consent Statement**
Informed consent was obtained from all subjects involved in the study.

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**Supplementary Information**

**Confirmatory Analyses: Subtracting the Number of Omissions from the Autobiographical Memory Specificity Score**

For each valence, the proportion of specific memories was calculated by dividing the number of specific memories by the total number of cue words and subtracting the number of omissions, as per Debeer et al. (2009) and Hitchcock et al. (2019). A higher proportion indicates a more specific recall of friendship memoires for that valence. Four additional outliers were detected demonstrating strongly lower specificity of both positive and negative autobiographical friendship memories, resulting in a confirmatory sample size of $N = 94$.

***Associations between Friendship Support and Autobiographical Friendship Memory Specificity***

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 222.43 | 230.06 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 227.93 | 243.19 |

**Table S1.1.1.** Model fit statistic for all robust hierarchical multiple regression models predicting positive autobiographical friendship memory specificity. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | 0.18 | 0.08 | [0.03, 0.34] | 2.40 | **.018** |
| Friendship support | 0.09 | 0.09 | [-0.09, 0.27] | 0.99 | .325 |
| **Model 2** | | | | | |
| Intercept | 0.49 | 0.99 | [-1.47, 2.45] | 0.50 | .621 |
| Friendship support | 0.08 | 0.10 | [-0.12, 0.27] | 0.76 | .451 |
| Maltreatment experiences | -0.03 | 0.09 | [-0.20, 0.15] | -0.30 | .762 |
| Age | -0.02 | 0.04 | [-0.10, 0.07] | -0.42 | .679 |
| Gender identity | 0.04 | 0.20 | [-0.36, 0.44] | 0.21 | .838 |

**Table S1.1.2.** Model estimates for all robust hierarchical multiple regression models predicting positive autobiographical friendship memory specificity. The best fitting model was #1. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 223.65 | 231.28 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 229.58 | 244.84 |

**Table S1.1.3.** Model fit statistic for all robust hierarchical multiple regression models predicting negative autobiographical friendship memory specificity. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | 0.29 | 0.05 | [0.19, 0.39] | 6.02 | **< .001** |
| Friendship support | 0.01 | 0.06 | [-0.11, 0.12] | 0.15 | .880 |
| **Model 2** | | | | | |
| Intercept | 0.50 | 0.63 | [-0.75, 1.74] | 0.79 | .431 |
| Friendship support | 0.02 | 0.06 | [-0.11, 0.14] | 0.29 | .774 |
| Maltreatment experiences | 0.03 | 0.06 | [-0.08, 0.14] | 0.49 | .625 |
| Age | -0.01 | 0.03 | [-0.06, 0.05] | -0.31 | .759 |
| Gender identity | -0.02 | 0.13 | [-0.27, 0.24] | -0.12 | .901 |

**Table S1.1.4.** Model estimates for all robust hierarchical multiple regression models predicting negative autobiographical friendship memory specificity. The best fitting model was #1. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

***Associations between Autobiographical Friendship Memory Specificity and Perceived Stress***

| Model | AIC | BIC |
|---|---|---|
| 1: Positive memory specificity | 264.96 | 272.59 |
| 2: Positive memory specificity + maltreatment experiences + age + gender identity | 270.04 | 285.30 |

**Table S1.2.1.** Model fit statistic for all robust hierarchical multiple regression models predicting perceived stress. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.03 | 0.11 | [-0.25, 0.19] | -0.26 | .796 |
| Positive memory specificity | 0.16 | 0.14 | [-0.12, 0.44] | 1.13 | .259 |
| **Model 2** | | | | | |
| Intercept | 0.46 | 1.36 | [-2.25, 3.16] | 0.34 | .738 |
| Positive memory specificity | 0.17 | 0.14 | [-0.11, 0.45] | 1.19 | .236 |
| Maltreatment experiences | 0.11 | 0.12 | [-0.12, 0.34] | 0.96 | .339 |
| Age | -0.01 | 0.06 | [-0.13, 0.11] | -0.13 | .898 |
| Gender identity | -0.18 | 0.27 | [-0.72, 0.36] | -0.66 | .511 |

**Table S1.2.2.** Model estimates for all robust hierarchical multiple regression models predicting perceived stress. The best fitting model was #1. *β* = standardized coefficients; 95% CI = 95% confidence interval.

| Model | AIC | BIC |
|---|---|---|
| 1: Negative memory specificity | 265.98 | 273.61 |
| 2: Negative memory specificity + maltreatment experiences + age + gender identity | 271.15 | 286.41 |

**Table S1.2.3.** Model fit statistic for all robust hierarchical multiple regression models predicting perceived stress. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.03 | 0.11 | [-0.24, 0.20] | -0.16 | .872 |
| Negative memory specificity | 0.07 | 0.15 | [-0.22, 0.36] | 0.48 | .632 |
| **Model 2** | | | | | |
| Intercept | 0.57 | 1.39 | [-2.21, 3.34] | 0.41 | .686 |
| Negative memory specificity | 0.07 | 0.15 | [-0.23, 0.36] | 0.45 | .654 |
| Maltreatment experiences | 0.11 | 0.12 | [-0.13, 0.34] | 0.89 | .376 |
| Age | -0.01 | 0.06 | [-0.13, 0.11] | -0.20 | .843 |
| Gender identity | -0.18 | 0.28 | [-0.74, 0.38] | -0.64 | .523 |

**Table S1.2.4.** Model estimates for all robust hierarchical multiple regression models predicting perceived stress. The best fitting model was #1. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval.

***Associations between Autobiographical Friendship Memory Specificity and Depressive Symptoms***

| Model | AIC | BIC |
|---|---|---|
| 1: Positive memory specificity | 248.78 | 256.41 |
| 2: Positive memory specificity + maltreatment experiences + age + gender identity | 243.53 | 258.78 |

**Table S1.3.1.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | $\beta$ | SE | 95% CI | $t$ | $p$ |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.09 | 0.09 | [-0.28, 0.09] | -1.02 | .310 |
| Positive memory specificity | 0.05 | 0.12 | [-0.18, 0.28] | 0.42 | .677 |
| **Model 2** | | | | | |
| Intercept | -0.21 | 1.06 | [-2.31, 1.90] | -0.20 | .844 |
| Positive memory specificity | 0.07 | 0.11 | [-0.15, 0.29] | 1.59 | .556 |
| Maltreatment experiences | 0.33 | 0.09 | [0.15, 0.51] | 3.65 | **< .001** |
| Age | -0.003 | 0.05 | [-0.10, 0.09] | -0.06 | .949 |
| Gender identity | 0.10 | 0.21 | [-0.33, 0.52] | 0.45 | .653 |

**Table S1.3.2.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.13, $p_{FDW}$ = .001 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

| Model | AIC | BIC |
|---|---|---|
| 1: Negative memory specificity | 247.83 | 255.46 |
| 2: Negative memory specificity + maltreatment experiences + age + gender identity | 242.68 | 257.93 |

**Table S1.3.3.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | $\beta$ | SE | 95% CI | $t$ | $p$ |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.10 | 0.09 | [-0.28, 0.08] | -1.12 | .265 |
| Negative memory specificity | 0.13 | 0.12 | [-0.11, 0.36] | 1.07 | .289 |
| **Model 2** | | | | | |
| Intercept | -0.17 | 1.03 | [-2.23, 1.88] | -0.17 | .868 |
| Negative memory specificity | 0.11 | 0.11 | [-0.11, 0.33] | 0.98 | .328 |
| Maltreatment experiences | 0.33 | 0.09 | [0.15, 0.51] | 3.72 | **< .001** |
| Age | -0.003 | 0.05 | [-0.09, 0.09] | -0.09 | .932 |
| Gender identity | 0.08 | 0.21 | [-0.33, 0.50] | 0.41 | .686 |

**Table S1.3.4.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.13, $p_{FDR}$ = .001 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

**Testing for Normality**

| | Outcome Variable | W | $p$ | Skewness | Kurtosis |
|---|---|---|---|---|---|
| Model 1 | Positive memory specificity | 0.883 | **< .001** | -0.78 | -0.25 |
| Model 2 | Negative memory specificity | 0.891 | **< .001** | -0.59 | -0.42 |
| Model 3 | Perceived stress | 0.991 | .789 | 0.07 | -0.51 |
| Model 4 | Depressive symptoms | 0.913 | **< .001** | 0.81 | -0.31 |

**Table S2.** Shapiro–Wilk tests to assess normality of main variables after outlier removal ($N$ = 98). W = Shapiro–Wilk test statistic. Bold denotes significant effects.

**Robust Hierarchical Multiple Regressions**
***Associations between Friendship Support and Autobiographical Friendship Memory Specificity***

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 276.30 | 284.06 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 280.36 | 295.87 |

**Table S3.1.1.** Model fit statistic for all robust hierarchical multiple regression models predicting positive autobiographical friendship memory specificity. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | 0.07 | 0.10 | [-0.12, 0.27] | 0.76 | .452 |
| Friendship support | 0.15 | 0.11 | [-0.07, 0.38] | 1.34 | .183 |
| **Model 2** | | | | | |
| Intercept | 0.22 | 1.25 | [-2.26, 2.70] | 0.18 | .859 |
| Friendship support | 0.10 | 0.12 | [-0.14, 0.35] | 0.83 | .407 |
| Maltreatment experiences | -0.05 | 0.11 | [-0.27, 0.17] | -0.43 | .669 |
| Age | -0.03 | 0.05 | [-0.14, 0.08] | -0.56 | .579 |
| Gender identity | 0.28 | 0.24 | [-0.21, 0.76] | 1.14 | .257 |

**Table S3.1.2.** Model estimates for all robust hierarchical multiple regression models predicting positive autobiographical friendship memory specificity. The best fitting model was #1. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval.

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 280.39 | 288.15 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 285.08 | 300.59 |

**Table S3.1.3.** Model fit statistic for all robust hierarchical multiple regression models predicting negative autobiographical friendship memory specificity. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | 0.09 | 0.10 | [-0.12, 0.29] | 0.83 | .410 |
| Friendship support | 0.02 | 0.12 | [-0.22, 0.26] | 0.16 | .869 |
| | | | | | |
| **Model 2** | | | | | |
| Intercept | 0.36 | 1.35 | [-2.33, 3.04] | 0.26 | .794 |
| Friendship support | 0.04 | 0.13 | [-0.23, 0.30] | 0.28 | .783 |
| Maltreatment experiences | 0.09 | 0.12 | [-0.15, 0.33] | 0.76 | .447 |
| Age | -0.02 | 0.06 | [-0.14, 0.10] | -0.36 | .718 |
| Gender identity | 0.10 | 0.27 | [-0.43, 0.62] | 0.36 | .721 |

**Table S3.1.4.** Model estimates for all robust hierarchical multiple regression models predicting negative autobiographical friendship memory specificity. The best fitting model was #1. *β* = standardized coefficients; 95% CI = 95% confidence interval.

***Association between Friendship Support and Perceived Stress***

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 268.95 | 276.71 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 274.64 | 290.15 |

**Table S3.2.1.** Model fit statistic for all robust hierarchical multiple regression models predicting perceived stress. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.01 | 0.10 | [-0.21, 0.19] | -0.11 | .410 |
| Friendship support | -0.44 | 0.12 | [-0.68, -0.21] | -3.74 | **< .001** |
| | | | | | |
| **Model 2** | | | | | |
| Intercept | -0.03 | 1.31 | [-2.64, 2.58] | -0.02 | .981 |
| Friendship support | -0.47 | 0.13 | [-0.72, -0.21] | -3.58 | **< .001** |
| Maltreatment experiences | -0.04 | 0.12 | [-0.27, 0.20] | -0.31 | .754 |
| Age | -0.01 | 0.06 | [-0.12, 0.11] | -0.14 | .891 |
| Gender identity | 0.10 | 0.26 | [-0.41, 0.61] | 0.40 | .686 |

**Table S3.2.2.** Model estimates for all robust hierarchical multiple regression models predicting perceived stress. The best fitting model was #1 with $f_p^2 = 0.16$ for friendship support. *β* = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

***Association between Friendship Support and Depressive Symptoms***

| Model | AIC | BIC |
|---|---|---|
| 1: Friendship support | 255.66 | 263.42 |
| 2: Friendship support + maltreatment experiences + age + gender identity | 253.50 | 269.01 |

**Table S3.3.1.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.10 | 0.09 | [-0.27, 0.07] | -1.14 | .256 |
| Friendship support | -0.30 | 0.10 | [-0.50, -0.10] | -2.97 | **.004** |
| **Model 2** | | | | | |
| Intercept | -0.34 | 1.03 | [-2.39, 1.71] | -0.33 | .742 |
| Friendship support | -0.21 | 0.10 | [-0.42, -0.01] | -2.07 | **.041** |
| Maltreatment experiences | 0.25 | 0.09 | [0.07, 0.44] | 2.72 | **.008** |
| Age | -0.01 | 0.05 | [-0.10, 0.08] | -0.20 | .844 |
| Gender identity | 0.24 | 0.20 | [-0.16, 0.64] | 1.18 | .242 |

**Table S3.3.2.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.07 for friendship support and $f_p^2$ = 0.08 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

***Associations between Autobiographical Friendship Memory Specificity and Perceived Stress***

| Model | AIC | BIC |
|---|---|---|
| 1: Positive memory specificity | 280.80 | 288.56 |
| 2: Positive memory specificity + maltreatment experiences + age + gender identity | 285.61 | 301.12 |

**Table S3.4.1.** Model fit statistic for all robust hierarchical multiple regression models predicting perceived stress. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.02 | 0.11 | [-0.24, 0.19] | -0.23 | .818 |
| Positive memory specificity | 0.18 | 0.11 | [-0.04, 0.41] | 1.65 | .102 |
| **Model 2** | | | | | |
| Intercept | 0.66 | 1.34 | [-2.00, 3.33] | 0.49 | .623 |
| Positive memory specificity | 0.20 | 0.11 | [-0.02, 0.43] | 1.82 | .072 |
| Maltreatment experiences | 0.13 | 0.12 | [-0.10, 0.36] | 1.13 | .262 |
| Age | -0.02 | 0.06 | [-0.14, 0.10] | -0.30 | .761 |
| Gender identity | -0.17 | 0.26 | [-0.68, 0.34] | -0.65 | .514 |

**Table S3.4.2.** Model estimates for all robust hierarchical multiple regression models predicting perceived stress. The best fitting model was #1. *β* = standardized coefficients; 95% CI = 95% confidence interval.

| Model | AIC | BIC |
|---|---|---|
| 1: Negative memory specificity | 278.87 | 286.62 |
| 2: Negative memory specificity + maltreatment experiences + age + gender identity | 284.11 | 299.62 |

**Table S3.4.3.** Model fit statistic for all robust hierarchical multiple regression models predicting perceived stress. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | *β* | SE | 95% CI | *t* | *p* |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.03 | 0.11 | [-0.24, 0.19] | -0.24 | .812 |
| Negative memory specificity | 0.20 | 0.11 | [-0.02, 0.41] | 1.84 | .069 |
| **Model 2** | | | | | |
| Intercept | 0.77 | 1.38 | [-1.97, 3.50] | 0.55 | .580 |
| Negative memory specificity | 0.19 | 0.11 | [-0.04, 0.41] | 1.66 | .100 |
| Maltreatment experiences | 0.09 | 0.12 | [-0.15, 0.32] | 0.73 | .466 |
| Age | -0.03 | 0.06 | [-0.15, 0.09] | -0.43 | .668 |
| Gender identity | -0.13 | 0.26 | [-0.65, 0.40] | -0.48 | .630 |

**Table S3.4.4.** Model estimates for all robust hierarchical multiple regression models predicting perceived stress. The best fitting model was #1. *β* = standardized coefficients; 95% CI = 95% confidence interval.

***Associations between Autobiographical Friendship Memory Specificity and Depressive Symptoms***

| Model | AIC | BIC |
|---|---|---|
| 1: Positive memory specificity | 260.87 | 268.63 |
| 2: Positive memory specificity + maltreatment experiences + age + gender identity | 255.73 | 271.24 |

**Table S3.5.1.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | $\beta$ | SE | 95% CI | $t$ | $p$ |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.10 | 0.09 | [-0.28, 0.07] | -1.19 | .236 |
| Positive memory specificity | 0.10 | 0.09 | [-0.07, 0.28] | 1.16 | .251 |
| **Model 2** | | | | | |
| Intercept | -0.02 | 1.01 | [-2.02, 1.99] | -0.02 | .987 |
| Positive memory specificity | 0.12 | 0.08 | [-0.05, 0.29] | 1.43 | .157 |
| Maltreatment experiences | 0.34 | 0.09 | [0.16, 0.51] | 3.87 | **< .001** |
| Age | -0.01 | 0.04 | [-0.10, 0.08] | -0.26 | .794 |
| Gender identity | 0.09 | 0.19 | [-0.30, 0.47] | 0.44 | .659 |

**Table S3.5.2.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.12, $p_{FDR}$ = .001 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

| Model | AIC | BIC |
|---|---|---|
| 1: Negative memory specificity | 255.49 | 263.24 |
| 2: Negative memory specificity + maltreatment experiences + age + gender identity | 251.35 | 266.86 |

**Table S3.5.3.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | $\beta$ | SE | 95% CI | $t$ | $p$ |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.10 | 0.09 | [-0.28, 0.07] | -1.20 | .234 |
| Negative memory specificity | 0.21 | 0.09 | [0.04, 0.39] | 2.40 | **.018** |
| **Model 2** | | | | | |
| Intercept | 0.13 | 1.03 | [-1.91, 2.17] | 0.13 | .898 |
| Negative memory specificity | 0.17 | 0.08 | [0.01, 0.34] | 2.07 | **.041** |
| Maltreatment experiences | 0.30 | 0.09 | [0.13, 0.48] | 3.44 | **< .001** |
| Age | -0.02 | 0.05 | [-0.11, 0.07] | -0.38 | .706 |
| Gender identity | 0.08 | 0.20 | [-0.31, 0.47] | 0.40 | .688 |

**Table S3.5.4.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.07, $p_{FDR}$ = .082 for negative memory specificity and $f_p^2$ = 0.11, $p_{FDR}$ = .001 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

***Association between Perceived Stress and Depressive Symptoms***

| Model | AIC | BIC |
|---|---|---|
| 1: Perceived stress | 212.86 | 220.62 |
| 2: Perceived stress + maltreatment experiences + age + gender identity | 204.31 | 219.82 |

**Table S3.6.1.** Model fit statistic for all robust hierarchical multiple regression models predicting depressive symptoms. AIC = Akaike information criterion; BIC = Bayesian information criterion.

| Parameters | $\beta$ | SE | 95% CI | $t$ | $p$ |
|---|---|---|---|---|---|
| **Model 1** | | | | | |
| Intercept | -0.10 | 0.06 | [-0.22, 0.02] | -1.64 | .104 |
| Perceived stress | 0.58 | 0.06 | [0.46, 0.70] | 9.39 | < **.001** |
| **Model 2** | | | | | |
| Intercept | -0.19 | 0.78 | [-1.74, 1.36] | -0.24 | .810 |
| Perceived stress | 0.56 | 0.06 | [0.43, 0.68] | 8.87 | < **.001** |
| Maltreatment experiences | 0.21 | 0.07 | [0.08, 0.35] | 3.20 | **.002** |
| Age | -0.01 | 0.03 | [-0.08, 0.06] | -0.27 | .786 |
| Gender identity | 0.17 | 0.15 | [-0.13, 0.46] | 1.12 | .267 |

**Table S3.6.2.** Model estimates for all robust hierarchical multiple regression models predicting depressive symptoms. The best fitting model was #2 with $f_p^2$ = 0.77 for perceived stress and $f_p^2$ = 0.15 for maltreatment experiences. $\beta$ = standardized coefficients; 95% CI = 95% confidence interval. Bold denotes significant effects.

**Spearman's Rank Correlations**

Non-parametric Spearman's rank correlations ($r_s$) are presented in Table 4.1 alongside significance levels. Correlation coefficients can be interpreted as small ($r_s$ = .15 to .24), medium ($r_s$ = .25 to .34), or large ($r_s \geq .35$) (Gignac & Szodorai, 2016; Schober et al., 2018). First, the correlations between friendship support and specificity of both positive ($r_s$ = .09, $p$ = .392) and negative ($r_s$ = .05, $p$ = .653) autobiographical friendship memories were non-significant (rejecting hypothesis 1.1). However, we observed a moderate negative correlation between friendship support and perceived stress ($r_s$ = -.34, 95% $CI_{bootstrap}$ [-0.51, -0.16], $p$ < .001; confirming hypothesis 1.2) as well as a moderate negative correlation between friendship support and depressive symptoms ($r_s$ = -.25, 95% $CI_{bootstrap}$ [-0.45, -0.04], $p$ = .018; confirming hypothesis 1.3). Second, the correlations between specificity of positive autobiographical friendship memories and perceived stress ($r_s$ = .13, $p$ = .209) as well as between positive memory specificity and depressive symptoms ($r_s$ = .11, $p$ = .335) were non-significant (rejecting hypotheses 2.1 and 2.2). Third, we observed a strong positive correlation between perceived stress and depressive symptoms ($r_s$ = .67, 95% $CI_{bootstrap}$ [0.53, 0.78], $p$ < .001; confirming hypothesis 3).

In addition to our a priori hypotheses, we observed a small positive correlation between specificity of positive and negative autobiographical friendship memories ($r_s$ = .21, 95% $CI_{bootstrap}$ [0.02, 0.39], $p$ = .032). Next, we observed a small positive correlation between specificity of negative autobiographical friendship memories and depressive symptoms ($r_s$ = .20, 95% $CI_{bootstrap}$ [0.01,

0.39], $p$ = .042). Further, we observed a moderate negative correlation between maltreatment experiences and friendship support ($r_s$ = -.27, 95% $CI_{bootstrap}$ [-0.45, -0.07], $p$ = .006). In addition, maltreatment experiences were positively correlated with depressive symptoms ($r_s$ =.39, 95% $CI_{bootstrap}$ [0.23, 0.54], $p$ < .001).

| | **Variables** | **1** | **2** | **3** | **4** | **5** | **6** | **7** |
|---|---|---|---|---|---|---|---|---|
| 1. | Age | - | | | | | | |
| 2. | Gender identity (female) | .01 | - | | | | | |
| 3. | Positive memory specificity | -.10 | .10 | - | | | | |
| 4. | Negative memory specificity | -.03 | .04 | **.21**$^{*}$ | - | | | |
| 5. | Maltreatment experiences | .07 | .12 | -.09 | .10 | - | | |
| 6. | Friendship support | .08 | .12 | .09 | .05 | **-.27**$^{**}$ | - | |
| 7. | Depressive symptoms | -.01 | .09 | .11 | **.20**$^{*}$ | **.39**$^{***}$ | **-.25**$^{*}$ | - |
| 8. | Perceived stress | -.05 | -.04 | .13 | .18 | .18 | **-.34**$^{***}$ | **.67**$^{***}$ |

**Table 4.1.** Spearman's rank correlations between study variables ($N$ = 97). Gender identity was dummy coded with female as the reference category (female = 1, male = 0). To simplify interpretation, the non-binary category ($n$ = 1) was excluded from these bivariate correlation analyses, which did not significantly affect the reported correlation coefficients. Significant correlations appear in bold. $^{***}p < .001$, $^{**}p < .01$, $^{*}p < .05$.

# Chapter 7

**Executive Summary & General Discussion**

## Executive Summary

Globally, approximately 60% of children and adolescents are exposed to at least one form of childhood adversity (Madigan et al., 2023). Chronic and repeated exposure to such stressful and potentially traumatic experiences, particularly during sensitive developmental periods, dramatically elevates the risk of both experiencing and perpetrating victimization as well as developing various forms of psychopathology later in life (McLaughlin, 2016; Widom, 1989b; Widom et al., 2008). Theoretical models propose that adversity-induced neurocognitive adaptations aid different forms of victimization (Cicchetti & Valentino, 2006; Sroufe & Rutter, 1984) and increase psychopathology vulnerability through their impact on social functioning (Gerin et al., 2019; McCrory et al., 2022). For example, the neurocognitive social transactional model of psychiatric vulnerability (introduced in **Chapter 1**; McCrory et al. (2022)) suggests that these adaptations may lead to social thinning (i.e., fewer protective social relationships) and stress generation (i.e., a social environment characterized by more stressful interpersonal experiences), thus exacerbating risks for victimization and psychopathology.

Importantly, young people who are able to maintain high levels of perceived friendship support show reduced risks of victimization and psychopathology following childhood adversity (Huang et al., 2013; van Harmelen et al., 2016, 2021; T. Williams et al., 2005). However, the underlying mechanisms that may explain this friendship buffering effect remain poorly understood. The social stress buffering literature suggests that the presence and availability of one or more supportive social partners can attenuate perceptions, reactions, and physiological responses to acute stress (Gunnar, 2017), thereby lowering allostatic load and ultimately promoting better health outcomes (Doan & Evans, 2011; Hennessy et al., 2009).

Building on these frameworks, this dissertation aimed to identify psychological, cognitive, and neural stress-related pathways through which social support, particularly friendships, reduce the risks of victimization and psychopathology in young people with childhood adversity. To advance a more nuanced understanding of these mechanisms, insights are drawn from literature reviews, cross-sectional analyses, and longitudinal analyses, employing both behavioral and neuroimaging techniques.

The **first part** of this dissertation examined how maladaptive neurocognitive and social functioning following maltreatment experiences during childhood or adolescence can increase the risk of experiencing and perpetrating victimization later in life. **Chapter 2** (Scheuplein et al., 2023) reviewed the cycle of victimization literature, highlighting the link between child maltreatment and victimization within and outside the family environment. It also outlined three

social functioning mechanisms underlying this association and reflected on the potential buffering role of social support. Specifically, victimization within the family environment has been reviewed in the context of the intergenerational transmission of maltreatment hypothesis. This hypothesis encompasses two perspectives: the victim-to-perpetrator perspective, where victims of maltreatment are more likely to become maltreating parents (Widom, 1989b), and the victim-to-victim perspective, where children of parents with a history of maltreatment are more likely to become victims themselves, even if their parents are not direct perpetrators (Madigan et al., 2019). Victimization outside the family environment has been reviewed in the context of the violence breeds violence hypothesis, which posits that being maltreated as a child increases the risk of becoming a violent perpetrator later in life (Fitton et al., 2020). In line with latent vulnerability and adaptive calibration models (Del Giudice et al., 2011; McCrory et al., 2022; McCrory & Viding, 2015), three mechanisms were reviewed as potential contributors to impaired social functioning and the association between child maltreatment and victimization: heightened attentional bias to threat, diminished reward processing and feedback learning, and emotion dysregulation. For example, a heightened attentional bias to threat may facilitate adaptive behaviors in high-stress environments. However, in non-threatening contexts, this adaptation may increase the risk of maladaptive behaviors, such as over-attributing hostile intent to others, which may provoke aggressive or avoidance behavior, impair social functioning, and increase risks for victimization and psychopathology (Crick & Dodge, 1994; N. V. Miller & Johnston, 2019). The chapter concluded by highlighting the role of safe, stable, and nurturing social support as a protective factor capable of mitigating victimization and psychopathology risk through potentially influencing these neurocognitive risk mechanisms (Schofield et al., 2013; van Harmelen et al., 2016). However, it became evident that breaking the cycle of victimization and improving health outcomes requires greater translation of knowledge about how neurocognitive mechanisms are shaped by childhood adversity and influenced by social support.

Hence, the **second part** of this dissertation zoomed in more closely on the psychological, cognitive, and neural stress-related pathways that link friendship support to reduced psychopathology risk in young people with childhood adversity. **Chapter 3** (Scheuplein & van Harmelen, 2022) systematically reviewed whether friendships reduce neural stress responses in young people with childhood adversity. In line with the PRISMA guidelines (Page et al., 2021), this pre-registered systematic literature review included empirical studies published in English involving young people with an average age between 10 and 24 years who had experienced childhood adversity. Friendships had to be assessed within the same average age range and neural stress responses had to be measured using neuroimaging techniques. After screening 4,297 records and 66 full-text articles for eligibility, only two studies matched all eligibility criteria. Two more studies

were included after broadening the scope to allow stress responses from various neurobiological systems. Ultimately, only two of these four studies directly investigated whether friendships buffer neurobiological stress responses in young people with childhood adversity. In a sample of institutionalized young people, Tang et al. (2021) found that high-quality friendships at age 12 can buffer the indirect effect of maladaptive stress physiology on peer problems at age 16. In contrast, in a small and well-functioning sample of young people with childhood adversity, Fritz, Stretton, et al. (2020) found no association between friendship support at ages 14 or 17 and affective behavioral or neural responses to social rejection. Hence, these findings highlight the critical need for future research to examine whether friendships aid mental health and well-being through mitigating neurobiological stress responses in young people with childhood adversity.

Thus, to deepen the mechanistic understanding of friendship stress buffering, **Chapter 4** (König et al., 2023) examined whether perceived friendship quality was associated with better mental health and well-being as well as reduced neural stress responses in young people with childhood adversity. This study analyzed cross-sectional behavioral and neuroimaging data from the Resilience After Individual Stress Exposure (RAISE) study (Moreno-López et al., 2021), which involved 102 young people (aged 16-26 years) in the United Kingdom (UK) who retrospectively self-reported low to moderate levels of childhood adversity. While no support was found for social thinning following childhood adversity, high-quality friendships were strongly associated with better mental health and well-being. A representative subset of 62 young people underwent functional magnetic resonance imaging while completing the Montreal Imaging Stress Task (Dedovic et al., 2005), an acute psychosocial stress paradigm. Acute stress exposure increased state anxiety and elicited enhanced neural activity in five predefined frontolimbic brain regions: the left hippocampus, bilateral insula, left medial prefrontal cortex (anterior cingulate cortex), right nucleus accumbens, and bilateral thalamus. Dimension-specific analyses revealed a weak interaction between threat experiences and friendship quality predicting left hippocampal reactivity to stress. Specifically, left hippocampal reactivity to acute stress increased with more severe threat experiences in participants reporting lower friendship quality. However, this effect did not survive multiple comparison correction and requires replication in larger, ideally longitudinal samples.

Although the COVID-19 outbreak and the reallocation of clinical research facilities shortened the data collection period of the RAISE study and led to a smaller neuroimaging sample, this collective, multidimensional stressor offered a rare opportunity to longitudinally follow the same sample of 102 vulnerable young people as part of the Resilience after the COVID-19 Threat (REACT) study (A. J. Smith et al., 2021). **Chapter 5** (König et al., 2025) therefore investigated friendship buffering effects on mental health symptoms before and at three

timepoints during the COVID-19 pandemic. Specifically, remote behavioral assessments were analyzed from before the pandemic (baseline), the first UK lockdown, the phased reopening, and the second UK lockdown. Compared to pre-pandemic baseline levels, anxiety symptoms peaked during the first lockdown and returned to baseline levels thereafter. Depressive symptoms on the other hand continued to rise following the COVID-19 outbreak. Perceived friendship quality was elevated during both lockdown periods but return to baseline levels during reopening. Social thinning was observed during the COVID-19 pandemic in that more severe childhood adversity was associated with lower friendship quality. Across all assessment timepoints, greater friendship quality was consistently associated with lower anxiety and depressive symptoms and vice versa. Notably, high-quality friendship support before the pandemic buffered anxiety and depressive symptoms during the pandemic through reducing perceived stress.

Finally, **Chapter 6** (König et al., 2025) investigated whether friendship support engages cognitive patterns shaped by childhood adversity to lower stress and boost mental health. Inspired by the neurocognitive social transactional model of psychiatric vulnerability (McCrory et al., 2022), this chapter examined whether friendship support promotes mental health in young people with childhood adversity through influencing the specificity of positive autobiographical friendship memories, which may, in turn, reduce stress perceptions. This study analyzed both quantitative and qualitative cross-sectional behavioral data from the first 100 participants of the Towards Health and Resilience in Volatile Environments (THRIVE) study, an ongoing longitudinal study of young people aged 18-24 years in the Netherlands, all of whom retrospectively self-reported low to moderate levels of childhood adversity. The findings indicated that while more severe childhood adversity was associated with social thinning, individuals who were able to maintain high levels of perceived friendship support self-reported lower levels of perceived stress and fewer depressive symptoms. Contrary to initial predictions, the specificity of positive autobiographical friendship memories was not associated with friendship support. These results, alongside the longitudinal findings in the previous chapter, suggest that friendship support may protect mental health in young people with childhood adversity through reducing perceived stress, rather than by influencing autobiographical memory processing.

Together, this dissertation marks an essential step toward a more nuanced understanding of the psychological, cognitive, and neural stress-related pathways through which social support, particularly friendships, reduce victimization and psychopathology risk in young people with childhood adversity. Each review and empirical chapter contextualized its findings within the broader literature, while also acknowledging relevant limitations. The following general discussion synthesizes key findings, addresses general limitations, and proposes directions for future research.

## General Discussion

Friendships play a pivotal role in buffering stress responses and safeguarding mental health in young people with childhood adversity. However, this seemingly straightforward conclusion requires careful contextualization to clarify its scope and limitations. This dissertation yielded key empirical insights into the interwoven relationships between childhood adversity, stress mechanisms, friendship support, and psychopathology (Figure 1).

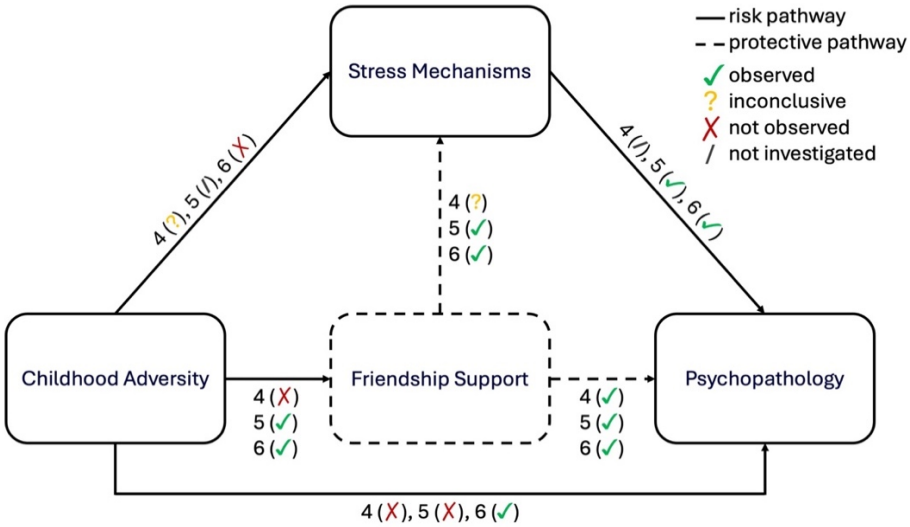


**Figure 1.** Key constructs and findings across empirical **Chapters 4, 5, and 6**. Arrows depict theory-based risk (solid) and protective (dashed) pathways. Constructs include childhood adversity (cumulative, threat-specific, deprivation-specific), stress mechanisms (neural, psychological), friendship support (availability, quality), and psychopathology (internalizing symptoms). Each pathway is annotated with the chapter number (4, 5, 6) and indicates if an effect consistent with the predicted pathway was observed (green tick), not observed (red cross), not investigated (gray slash), or yielded inconclusive findings (orange question mark).

To contextualize the empirical findings of this dissertation, it is useful to first consider the specific characteristics of the samples examined in **Chapters 4, 5, and 6**. Across the three empirical studies, data were analyzed from a total of 202 adolescents and young adults aged 16-26 years, each with a retrospectively self-reported history of childhood adversity within the family environment. Specifically, **Chapters 4 and 5** drew on data from a sample of 102 British young people ($M_{age}$ = 22.24, 64% female), while **Chapter 6** presents findings from a sample of 100 Dutch young people ($M_{age}$ = 21.23, 79% female). Although both sample sizes provided sufficient statistical power for the conducted analyses, the relatively small sample sizes highlight a common limitation in research involving

vulnerable populations, where recruitment and retention pose significant challenges (Bornstein et al., 2013). Based on established cut-off scores for the Childhood Trauma Questionnaire (Bernstein et al., 1994), both the British and Dutch sample can be characterized reporting low to moderate levels of childhood adversity. Additionally, both samples self-reported on average high level of perceived friendship support, indicating particularly well-functioning groups of vulnerable young people. Data collection for both samples took place remotely and in-person, which could have unwillingly led to the exclusion of individuals who were unable to access the internet to complete parts of the study.

On the one hand, some findings were consistently observed across the two independent samples of young people with childhood adversity, revealing broader patterns in line with previous research. First, young people who experienced more severe childhood adversity were at heightened risk of diminished access to or maintenance of supportive friendships (**Chapters 5, 6)**. In **Chapter 5**, the REACT study analyzed longitudinal data and demonstrated patterns of social thinning following the COVID-19 outbreak. This finding aligns with the stress sensitization hypothesis (Hammen, 2015; Hammen et al., 2000), which suggests that individuals with a history of childhood adversity are more sensitive to later stressors. As shown by Wade et al. (2019), this stress sensitization can affect externalizing behavior, thereby impairing social functioning. In **Chapter 6**, the THRIVE study analyzed cross-sectional data and found a moderate negative association between childhood adversity and friendship support. This finding aligns with prior cross-sectional studies reporting lower friendship support in young people with more severe childhood adversity (McLafferty et al., 2018; Nevard et al., 2021; Salzinger et al., 1993).

Second, vulnerable young people with higher levels of perceived friendship support reported improved psychosocial functioning (**Chapters 4, 5, 6**). In **Chapter 4**, the RAISE study analyzed cross-sectional data and found a moderate positive association between friendship quality and psychosocial functioning. Furthermore, **Chapter 5**, which prospectively examined the same sample of vulnerable young people, found that higher friendship quality also predicted reduced internalizing symptoms, particularly anxiety and depressive symptoms, during the COVID-19 pandemic. Similarly, in **Chapter 6**, greater friendship support was moderately associated with fewer depressive symptoms. These robust friendship buffering findings replicate previous cross-sectional and longitudinal research highlighting the critical role of social support, especially friendship support, in promoting mental health and well-being in young people with childhood adversity (Fritz, de Graaff, et al., 2018; Lagdon et al., 2021; Salazar et al., 2011; van Harmelen et al., 2016, 2021).

Third, vulnerable young people with greater friendship support reported lower levels of perceived stress (**Chapters 5, 6**). In **Chapter 5**, pre-pandemic friendship quality longitudinally buffered anxiety and depressive symptoms through reducing perceived stress during the COVID-19 pandemic. In **Chapter 6**, greater friendship support was cross-sectionally associated with lower levels of perceived stress, which, in turn, were linked to fewer depressive symptoms. Critically, these findings align with and extend the social stress buffering literature (Gunnar, 2017; Gunnar & Hostinar, 2015), emphasizing the pivotal role of friendship support in mitigating stress responses in vulnerable young people (C.-Y. S. Lee & Goldstein, 2016; Shahar et al., 2009) and, thereby, reducing psychopathology risk (Achterberg et al., 2021; Gotlib et al., 2020).

On the other hand, some findings appeared more specific to individual studies. First, despite robust evidence linking childhood adversity to various forms of youth psychopathology (Clark et al., 2010; Francis et al., 2023; Kessler et al., 2010; McLaughlin, 2016), this association was not observed in one of the two samples investigated (**Chapters 4, 5**). Specifically, the British sample analyzed in **Chapters 4 and 5** showed no association between childhood adversity and psychopathology, either cross-sectionally or longitudinally. The absence of such a relationship may reflect a relatively well-functioning sample of young people who reported only low to moderate levels of childhood adversity and, on average, high levels of perceived friendship quality.

Second, it remains unclear whether friendship support buffers neural stress responses in young people with childhood adversity (**Chapters 3, 4**). As highlighted in the systematic review presented in **Chapter 3**, only two studies have previously investigated the stress buffering role of friendship support at the neurobiological level in this population. Tang et al. (2021) found that low levels of friendship quality were associated with blunted sympathetic nervous system reactivity to social rejection feedback at age 12, linking early institutionalization experiences with greater peer problems at age 16. In contrast, Fritz, Stretton, et al. (2020) found that friendship support at ages 14 or 17 was not associated with neural responses to social rejection feedback at age 18 in a sample of young people with childhood adversity. Similarly, **Chapter 4**, does not provide conclusive evidence of whether friendship support buffers frontolimbic responses to experimentally induced acute psychosocial stress. Although high-quality friendships were associated with reduced left hippocampal reactivity to acute stress in young people with threat experiences, this interaction effect did not survive correction for multiple comparisons. While this uncorrected, dimension-specific finding aligns with previous research linking childhood adversity, particularly threat exposure, to structural and functional alterations in the hippocampus, which are known risk factors for later-life psychopathology (Y. Chen et al., 2008; Cohodes et al., 2021; McLaughlin et al., 2014), future research

in needed to replicate and extend these findings in larger, ideally longitudinal samples.

Third, although not a primary focus of **Chapter 6**, young people with more severe childhood adversity did not report higher levels of perceived stress. On average, this Dutch sample reported low to moderate levels of perceived stress in the four weeks prior to assessment, suggesting the presence of protective factors, such as friendship support. However, it is possible that in the absence of such protective factors, more severe childhood adversity, or exposure to acute stress (e.g., a global pandemic), vulnerable young people may report higher levels of perceived stress, as demonstrated in previous studies (Bourassa et al., 2023; Gotlib et al., 2020; McLaughlin, Conron, et al., 2010).

**Friendships Matter**

Friendships play a vital role in the lives of young people, particularly when it comes to mitigating psychopathology risk following childhood adversity (**all Chapters**). Simultaneously, critical knowledge gaps remain in the understanding of stress-related mechanisms that underpin these protective effects (**Chapter 3**), insights that are essential for the development of targeted prevention and intervention strategies.

**Chapters 4, 5, and 6** provide robust empirical evidence affirming *that* friendships matter by demonstrating consistent positive associations between friendship support and mental health in young people with childhood adversity. While prior research had already established this link (A. S. Masten et al., 2003; Powers et al., 2009; van Harmelen et al., 2016, 2021), replicating this powerful insight in two independent, hard-to-recruit samples of young people with childhood adversity holds considerable value. It emphasizes that investing time in the formation and maintenance of friendships can help mitigate the disproportionately high risk of experiencing mental health problems faced by those with a history of childhood adversity. This is particularly relevant for individuals with multiple adversities, who, as noted in **Chapter 1**, are 3.7 times more likely to develop anxiety and 4.7 times more likely to experience depression (K. Hughes et al., 2017). Reducing the prevalence of adversity-related mental health conditions could also alleviate the broader societal and economic burdens these issues impose (Bellis et al., 2019), benefitting not only vulnerable individuals but also society at large.

To optimally support young people with childhood adversity, who are known to be at greater risk for social thinning (**Chapters 5, 6**; McCrory et al. (2019), (2022)), future research should focus on identifying behaviors that promote the initiation and maintenance of supportive friendships (Oswald et al., 2004). Additionally, it is essential to explore how these skills can be safely harnessed in

an increasingly digital world, where vulnerable youth face serious risks, such as exposure to cyberbullying or the normalization of self-harm behavior (Daine et al., 2013).

**Chapter 5** provides rare longitudinal insights into *how* friendships matter by identifying a psychological pathway through which friendships provide stress-buffering mental health benefits for young people with childhood adversity. Specifically, this prospective longitudinal study demonstrated that pre-pandemic levels of perceived friendship quality mitigated anxiety and depressive symptoms during the COVID-19 pandemic through reducing levels of perceived stress. Although **Chapter 6**, based on cross-sectional data, could not examine such a longitudinal friendship stress buffering pathway, it nonetheless confirmed the buffering role of friendships, showing that higher perceived friendship quality was associated with lower levels of perceived stress and fewer depressive symptoms.

These results integrate well with prior research highlighting the relationship between elevated levels of subjectively appraised stress (i.e., perceived stress) and greater physiological stress responses, including heightened circulating levels of pro-inflammatory biomarkers (e.g., interleukin-6 (IL-6) or C-reactive protein (CRP)) (Knight et al., 2021), accelerated biological aging (Bourassa et al., 2023; Epel et al., 2004), along with poor physical and mental health outcomes that accompany these allostatic states (Christensen et al., 2019; Guidi et al., 2021; McEwen, 2005). In the context of childhood adversity, persistent and severe exposure to and perception of stress is believed to disrupt neuroendocrine and immune system regulation, contributing to the onset and maintenance of treatment-resistant psychopathology (Ioannidis et al., 2020; G. Miller et al., 2009; Mondelli et al., 2015). For example, a prospective longitudinal study demonstrated that early exposure to adverse experiences (prior to age 8) predicted elevated levels of IL-6 and CRP at age 10 as well as increased levels of CRP at age 15 (Slopen et al., 2013). Furthermore, structural equation modeling by Knight et al. (2021) demonstrated that perceived stress was associated with flattened diurnal cortisol slopes (indicating HPA axis dysregulation), which, in turn, were associated with heightened systemic inflammation in U.S. adults with traumatic life experiences. Prolonged systemic inflammatory responses have been linked to glucocorticoid resistance, diminishing the anti-inflammatory effects of glucocorticoids and further elevating levels of peripheral pro-inflammatory biomarkers (Barnes, 1998; Barnes & Adcock, 2009). Through permeating the vascular blood-brain barrier, pro-inflammatory biomarkers are thought to exert disruptive effects on brain development and functioning, thereby increasing psychopathology risk (Danese & Baldwin, 2017; A. H. Miller & Raison, 2016).

Conversely, social relationships, including friendships, may help counteract or buffer these effects. Meta-analytic findings across 47 studies have shown that

social support and social integration were robustly associated with lower levels of inflammatory markers, such as IL-6 and CRP (Uchino et al., 2018). Furthermore, longitudinal research with breast cancer survivors revealed that lower perceived social support before treatment predicted higher IL-6 levels, greater pain, and more depressive symptoms post-treatment, compared to those with greater levels of perceived social support pre-treatment (S. Hughes et al., 2014). These findings illustrate the need for resilience research to adopt a complexity theory approach that captures the dynamic interplay between multiple psychological, social, and neurobiological systems over time, ideally through prospective longitudinal study designs (Ioannidis et al., 2020).

To advance the understanding of *how* and *why* friendships matter for young people with childhood adversity, future research should also address several conceptual limitations of the studies presented in this dissertation. First, each empirical chapter (**Chapters 4, 5, 6**) utilized a single friendship support index that measured the subjectively self-reported perception of support, leaving it unclear which specific aspects of friendship support are most critical for providing protective, stress-buffering benefits. Friendships typically involve characteristics like mutuality, reciprocity, trust, and a sense of obligation (Bukowski et al., 1998; Dunbar, 2018; Hartup & Stevens, 1997). Additionally, the principal of homophily suggests that social networks, including friendships, often form based on similarities across dimensions, such as age, gender, language, place of origin, educational history, hobbies and interests, sense of humor, and worldview (Dunbar, 2018; McPherson et al., 2001). Thus, the pathway towards mental health may vary depending on the characteristics or shared dimensions that define a friendship. For example, friendships based on a shared sense of humor are more likely to involve laughter, which has been shown to increase endorphin secretion, reduce endocrine release, lower levels of perceived stress, and activate brain regions associated with reward processing, such as the thalamus or caudate nucleus (Manninen et al., 2017; Mora-Ripoll, 2011; Yim, 2016). Consequently, by stimulating laughter, friendships may reduce psychological and neurobiological stress responses, thereby promoting mental health.

However, while similarity within friendships can bolster their protective effects, individuals with childhood adversity are at heightened risk of forming friendships that may be more harmful than beneficial to their mental health. For example, Raposa et al. (2015) conducted a prospective longitudinal study following individuals from birth to age 25 and found that those who experienced adversity by age 5 were more likely to have a best friend at age 20 who struggles with psychopathology. This, in turn, increasing their own risk of depressive symptoms over the subsequent two to five years. Hence, future research should carefully examine which specific qualities make friendships effective buffers and which aspects may render them risk factors for vulnerable youth.

Second, the research presented in this dissertation examined the buffering role of friendship support in isolation, without accounting for potential interrelations with other protective factors. The drawback of this approach becomes apparent when considering the findings by Fritz, Fried, et al. (2018), who applied network modeling to examine the interrelations between protective factors in 14-year-olds with and without childhood adversity. Their research revealed that expressive suppression (i.e., the ability to intentionally inhibit or suppress outward emotional expression) had a distinct relationship with friendship support across these groups. Specifically, low expressive suppression was associated with low friendship support in the childhood adversity group but with high friendship support in the group without childhood adversity. Regarding the group of young people with childhood adversity, this finding puts forward three possible interpretations, as outlined by the authors: (1) ineffective emotional communication leads to friendship withdrawal, (2) friendship withdrawal contributes to ineffective emotional communication, or (3) these two factors influence each other reciprocally over time (Fritz, Fried, et al., 2018). This suggests that protective factors can sometimes interfere with, rather than strengthen, one another. Future translational research employing advanced modeling techniques is needed to uncover such potentially dysfunctional interrelations. Identifying these dynamics could help make interventions more targeted and effective, for example, by teaching young people with childhood adversity appropriate emotional communication skills, which may, in turn, foster more supportive friendships.

**Evolving Perspectives on Childhood Adversity**

Childhood adversity is a common and powerful risk factor for negative health outcomes in later life, including internalizing and externalizing psychopathology (Grummitt et al., 2021; Madigan et al., 2023). For example, as outlined in **Chapter 1**, young people with childhood adversity are three to four times more likely to develop internalizing psychopathology, such as anxiety or depression, compared to their peers without such experiences (K. Hughes et al., 2017). Relatedly, **Chapter 2** highlights that the children of parents who experienced child maltreatment are two to three times more likely to experience maltreatment themselves, compared to those with non-maltreated parents (Madigan et al., 2019). This intergenerational cycle of victimization may, in turn, contribute to the development of externalizing psychopathology, such as aggressive behavior (Richey et al., 2016), potentially through affecting a range of psychological, cognitive, and neural stress-related pathways critical for adaptive social functioning (Alink et al., 2019).

To adequately predict individualized health risks associated with childhood adversity, identify the mechanisms underlying these associations, and develop effective interventions to prevent or mitigate its detrimental consequences, it is

essential to rethink how childhood adversity is conceptualized, operationalized, and measured (Danese, 2020; Danese & Lewis, 2022). Conceptualization involves defining what constitutes childhood adversity, while operationalization translates these theory-based definitions into specific, measurable components. Measurement then employs reliable and valid tools to quantify childhood adversity based on the chosen operational framework.

As outlined in **Chapter 1** and central to all empirical studies presented in **Chapters 4, 5, and 6**, childhood adversity is *conceptualized* as the chronic or repeated exposure to stressful and potentially traumatic experiences during childhood or adolescence (before age 18) that represent a deviation from the "expectable" environment, such as abuse, neglect, bullying, or exposure to war (Cicchetti & Valentino, 2006; McLaughlin, 2016; Nelson & Gabard-Durnam, 2020). These often co-occurring experiences require young people to adapt their psychological, social, and neurobiological functioning, and the strategies they employ may increase the risk for later-life victimization and psychopathology (Brown et al., 2019; Clark et al., 2010; Danese & McEwen, 2012; Lupien et al., 2009; Widom et al., 2008).

Two predominant approaches to *operationalize* childhood adversity are the cumulative risk and dimensional models of adversity. The quantitative, cumulative risk approach aggregates the number of distinct adverse experiences into a single cumulative risk or adverse childhood experiences (ACEs) score (Evans et al., 2013; Felitti et al., 1998). This approach has gained wide acceptance in public policy and clinical practice due to its straightforward calculation, interpretability, and predictive power for group-level health outcomes (Lacey & Minnis, 2020). However, it has faced criticism for its limited accuracy in predicting individual health risks, variability in prediction accuracy based on the reporter, and its failure to account for critical features of adversity, such as type, severity, chronicity, and developmental timing (Baldwin et al., 2021; Choi et al., 2023; McLaughlin & Sheridan, 2016). These limitations are thought to hinder its ability to identify specific mechanistic risk pathways that could inform targeted intervention. Alternatively, the qualitative, dimensional approach aims to specify mechanistic pathways linking core dimensions of adversity (threat/harshness, deprivation, and unpredictability) to later-life health outcomes (Berman et al., 2022; McLaughlin et al., 2014, 2021). While this more sophisticated approach enables the assessment of how mechanistic pathways vary with features of exposure, including frequency and severity, challenges remain to be addressed regarding the conceptualization, operationalization, and measurement of these dimensions (Berman et al., 2022; McLaughlin et al., 2021).

Recognizing the value of both approaches (McLaughlin et al., 2021; K. E. Smith & Pollak, 2021), **Chapters 4 and 5** utilized these frameworks to assess friendship

buffering of neural and psychological stress responses in British young people with childhood adversity. To integrate both approaches, a principal component analysis was applied to a range of retrospectively self-reported childhood adversity measures (see Brieant et al. (2024) for an in-depth overview of leveraging multivariate approaches to operationalize childhood adversity). In line with dimensional models of adversity (McLaughlin & Sheridan, 2016), this dimensionality reduction technique identified two components resembling threat and deprivation experiences, which were subsequently used to compute dimensional scores. These scores were also combined into a cumulative childhood adversity index, weighted by their explained variance, with higher scores indicating more severe adversity.

One objective of **Chapter 4** was to investigate whether greater friendship quality was associated with reduced frontolimbic reactivity to acute stress. Results indicated that high-quality friendships were linked to reduced left hippocampal reactivity to acute stress in young people with childhood threat experiences. While this interaction effect did not survive correction for multiple comparisons, it underscores the value of assessing the severity of different adversity dimensions for specifying the neural mechanisms potentially underlying psychopathology risk (Cohodes et al., 2021; McLaughlin, Weissman, et al., 2019; Puetz et al., 2020). In **Chapter 5**, the focus shifted toward investigating the buffering effects of friendships on mental health symptoms before and during the COVID-19 pandemic. While no specific hypotheses were proposed regarding different dimensions of childhood adversity, uncorrected exploratory analyses – reported in the supplementary materials – revealed noteworthy findings. Cumulative childhood adversity and deprivation-specific, but not threat-specific, experiences were negatively associated with friendship quality, with more severe adversity linked to lower perceived friendship support. In contrast, threat-specific, but not cumulative or deprivation-specific, experiences were positively associated with anxiety and depressive symptoms, with more severe threat exposure linked to increased symptomatology. Together, these uncorrected findings highlight the value of integrating both cumulative and dimensional approaches when investigating health and developmental consequences following childhood adversity.

Little consensus exists regarding how to ideally *measure* childhood adversity, partly due to variation in measurement approaches across studies. For practical reasons, such as costs and time efficiency, most empirical research (incl. **Chapters 4, 5, 6**) relies on retrospective self-reports to capture individuals' subjective appraisals and memories of past experiences. Evidence from meta-analyses and cohort studies suggests that subjective, self-reported perceptions of childhood adversity are more strongly associated with psychopathology risk than objective, court-substantiated experiences (Danese & Widom, 2020; Francis et

al., 2023). Furthermore, meta-analytic findings from Baldwin et al. (2019) indicate that prospective and retrospective measures of childhood adversity identify largely distinct groups of individuals, each with differential risk pathways to psychopathology.

To address some of these challenges, researchers have recommended tools that differentiate between dimensions of environmental experiences, account for participants' developmental stage, and incorporate input from multiple reporters, both prospectively and retrospectively (Berman et al., 2022; E. S. Young et al., 2020). These research-oriented recommendations are particularly valuable for guiding novel data collection efforts and interpreting previously collected data. The empirical studies presented in this dissertation (**Chapters 4, 5, 6**) employed multiple measures of childhood adversity and psychosocial functioning, enabling a more accurate and reliable capture of the complexity and multidimensionality of these constructs.

A crucial next step is the development of robust and culturally sensitive tools to accurately identify vulnerable young people at greatest risk of psychopathology and, therefore, most in need of intervention (Danese, 2020). This would mark a critical advancement in screening practices, reduce barriers to care, and advance the identification of specific mechanistic risk pathways linking childhood adversity and psychopathology, alongside protective factors that buffer against psychopathology risk. A concrete example of how such a clinically useful tool could be operationalized is provided by S. J. Lewis et al. (2019), who utilized data from a population-representative UK cohort study of young people to investigate psychosocial and clinical risk factors for psychopathology following adversity exposure. One key, preliminary finding highlights the potential of leveraging these factors to robustly improve individualized risk stratification, representing an important step toward understanding and accounting for individual differences in response to adverse experiences.

**Towards Generalizability and Cultural Sensitivity**

Selecting robust, accurate, and reliable measures remains a scientific challenge and necessity to ensure that findings generalize to real-world experiences. Self-report measures are known to be susceptible to recall and social desirability biases (Fadnes et al., 2009; Jordan & Troth, 2020; Latkin et al., 2017), while standardized laboratory-based paradigms are often criticized for lacking ecological validity (S. S. Dickerson & Kemeny, 2004). The prospective longitudinal study presented in **Chapter 5** leveraged a real-world stressor (i.e., COVID-19 pandemic) to examine friendship stress buffering, providing unique insights into how young people with childhood adversity adapt during acute, real-life stress exposure. To build on these findings, future research could integrate experience sampling methodology (ESM) to assess friendship support and stress

responses both inside and outside the laboratory. For example, Vaessen et al. (2023) examined neural stress responses during the Montreal Imaging Stress Task (MIST) alongside daily-life stress and affect using ESM. Consistent with the findings presented in **Chapter 4**, the MIST elicited limbic reactivity, which was associated with higher overall daily stress ratings, supporting its ecological validity in assessing stress responses (Vaessen et al., 2023). Regarding friendship support, the perceived quality of support may not always align with the actual support received (Haber et al., 2007). While research suggests that perceived, rather than actual received, support is a stronger predictor of mental health outcomes (McDowell & Serovich, 2007), future research could benefit from assessing received support in real-life situations, taking into account the context and need for support (Melrose et al., 2015).

Large, longitudinal, publicly available data sets, such as the Adolescent Brain Cognitive Development (ABCD) Study (Casey et al., 2018) or the Environmental Risk (E-Risk) Longitudinal Twin Study (Fisher et al., 2015), offer powerful resources to replicate and expand the empirical findings presented in this dissertation at both the individual and group levels (Kievit et al., 2022). Specifically, these substantially larger samples increase statistical power, thereby expand analytical flexibility to investigate the complex interplay between different features of adverse experiences (Brieant et al., 2024), a range of stress-regulatory systems (Ungar et al., 2023), multiple protective factors (Fritz, Fried, et al., 2018), and the dimensional nature of psychopathology (Lahey et al., 2012; Parkes et al., 2021).

Furthermore, leveraging rich secondary data sets can substantially improve the capacity to systematically study cross-cultural effects and diverse demographics, allowing for more nuanced insights into the sociocultural and policy-driven lived experiences of young people (Nketia et al., 2021; Saragosa-Harris et al., 2022). Whether the empirical findings presented in this dissertation (**Chapters 4, 5, 6**) – derived from predominantly female, white, and well-educated samples of young people living in the UK and the Netherlands – can be generalized to populations with vastly different sociocultural and contextual characteristics remains an open question.

Increasingly, there have been calls for the adoption of culturally and contextually sensitive approaches to improve the replicability and generalizability of research on risk and resilience following trauma exposure (Fried et al., 2018; Ungar et al., 2023). This is particularly timely given the anticipated demographic shifts in the Global South and the diverse experiences of adversity they entail. For example, by 2050, over one-third of the world's young people aged 15 to 24 years are projected to live in Africa (United Nations Department of Economic and Social Affairs, Population Division, 2022; D. Walsh & Morales, 2023). Meanwhile, in 2024,

prevalence estimates in sub-Saharan Africa suggest that approximately 72% of females and 82% of males aged 18-24 years have experienced at least one form of childhood adversity (Amene et al., 2024), rates that are more than three times higher than the 22.6% average prevalence estimated across 28 European countries (K. Hughes et al., 2021). To address the global burden of adversity and ensure that interventions are equitable, effective, and globally relevant, future research must therefore prioritize understanding the protective factors and mechanisms underlying risk and resilience within diverse samples (Ghai, 2021).

**Concluding Remarks**

In a world where childhood adversity remains a pervasive public health emergency with profound and long-lasting health and developmental consequences, understanding and leveraging the protective power of friendships presents a promising pathway toward building resilience in vulnerable youth. This dissertation set out to investigate the stress-related mechanisms through which social support, particularly friendships, buffer against victimization and psychopathology risk in young people with childhood adversity. Across five chapters, insights are presented from literature reviews (**Chapters 2, 3**), cross-sectional analyses (**Chapters 4, 6**), and longitudinal analyses (**Chapter 5**), demonstrating that childhood adversity is a potent risk factor for social thinning, victimization, and both internalizing and externalizing psychopathology. Conversely, friendship support emerged as a critical protective factor capable of reducing perceived stress and subsequently lowering internalizing symptoms. To more effectively inform targeted, equitable, and sustainable preventative interventions for young people with childhood adversity, future interdisciplinary research should adopt a complexity theory approach, capturing the intricate and dynamic interplay between psychological, social, and neurobiological systems over time, ideally through large, prospective longitudinal studies with diverse samples.

# Appendices

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## Dutch Summary (*Nederlandse Samenvattin)*

### Vriendschap als buffer tegen stress bij jongvolwassen met ingrijpende jeugdervaringen

Ongeveer 60% van de kinderen en adolescenten maakt ten minste één ingrijpende jeugdervaring mee, zoals mishandeling, verwaarlozing, ouderlijke psychische problemen, armoede, pesten of oorlog. Langdurige en herhaalde blootstelling aan dergelijke stressvolle en mogelijk traumatische ervaringen, vooral tijdens gevoelige ontwikkelingsfases, verhoogt het risico op zowel daderschap als slachtofferschap en op diverse mentale problemen later in het leven. Maar deze ervaringen en gerelateerde gezondheidsproblemen zijn te voorkomen. Daarom is het belangrijk om te onderzoeken welke risico- en beschermende factoren een rol spelen bij de impact van ingrijpende jeugdervaringen.

Theoretische modellen stellen dat neurocognitieve aanpassingen als gevolg van zulke ervaringen het risico op slachtofferschap en psychische kwetsbaarheid verhogen via hun invloed op sociaal functioneren. Het neurocognitieve sociaal-transactionele model van psychische kwetsbaarheid (geïntroduceerd in **Hoofdstuk 1**) stelt bijvoorbeeld dat deze aanpassingen kunnen leiden tot sociale uitdunning (minder beschermende relaties) en stressgeneratie (meer stressvolle interpersoonlijke ervaringen), met verhoogde kwetsbaarheid tot gevolg.

Belangrijk is dat jongeren die steun van vrienden ervaren, een verminderd risico lopen op slachtofferschap en psychische kwetsbaarheid. Dit fenomeen heet stress-buffering. Het is nog grotendeels onduidelijk welke mechanismes het stress-bufferende effect van vriendschap verklaren. De literatuur over sociale stressbuffering suggereert dat de aanwezigheid van steunende partners kan zorgen voor minder sterke waarnemingen, reacties en fysiologische processen bij acute stress, wat leidt tot een lagere stressrespons en betere gezondheid.

Voortbouwend op dit kader onderzoekt dit proefschrift de psychologische, cognitieve en neurobiologische mechanismes waarmee sociale steun, met name vriendschap, het risico op slachtofferschap en psychische kwetsbaarheid bij jongeren met ingrijpende jeugdervaringen kan verminderen. Hiervoor is gebruikgemaakt van literatuuronderzoek, en van cross-sectionele en longitudinale analyses met gedragsdata en hersenscans.

Het eerste deel van dit proefschrift laat zien hoe ongunstige (maladaptieve) neurocognitieve en sociale aanpassingen na kindermishandeling het risico verhogen om later slachtoffer of dader te worden. **Hoofdstuk 2** bespreekt de literatuur over de spiraal van geweld, met de link tussen kindermishandeling en slachtofferschap binnen en buiten het gezin. Drie mechanismes en de mogelijke

beschermende rol van sociale steun worden besproken. Slachtofferschap binnen het gezin wordt besproken in het kader van de hypothese van intergenerationele overdracht van mishandeling, met twee perspectieven: slachtoffer-naar-pleger (waarbij slachtoffers later zelf mishandelen) en slachtoffer-naar-slachtoffer (waarbij kinderen van slachtoffers zelf ook slachtoffer worden, zelfs als de ouder geen dader is). Slachtofferschap buiten het gezin wordt besproken in het kader van de hypothese dat geweld geweld voortbrengt.

In lijn met modellen van latente kwetsbaarheid en adaptieve kalibratie worden drie mechanismes besproken die verminderde sociale functie en geweldpleging kunnen verklaren: (1) sterkere aandachtsvertekening (bias) voor dreiging; (2) zwakkere beloningsverwerking en een verminderd vermogen om te leren van feedback; (3) zwakkere emotieregulatie. Een sterkere dreigingsbias kan adaptief zijn in een bedreigende omgeving, maar in veilige contexten leidt dit tot maladaptief gedrag zoals vijandige intenties toeschrijven aan anderen, wat samenhangt met agressie, vermijding, minder sociaal functioneren en verhoogde kwetsbaarheid. Het hoofdstuk laat zien dat veilige, stabiele sociale steun beschermt tegen slachtofferschap en mentale problemen, mogelijk via invloed op deze drie risicomechanismes. Om de spiraal van geweld te doorbreken, is echter meer kennis nodig over hoe ingrijpende jeugdervaringen en sociale steun neurocognitieve processen beïnvloeden.

Het tweede deel van het proefschrift richt zich op mechanismes die vriendschap koppelen aan verminderde psychische kwetsbaarheid bij jongeren met ingrijpende jeugdervaringen. **Hoofdstuk 3** beschrijft een systematisch literatuuronderzoek naar de vraag of vriendschappen neurale stressreacties verminderen bij jongeren met zulke ervaringen. Het literatuuronderzoek richtte zich op Engelstalige empirische studies bij jongeren (gemiddeld 10–24 jaar) met ingrijpende jeugdervaringen. Vriendschap moest binnen dit leeftijdsbereik zijn beoordeeld en neurale stressreacties gemeten met MRI. Van de 4.297 zoekresultaten en 66 beoordeelde artikelen voldeden slechts twee studies aan de criteria. Twee extra studies werden toegevoegd na het verbreden van de criteria door ook andere neurobiologische stresssystemen mee te nemen. Slechts twee van deze vier studies onderzochten direct of vriendschappen stressreacties bufferen. In een steekproef van geïnstitutionaliseerde jongeren vond Tang et al. (2021) dat vriendschapskwaliteit op 12-jarige leeftijd het effect van maladaptieve stressfysiologie op problemen met leeftijdsgenoten op 16-jarige leeftijd kon verminderen. Daarentegen vond Fritz et al. (2020) in een kleine steekproef van goed functionerende jongeren geen verband tussen vriendschap en gedrag of hersenactiviteit bij sociale afwijzing. Deze bevindingen onderstrepen dat meer onderzoek nodig is naar het effect van vriendschap op neurobiologische stressreacties bij jongeren met ingrijpende jeugdervaringen.

**Hoofdstuk 4** onderzocht of waargenomen vriendschapskwaliteit samenhangt met betere mentale gezondheid en verminderde neurale stressreacties bij jongeren met ingrijpende jeugdervaringen. Hiervoor werden gedrags- en fMRI-data geanalyseerd van het *Resilience After Individual Stress Exposure* (RAISE) onderzoek, waaraan 102 jongeren (16–26 jaar) in het Engeland deelnamen. Hoewel geen bewijs werd gevonden voor sociale uitdunning na ingrijpende jeugdervaringen, was er een sterke associatie tussen hoge vriendschapskwaliteit en betere mentale gezondheid. Bij een representatieve subset van 62 jongeren werd fMRI-data verzameld tijdens een taak die acute stress opwekte. Deze stress verhoogde angst en neurale activiteit in vijf frontolimbische hersengebieden. Een zwakke interactie werd gevonden tussen dreigingservaringen en vriendschapskwaliteit die hippocampusactiviteit voorspelde. Specifiek nam deze hersenactiviteit toe bij ernstigere dreigingservaringen en lage vriendschapskwaliteit. Dit effect was echter niet significant na correctie voor meervoudige vergelijkingen en vereist replicatie.

Hoewel COVID-19 de fMRI-dataverzameling van RAISE stillegde, bood deze collectieve stressor een unieke kans om dezelfde steekproef longitudinaal te volgen in het *Resilience after the COVID-19 Threat* (REACT) onderzoek. **Hoofdstuk 5** onderzocht het bufferende effect van vriendschap op mentale gezondheid vóór en tijdens drie tijdstippen in de pandemie. Via op afstand verzamelde gedragsdata werd mentale gezondheid vergeleken tussen de meting voor de pandemie, de eerste lockdown, de heropening en de tweede lockdown in Engeland. Angst symptomen piekte tijdens de eerste lockdown en normaliseerde daarna; depressieve symptomen bleven stijgen. Vriendschapskwaliteit was verhoogd tijdens beide lockdowns, maar keerde terug naar het niveau van vóór de pandemie tijdens de heropening. Sociale uitdunning werd waargenomen: ernstiger jeugdtrauma hing samen met lagere vriendschapskwaliteit. Over alle metingen heen was hogere vriendschapskwaliteit geassocieerd met lagere angst en depressieve symptomen. Vriendschapskwaliteit vóór de pandemie beschermde de mentale gezondheid tijdens de pandemie door waargenomen stress te verminderen.

Ten slotte onderzocht **hoofdstuk 6** of vriendschap kan zorgen voor minder stress na ingrijpende jeugdervaringen door cognitieve patronen te veranderen en zo de mentale gezondheid te bevorderen. Geïnspireerd door het neurocognitieve sociaal-transactionele model van psychische kwetsbaarheid, onderzocht dit hoofdstuk of vriendschap de mentale gezondheid bevordert bij jonge mensen met ingrijpende jeugdervaringen door de specificiteit van positieve autobiografische herinneringen aan vriendschap te beïnvloeden, wat vervolgens de ervaren stress zou kunnen verminderen. Dit onderzoek analyseerde zowel kwantitatieve als kwalitatieve cross-sectionele-gedragsdata van de eerste 100 deelnemers van het *Towards Health and Resilience in Volatile Environments* (THRIVE) onderzoek,

een lopend longitudinaal onderzoek naar jonge mensen van 18-24 jaar in Nederland, die retrospectief een laag tot gematigd niveau van jeugdtrauma hebben gerapporteerd. Dit onderzoek laat zien dat ernstiger jeugdtrauma geassocieerd was met sociale uitdunning, terwijl vriendschap juist samenhing met lagere scores op zelf-rapportages van stress en depressieve symptomen. Anders dan verwacht, was de specificiteit van positieve autobiografische herinneringen aan vriendschap niet geassocieerd met sociale steun uit vriendschap. Deze resultaten, samen met de longitudinale bevindingen beschreven in het vorige hoofdstuk, suggereren dat vriendschap de mentale gezondheid van jonge mensen met ingrijpende jeugdervaringen kan bevorderen door stress te verminderen, in plaats van door de verwerking van autobiografische herinneringen te beïnvloeden.

Samengevat markeert dit proefschrift een belangrijke stap naar een meer genuanceerd begrip van de psychologische, cognitieve en neurobiologische mechanismes waardoor sociale steun, met name vriendschap, het risico op slachtofferschap en mentale problemen kan verminderen bij jongeren met ingrijpende jeugdervaringen.

**Conclusies**

In een wereld waar ingrijpende jeugdervaringen een gezondheidscrisis vormen vanwege de ernstige en langdurige gevolgen voor de gezondheid en ontwikkeling, biedt het begrijpen en benutten van de beschermende kracht van vriendschappen een veelbelovende weg naar het opbouwen van veerkracht bij kwetsbare jongeren. Dit proefschrift heeft als doel de stress-gerelateerde mechanismes te onderzoeken waarlangs sociale steun, met name vriendschappen, het risico op gedragsproblemen en psychische kwetsbaarheid bij jonge mensen met ingrijpende jeugdervaringen vergroot. In vijf hoofdstukken worden inzichten gepresenteerd uit literatuuronderzoeken (**Hoofdstukken 2, 3**), cross-sectionele analyses (**Hoofdstukken 4, 6**) en longitudinale analyses (**Hoofdstuk 5**), die laten zien dat ingrijpende jeugdervaringen een krachtige risicofactor zijn voor sociale uitdunning, slachtofferschap en zowel internaliserende als externaliserende problemen. Vriendschap bleek daarentegen een belangrijke beschermende factor die stress kan verlagen en vervolgens internaliserende symptomen kan verminderen. Om een bijdrage te kunnen leveren aan gerichte interventies voor jonge mensen met ingrijpende jeugdervaringen, zou toekomstig interdisciplinair onderzoek een benadering vanuit de complexiteitstheorie moeten hanteren, waarbij de complexe en dynamische wisselwerking tussen psychologische, sociale en neurobiologische systemen wordt onderzocht, bij voorkeur door middel van grote, prospectieve longitudinale studies met representatieve steekproeven.

**Curriculum Vitae**

Maximilian König (né Scheuplein) was born on September 2nd, 1995, in Frankfurt am Main, Germany.

In 2014, he completed his Abitur (high-school degree) at Carl-Schurz-Schule in Frankfurt am Main and began his BSc in Psychology at Goethe University Frankfurt that same year. During his undergraduate studies, he worked as a Research Assistant in Prof.dr. Melissa Lê-Hoa Võ's lab, contributing to research on visual cognition. Concurrently, he founded and led *Hidden Stories*, a local non-profit under Enactus Frankfurt, supporting community integration of refugees during the European migrant crisis.

In 2015, Maximilian expanded his research experience as a Visiting Scientist at King's College London, working with Prof.dr. Eva Loth on the EU-AIMS Longitudinal European Autism Project. This interdisciplinary project investigated treatment approaches and risk factors related to brain development and social behavior in children, adolescents, and adults with autism spectrum disorders.

In 2018, he earned his MSc in Cognitive Neuroscience with distinction at University College London, working with Prof.dr. Sarah-Jayne Blakemore to study social cognitive development during adolescence. At the same time, he supported the MYRIAD project as a Data Collection Assistant, investigating the effects of mindfulness training on adolescent mental health.

From 2018 to 2020, Maximilian pursued his interests in reinforcement learning and developmental neuroscience as a Graduate Research Assistant and Lab Manager, first in Dr. MaryAnn Noonan's lab at the University of Oxford, and later in Prof.dr. Catherine Hartley's lab at New York University (NYU). At NYU, he also developed a passion for translating research into public health policy and engaged with the New-York based *Science Action and Advocacy Network* to work on social justice issues, particularly juvenile justice reform.

In 2020, Maximilian began his PhD in Developmental Psychopathology at Leiden University under the supervision of Prof.dr. Anne-Laura van Harmelen. He also participated in the interdisciplinary Social Resilience and Security Program, which seeks to understand and address transgressive behaviors in society. As the founder and co-director of *Resilient Minds*, Maximilian organized 38 international meetings with researchers from seven countries, fostering collaboration in stress resilience and developmental psychopathology research. Additionally, he served as co-chair of the Trainee Committee and the LGBTQIA+ Affinity Group of the Flux Society for Developmental Cognitive Neuroscience.

For a full CV, visit: www.mxkoenig.com.

**List of Publications**

***Journal Articles*** (*indicates joint first authorship)

9. **König, M.**, Smith, A. J., Moreno-López, L., Davidson, E., Dauvermann, M., Orellana, S., McCormick, E. M., Peris, T. S., Kaser, M., Ioannidis, K., & van Harmelen, A-L. (2025). Friendship buffering effects on mental health symptoms before and during the COVID-19 pandemic: A UK longitudinal study of young people with childhood adversity. *Development and Psychopathology, 1-16*.
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2. Vermeulen, S., **Scheuplein, M.**, van Harmelen, A-L., & Alink, L. (2023). Kindermishandeling: Impact op slachtoffer en maatschappij. *Handboek Slachtoffers, 1*, 157-167.
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Preprint | Open Access Resources

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