

The sequelae of childhood maltreatment:  
a multi-level longitudinal investigation of  
brain structure, symptomatology and social  
support

Philip Kelly

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## **Executive summary**

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## Background

Childhood maltreatment is associated with a wide range of adverse outcomes across the lifespan, including significant relationships with poor physical health, educational attainment, economic outcomes, and most notably an increased risk towards the emergence of psychopathology in adolescence and adulthood. Not all individuals who have experienced maltreatment will go on to develop psychopathology/elevated psychiatric symptomatology, as the pathway from maltreatment is probabilistic and not deterministic. Understanding the multifinality of maltreatment is vital for academics and clinicians to provide targeted, effective, and preventative interventions for this group at high-risk of poor outcomes. Developmental psychopathologists have called for the inclusion of ‘multi-level investigations’ which meaningfully integrate multiple levels of functioning, such as on a neural, behavioural and social level, to understand the risk and resilience factors that may characterise these pathways. Social support is one of a number of factors that is proposed to engender resilience against poor outcomes following maltreatment. Three hypotheses have been proposed that attempt to explain how social support interacts with the experience of maltreatment: as a direct protective factor, as a moderator, and as a mediator. However, the existing literature within this domain has been reported to be ambiguous and in some cases contradictory. This thesis aims to investigate the sequelae of childhood maltreatment and further characterise the multifinality of maltreatment across multiple levels of functioning and risk and resilience factors.

## Systematic review

To date, no systematic review has attempted to synthesise the available literature to evaluate the evidence for the role of social support in the emergence of internalising disorders in maltreated individuals. The current breadth of research is vast, but the consensus regarding how it supports the three hypotheses of how social support interacts with the experience of maltreatment is unclear. Therefore, the systematic review aimed to synthesise the existing literature of the role of social support in the emergence of internalising symptomatology in maltreated individuals under the theoretical structure of the three proposed hypotheses.

### Method

Online bibliographic databases were systematically reviewed in January 2019 and a total of 21 studies met the inclusion criteria. 14 studies were included in the meta-analysis regarding social support as a direct protective factor. 7 studies each were included in the narrative synthesis of results regarding the role of social support as a mediator and a moderator due to the heterogeneity in the design of the studies, analytic process, and reporting of statistics.

### Results

Meta-analytic evidence indicated that social support was a reliable protective factor across these studies, of small effect size, consistent across the lifespan and regarding the source of the support. The role of social support as a mediator was largely supported by the narrative synthesis, however this appeared to vary by the source of social support and was of a small effect. However, there was not reliable evidence to

support the view that social support moderates the relationship between maltreatment experience and internalising symptomatology. Concerns were raised regarding the heterogeneity of how social support and maltreatment experience was conceptualised and operationalised.

### *Implications and conclusion*

The review highlighted the importance of social support as resilience factor in the protection against the emergence of internalising symptomatology. However, the way social support engenders this protective effect is complex beyond being a direct protective factor. While the current research supports the view that social support may act as a mediator in developmental pathways from maltreatment to internalising symptomatology, the underlying mechanistic processes are unclear. These findings provide clear impetus for researchers to start to map out what these intermediary and underlying processes are. In turn this may contribute to clarifying and strengthening conceptualisations of the poorly defined construct of social support. In general, this research has implications for the use systemic approaches with maltreated individuals, considering how these individuals may elicit, respond to, and seek out social support from family and peers.

## **Empirical study**

Existing research has identified a broad pattern of structural differences within the brain associated with maltreatment experience, namely relating to grey matter volume. Grey matter volume is purported to be a broad metric of cortical structure, determined by several underlying indices (cortical thickness, surface area, local gyrification) that have distinct genetic and developmental trajectories. Existing

literature has pointed to the separable effects of maltreatment on these fine-grained indices which provides a convincing rationale for the investigation of these cortical characteristics concomitantly. One limitation of the current research is that is predominantly limited to cross-sectional designs, thus restricting the inferences one can make regarding the importance of atypical structure associated with maltreatment on behaviour and symptomatology. Furthermore, few studies have integrated social support, a noted resilience factor, into the mapping of the predictive nature of maltreatment related cortical differences to later symptomatology. The aims of the study were to systematically investigate the predictive nature of structural differences associated with maltreatment on future symptomatology in maltreated adolescents, compared to non-maltreated counterparts. The subsequent objective was to examine whether this relationship was moderated by social support.

### Method

Thirty-three children with documented maltreatment and thirty-three matched controls underwent a structural MRI of their brain, alongside completing a battery of questionnaires relating to psychiatric symptomatology and current social support (T1). Two years following the MRI (T2), the participants completed the psychiatric symptomatology questionnaires again.

### Results

The imaging analysis demonstrated a broad pattern of reductions in local gyrification associated with maltreatment, including in the superior frontal, superior parietal, fusiform and inferior frontal regions. A cortical thickness increase within the maltreated group was detected within the caudal middle frontal region compared to

the non-maltreated group, while there no group differences detected in cortical surface area after correcting for multiple comparisons. Structural gyrification values within the superior parietal region were found to negatively correlate with future anxiety scores (T2) whilst controlling for T1 scores within the maltreated group. Furthermore, frequency of social support was found to moderate this relationship, however in a direction that was not predicted; greater frequency of social support was found to moderate the association between structure and symptomatology for individuals with similar levels of gyrification to the non-maltreated group, and not for those with the greatest differences to the non-maltreated group. An unexpected supplemental finding that was of great interest was detected; that maltreatment severity was found to negatively correlate with social support importance and frequency.

#### Implications and conclusions

Atypical gyrification within the superior parietal region associated with maltreatment may be predictive of later symptomatology, though this was of small effect.

Furthermore, social support may be the most beneficial in protecting against later symptomatology to those who present with the smallest cortical structural differences associated with maltreatment. Regional differences broadly were not associated with symptomatology at T1 or T2, nor did social support present strong moderation effects. This suggests that we may be asking the wrong questions in the relationship between cortical structure, social support, and symptomatology within maltreated individuals, and a greater focus on intermediary processes is needed to delineate the functional importance of the reported structural differences associated with maltreatment exposure. The dose-response relationship between in-home maltreatment severity and out-of-home and in school peer support has clear clinical implications within the



school environment and suggests that individuals with the greatest abuse may act in ways that impacts how social support is evoked.

## Integration, impact and dissemination

### Integration

The two chapters had clear underlying themes running through them, particularly focussed on understanding the role of social support in maltreated individuals. As such several unified inferences could be made from the findings:

- As we cannot infer whether the detected structural differences represent adaptations that represent effective coping strategies, or maladaptive calibrations that confer greater risk of psychopathology, future research is needed to distinguish neural underpinnings of risk and resilience within the brain.
- Results from both chapters indicated that the ‘buffering hypothesis’, how social support may moderate the effect of maltreatment, may not sufficiently capture the complexities of social support for maltreated individuals for it to be clinically valuable.
- Outcomes from the empirical study and review pointed towards the importance of social support as a mediator in the multifinality of maltreatment, and potential candidate mechanisms for ecologically valid measures of social interaction as intermediary processes were suggested (e.g. trust, mentalizing, emotional regulation)

- The great amount of heterogeneity in the social support measures in the review alongside little evidence for social support as a protective factor in the empirical paper may suggest that social support is a poorly conceptualised and operationalised construct, in line with existing literature.

## Impact

A few key beneficiaries, among others are noted below:

- Academic
  - There is clear value in exploring separable indices of cortical structure which indicate distinct atypical structure associated with maltreatment
  - Intermediary processes between structure and symptomatology and ecologically valid measures of social interaction in maltreated individuals are vital steps in delineating the multifinality of maltreatment and represents clear candidates for future research.
  - Longitudinal designs are integral to effectively characterising the developmental pathways from maltreatment to poor mental health outcomes.
- Clinician based
  - Drop-out and difficulties in engagement in therapy have been extensively noted within maltreated populations. The findings indicate that these individuals may behave in certain ways that may impact socialised support, and clinicians should prioritise consideration of ‘relationship to help’ for these clients.

- Social workers
  - o The findings indicated that social care assessments should consider how a maltreated child may need support, given that elevated trauma may impact the way they elicit social support, and that a lack of social support is not simply a deficit to fill.
- General public
  - o Public interest in the brain and neuroimaging is substantial, while critique and understanding of findings is low. Elevated interest will impact the public's understanding of the impact of maltreatment and the probabilistic nature of poor outcomes, but the way it is disseminated needs to be appropriate and proportionate.

### Dissemination

It is planned that the systematic and empirical study are made broadly available to a range of audiences through specific routes including publication in academic journals chosen for their clinical and academic audiences, spoken and poster presentations at research and clinical, national and international conferences over the next year, and presentation to interdisciplinary teams within the current course placement and upcoming psychiatry conferences. Service-user involvement will be vital in disseminating the findings to a wider non-clinical audience, by consulting on the language used, deciding the salient findings and how these are explained, and advising on appropriate channels to reach other service user groups. The outcomes from this service-user involvement will also support dissemination to the general public through social media, and proposed presentations to schools.



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**Systematic review:**

**What is the role of social support in relation to internalising disorders in individuals who have experienced childhood maltreatment?**

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## Abstract

**Background:** Social support has been identified as a prominent resilience factor that may protect against the emergence of internalising symptomatology in individuals who have experienced childhood maltreatment. How social support generates this protective effect has been hypothesised in three ways: as a direct protective factor, as a moderator, and as a mediator. However, to date, no systematic review has been conducted to evaluate the evidence for these three hypotheses in relation to the role of social support in the emergence of internalising disorders in maltreated individuals. Understanding how social support interacts with childhood maltreatment would better inform clinicians in the delivery of therapeutic and preventative interventions.

**Method:** Online bibliographic databases were systematically reviewed in January 2019 and a total of 21 studies met the inclusion criteria. 14 studies were included in the meta-analysis regarding social support as a direct protective factor. 7 studies each were included in the narrative synthesis of results regarding the role of social support as a mediator and a moderator. **Results:** Social support was found to be a consistent protective factor, of small effect size, for individuals with maltreatment histories across their lifespan, regardless of source of the support. The role of social support as a mediator was largely supported, however this appeared to vary by the source of social support and was of a small effect. This systematic review did not find reliable evidence to support the view that social support moderates the relationship between maltreatment experience and internalising symptomatology. The heterogeneity of how social support and maltreatment has been operationalised in extant studies is highlighted. **Conclusions and implications:** This research has implications for systemically focussed interventions that utilise social support, and provides impetus

for researchers to investigate the underlying mechanistic processes by which social support mediates the association between maltreatment experience and internalising symptomatology.

## Introduction

A wealth of studies continue to document the deleterious impact of maltreatment and abuse experienced within childhood on later physical health, social, economic and psychological outcomes (Gilbert et al., 2009; Jaffee, 2017). Subtypes of maltreatment, physical, emotional, sexual abuse, and neglect have been conceptualised as a ‘pathogenic relational experience’ (Cicchetti & Lynch, 1995), typically occurring between the child and their primary caregivers, that embeds an increased risk of psychopathology and other adverse outcomes (Valentino, 2017). A considerable body of evidence links maltreatment with a myriad of specific mental health disorders (Bernet & Stein, 1999; Comijs et al., 2013) and physical health difficulties (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Dong et al., 2004). As such it represents a salient societal concern, warranting systematic investigation of its sequelae, and the risk and resilience factors influencing outcome. Such investigation may help the development of effective and targeted interventions as well as with preventative strategies.

### Sequelae of maltreatment

Childhood maltreatment has been associated with a wide of adverse outcomes, both in adolescence and adulthood, and is as a predictor of educational, economic and employment outcomes (Currie & Widom, 2010; Metzler, Merrick, Klevens, Ports, & Ford, 2017). For example, individuals who have experienced maltreatment are at twice the risk of not being in education or employment by age 18 than those who had not experienced maltreatment (Jaffee et al., 2018). Some estimates indicate that the financial cost of childhood maltreatment, at a societal level, at approximately \$103.7



billion directly, and \$70.6 billion indirectly, within the United States (Wang & Holton, 2007).

### The concept of multifinality: Probabilistic developmental trajectories to psychiatric outcomes

Understanding the multiple pathways towards an outcome (equifinality) and the multiple pathways towards different outcomes (multifinality) informs an ‘emergent interdisciplinary science’ in developmental psychopathology (Cicchetti & Rogosch, 1996). Multifinality is pertinent within the field of childhood maltreatment, given that not all maltreated individuals will go on to develop psychiatric disorders/symptomatology (McGloin & Widom, 2001), but there is an apparent significant minority who are at elevated risk of mental health difficulties across the lifespan. As such the risk of mental health problems is probabilistic rather than deterministic (Cicchetti, 2013; McCrory & Viding, 2015). Multifinality in this sense characterises how individuals, following childhood maltreatment, may respond or interact with vulnerability and protective factors at multiple levels of ecology to allow for diversity in developmental outcomes (Cicchetti & Rogosch, 1996).

As not all individuals who have experienced maltreatment will go on to experience psychopathology, it is of clinical value to understand the factors that confer *risk* and *resilience* in developmental trajectories towards normative function or psychopathology. Researchers have identified several putative mechanisms (mediators) and factors that serve to moderate the influence of maltreatment. Such mechanisms include variations within the domains of emotional regulation (Kim & Cicchetti, 2010), threat processing (Kelly et al., 2015), reward processing (Hanson et

al., 2015) and executive functioning (Mueller et al., 2010). Moderation of the relationship between maltreatment experience and psychopathology has been demonstrated on multiple levels of ecology, including at a genetic (Cicchetti & Rogosch, 2014) and physical level (McLaughlin, Alves, & Sheridan, 2014). One external factor that has received considerable attention as a putative mechanism and moderator in developmental trajectories of maltreatment is social support.

### Mental health sequelae of maltreatment: Internalising outcomes

Childhood maltreatment has been shown to be a robust risk factor for mental health problems in adolescence and adulthood (Cicchetti & Toth, 2005). The onset of psychopathology across the life course has been proposed to be attributable in nearly a third of cases to adverse childhood experiences (Kessler et al., 2010; McLaughlin et al., 2011). In particular, predicted attribution rates for maltreatment-related psychiatric disorders range from 22% to 32% among women and 20% to 24% for men (Afifi et al., 2008). The association between childhood maltreatment and internalising disorders has been well established, with increased risk for anxiety disorders (Keyes et al., 2012), major depressive disorder (Nanni, Uher, & Danese, 2012), PTSD and trauma symptoms (Brewin, Andrews, & Valentine, 2000) within adolescence and adulthood. Importantly, even when maltreated children do not present with diagnosable disorders, they often show elevated sub-threshold symptoms compared to non-maltreated peers (Cicchetti & Toth, 2005; Kearney, Wechsler, Kaur, & Lemos-Miller, 2010; Stewart, Livingston, & Dennison, 2008).

In the general population, anxiety disorders are found to be the most prevalent class of lifetime disorders (Kessler, Berglund, Demler, Jin, & Walters, 2005), and are

amongst the most well-researched within the field of adolescent and developmental psychopathology (Paus, Keshavan, & Giedd, 2008). Furthermore, recent NHS statistics report that emotional disorders were the most prevalent type of disorder experienced by 5 to 19 year olds in 2017 (8.1%), with the greatest increase in prevalence rates since 1999, compared to behavioural and hyperactive (externalising) disorders which have remained at similar prevalence rates over this period (NHS Digital, 2018). Given the extensive literature within this field and the greater prevalence rates of anxiety and affective (internalising) disorders, it is of academic and clinical value to systematically focus on understanding of the factors that characterise the pathways from childhood maltreatment towards the emergence of internalising disorders.

### Social support

Social support is a multidimensional concept that incorporates a range of interpersonal behaviours and attracts differing, but overlapping, definitions (Thoits, 1982). Typically social support can be delineated into three broad dimensions: social networks (breadth and frequency of a person's contact with others), perceived social support, and enacted support (practical and material support; Hyman, Gold, & Cott, 2003). Perceived social support may encapsulate the subjective belief that others are able to provide emotional and practical support within safe and nurturing relationships (Merrick, Leeb, & Lee, 2013). While others summarise perceived social support as the "cognitive appraisal of being reliably connected to others" (Barrera, 1986), enacted support may represent the 'reality' of this perception/belief of available support. Greater social networks may therefore allow for increased opportunities for

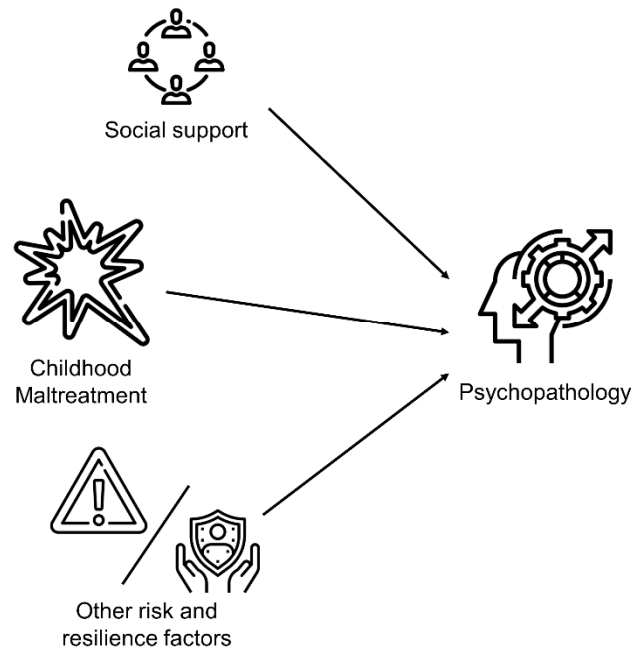
enacted support and thus influence the cognitive appraisal of opportunities for enacted support (Thoits, 1982), illustrating how the three domains may be inter-connected.

The association between social support and physical and mental health outcomes has been clearly documented within the general population (Holt-Lunstad, Smith, & Layton, 2010; Seeds, Harkness, & Quilty, 2010; Väänänen, Marttunen, Helminen, & Kaltiala-Heino, 2014), such that individuals with low levels of perceived social support and smaller social networks show greater risk towards the emergence of common mental health disorders (Brugha et al., 2005; Wang, Mann, Lloyd-Evans, Ma, & Johnson, 2018), and experience a more severe trajectory of anxiety and depressive disorders (Miers, Blöte, de Rooij, Bokhorst, & Westenberg, 2013). Investigations of social support in the context of maltreatment has been as expansive. Theories accounting for the role of social support in the emergence of psychopathology following adversity have postulated that it acts: (i) as a direct protective factor; (ii) as a mediator; (iii) and finally, as a moderator of maltreatment.

#### *Social support as a direct protective factor*

In line with the research within the general population, researchers have suggested that social support exerts a positive effect on well-being, regardless of experience of maltreatment, such that it represents a 'direct protective factor'. Therefore, the influence of social support may act in a cumulative manner alongside other interpersonal experiences, such as maltreatment, and risk and protective factors, to protect against the emergence of internalising psychopathology (**Figure 1**). This has been supported by a recent meta-analysis detailing the protection social support imparts

against depression in the general population (Gariépy, Honkaniemi, & Quesnel-Vallée, 2016).

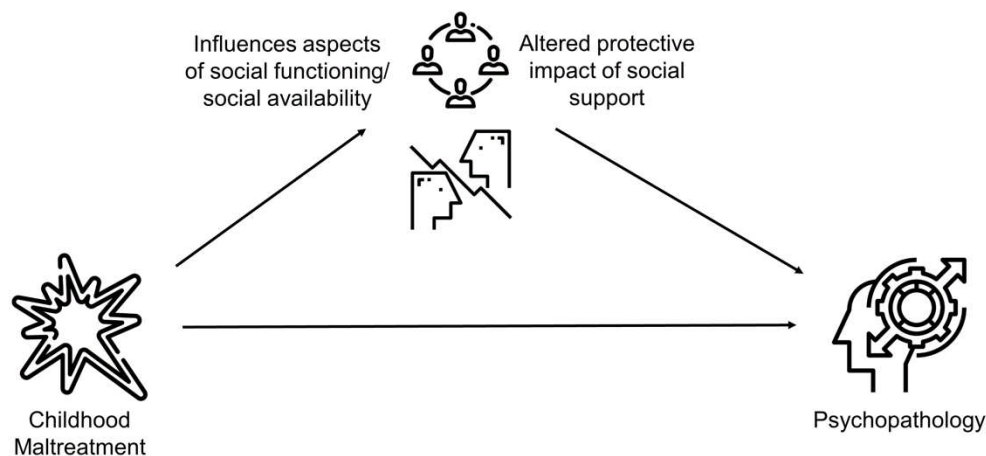


*Figure 1. Social support as a direct protective factor*

*Social support as a mediator*

Social support has also been proposed to play a role as a mediator in the developmental trajectories of trauma to psychopathology (Jaffee, 2017; Pepin & Banyard, 2006; Runtz & Schallow, 1997). A consistent finding in the research shows that childhood maltreatment is associated with lower levels of perceived social support (Horan & Widom, 2015; Lamis, Wilson, King, & Kaslow, 2014). Through potentially poor emotional recognition and regulation skills (Kim & Cicchetti, 2010; Perlman, Kalish, & Pollak, 2008) individuals may display behaviour that impacts on interpersonal functioning, such that they may act in an anxious, dismissive, avoidant, or aggressive manner (Darwish, Esquivel, Houtz, & Alfonso, 2001), leading to lower levels of social support. The degradation of available and perceived social support,

through these negative interactions, may therefore diminish the potentially positive effects of social support which may typically divert developmental trajectories away from psychopathology (**Figure 2**).

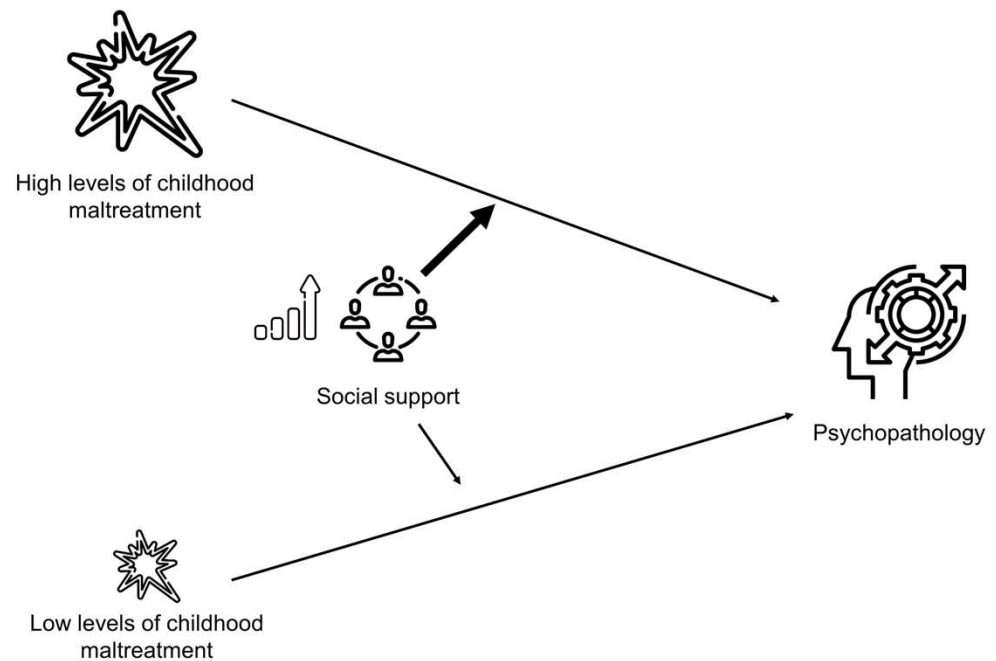


*Figure 2. Social support as a mediator*

*Social support as a moderator*

Cohen and Wills (1985) proposed a hypothesis such that social support acts as a ‘buffer’ to the detrimental impact of traumatic experiences, such as childhood maltreatment. This ‘buffering hypothesis’ states that high social support exerts a greater protective influence on the emergence of psychopathology in those who have experienced high levels of stress, including childhood maltreatment, such that symptomatology levels decrease to a level that are equivalent those who have not experienced stress. Conservative descriptions of this hypothesis state that social support *moderates* the severity of the maltreatment, such that the protective factor increases as severity increases (**Figure 3**), whereas non-maltreated individuals are

presumed to have low levels of symptomatology ‘regardless of their social support’ (Jaffee, 2017).



**Figure 3.** *Social support as a moderator*

To our knowledge, no previous research has systematically reviewed the current literature pertaining to these hypotheses within maltreated individuals; however existing literature reviews have briefly touched on the disparate evidence, notably highlighting the ambiguous and contradictory nature of current findings (Jaffee, 2017). Social support represents a compelling and tangible candidate for clinicians to integrate and harness in therapeutic and preventative interventions. To gain a clearer understanding how it interacts with childhood maltreatment is therefore of significant academic and clinical value.

## The current study

The current study aimed to analyse and synthesise existing quantitative research investigating the role of social support on the emergence of internalising symptomatology and disorders in adults and adolescents who have experienced childhood maltreatment. The review specifically aimed to investigate the role of social support as i) a direct protective factor, ii) as a mediator, and iii) as a moderator of the association of maltreatment on internalising symptomatology. Given the evidence indicating greater prevalence rates of anxiety and affective disorders across the lifespan following maltreatment, as well as current space limitations, this systematic review will focus specifically on investigating internalising symptomatology only. Furthermore, factors that characterise the experience of maltreatment, source of social support, developmental age and form of internalising symptomatology will be investigated and synthesised across studies.



## Method

This review was designed and conducted with reference to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2015). This study took place between December 2018 and March 2019.

### Search strategy

Relevant studies were identified by searching major internet-based bibliographic databases: PSYCINFO, Scopus, and PubMed. This was carried out between 17<sup>th</sup> - 20<sup>th</sup> January 2019. The search terms covered three main concepts; childhood maltreatment, social support and internalising psychopathology. The search terms and Boolean operators used are documented in **Table 1**. The search strategy for the databases included empirical papers, abstracts and dissertations. Bibliographies of previous reviews and retrieved articles were also searched for completeness. Following the initial searches of the databases, references were imported into 'Mendeley reference manager' and duplicates were removed. Screening underwent a three-stage process, titles were initially screened for eligibility, followed by abstracts, and subsequently full texts, excluding inappropriate studies at each stage. When reviewing full texts, reasons for exclusion were noted. Eligible texts were reviewed to identify further studies that were applicable to the review aim, and these were subsequently reviewed for eligibility. In order to ascertain the reliability of the search terms, key articles identified before the search were identified again through the systematic database searches.

*Table 1. Search terms used within the systematic review*

Concept	Search Terms
Childhood maltreatment	"Maltreatment AND child" OR "childhood maltreatment" OR "abuse AND child" OR "violence AND child" OR "childhood abuse" OR "child abuse" OR "childhood trauma" OR "physical abuse AND child" OR "sexual abuse AND child" OR "emotional abuse AND child" OR "neglect AND child" OR "domestic violence AND child" OR "domestic abuse AND child"
Social support	Social Support OR Friendship
Internalising psychopathology	"internalising" OR "internalizing" OR "anx*" OR "depress*" OR "ptsd" OR "post-traumatic stress" OR post-traumatic OR "panic" OR "phobia" OR "OCD" OR "obsessive-compulsive"

### Eligibility criteria

#### Study characteristics

The inclusion criteria for the studies in the present review were published, peer-reviewed journals. The location of the studies was restricted to Europe/America due

to cultural variations in the classification of maltreatment (Korbin, 1991; Raman & Hodes, 2012). Study designs were restricted to cross-sectional, longitudinal, and case-control studies; case studies were excluded. Publications were not restricted to a time frame.

### Analysis

Descriptive and qualitative studies without statistical analysis were excluded. Analysis between a maltreated sample and a control group, and studies that investigated variation within a maltreated group were included. Studies were included if primary analysis was specifically investigating the moderation, mediation, and correlation of social support with internalising disorders in the context of childhood maltreatment.

### Participant characteristics

Studies that investigated both sexes, or one sex (e.g. only female participants) were included. Studies were excluded if the sample were from an incarcerated or inpatient population, as social support perception and availability may be confounded by these restrictive and non-generalisable social environments. Community and clinical outpatient samples were included.

### Measurement of Maltreatment

Both self-report and file-report/officially documented forms of measurement for childhood maltreatment were included. Singular and multiple forms of maltreatment were included; sexual abuse, neglect, emotional abuse, physical abuse, domestic violence. Community violence, war trauma and pre-natal trauma as a form of

maltreatment were excluded due to variation and ambiguity in the inter-personal nature of the experience.

#### *Types of outcome measures*

Self-report and parent/teacher reported outcome measures on social support were included. Measures that investigated perceived or enacted social support independently or concomitantly were included. Exploring the independent impact of ‘perceived’ and ‘enacted’ social support was not within the scope of this systematic review.

Common forms of internalising disorders were included such as anxiety, depression and PTSD. However, a broad search strategy was employed to include other internalising disorders such as panic, phobias, and OCD. Measures that indicated symptomatology, diagnosis, or identified symptom clustering of internalising psychopathologies (e.g. Emotional difficulties & peer difficulties subscales of the Strength and Difficulties Questionnaire; Goodman, 1997) were included. Comorbidity of externalising disorder was not an exclusion term, if there was a presence of an internalising symptomatology. Postpartum depression, Bipolar, PTSD with a reasonable assumption that it is associated to trauma in adulthood, or other excluded traumas described in the previous section, were excluded. Other psychopathologies that do not clearly map onto the nosological internalising/externalising framework, such as schizophrenia and psychosis, were excluded. Studies that explored ‘post-traumatic growth’ and other scales of resilience as outcomes were excluded.

### Confounding variables

Studies in which individuals had comorbid neurodevelopmental (e.g. Autism, ADHD, specific and general learning disabilities) or neurological disorders, or traumatic brain injury, were excluded. Concurrent psychiatric medication was not an exclusion factor, neither were physical health difficulties/disorders.

### Data Collection and analysis

#### Data extraction process

Data was extracted from each article by the author. The following information was extracted from each study:

**Study characteristics:** aims and hypothesis of investigation, design.

**Sample characteristics:** sampled population (i.e. clinical or community), sample size, age, sex, ethnicity, location, socioeconomic indicators, presence of control group, inclusion/exclusion criteria noted or applied.

**Outcome measures and characteristics:** measure of social support and reported reliability and validity statistics; symptomatology measure used and reliability and validity statistics; Comorbid symptomatology measured, or other independent/dependant variables collected.

**Maltreatment measurement and characteristics:** measure used to assess maltreatment experience, source of information (e.g. self-report, file-report), characterisation of maltreatment (e.g. subtypes recorded, incidence of subtypes in sample).

*Analysis:* analytic procedure and statistics used; controlled for confounding variables, or whether used group matching.

*Outcomes of the study:* Findings of the study; relation to the stipulated theories of social support (i.e. moderation, mediation, direct protective factor); specific significance and effect statistics

#### Quality assessment of studies

An idiosyncratic quality assessment tool, adapted from the Mixed-Methods Appraisal Tool (Pluye et al., 2011) and The Quality Assessment Tool for Quantitative Studies (National Collaborating Centre for Methods and Tools, 1998), was designed for this systematic review to effectively reflect the aims of the review and characteristics of the population and variables of interest. The tool uses a six-item scale, across four areas (sampling strategy, data measurement, confounders, and analyses), in which studies are evaluated to satisfy the requirement within each area, and a response of yes/no is provided. Studies that were deemed to satisfy all criteria received a ‘strong’ score, those that satisfied four or more areas received a ‘moderate’ rating, and those that satisfied three or less ratings received a ‘weak’ rating. Weak studies would be reviewed on a case by case basis as to whether they should be excluded from the systematic review. The quality assessment tool is provided in Appendix 1.

#### Synthesis of results

Studies were synthesised based on the three hypotheses of how social support may exert influence following childhood maltreatment; as a direct protective factor, as a mediator, and as a moderator.

Due to the homogeneity in the reporting of zero-order correlations, a meta-analytic approach was undertaken to explore the strength of the correlation between social support and internalising symptomatology; reflecting social support as a ‘direct protective factor’. Subgroup analysis was undertaken to look at the strength of correlation within samples of maltreated individuals, and across samples of maltreated and non-maltreated individuals. Meta-regression was used to analyse mean age, and source of support, as a moderator. Analyses were performed with the Comprehensive Meta-Analysis 2.2 software (Borenstein, Hedges, Higgins, & Rothstein, 2005). A random effects model was used to analyse the data, as the studies were not believed to be functionally identical and thus a fixed effect model would not be appropriate (Hedges & Vevea, 1998). To address potential publication bias, funnel plots, Orwin’s Fail-safe N, Kendall’s Tau b, and the Duval and the Tweedie Trim and Fill method were used. The Fail-Safe N test can estimate the number of studies that would need to be collected to reduce the obtained effect size down to a value that has no practical significance. Begg and Mazumdar suggest that the rank correlation (Kendall’s tau b) between the treatment effect and standard error gives an approximation of the correlation between study size and effect size, and thus whether smaller studies are more likely to be included when they show a relatively large effect (Begg & Mazumdar, 1994). The Trim and Fill approach imputes effect sizes until the error distribution closely resembles normality, to offer a more unbiased estimate of effect size than the one obtained through meta-analysis (Duval & Tweedie, 2000).

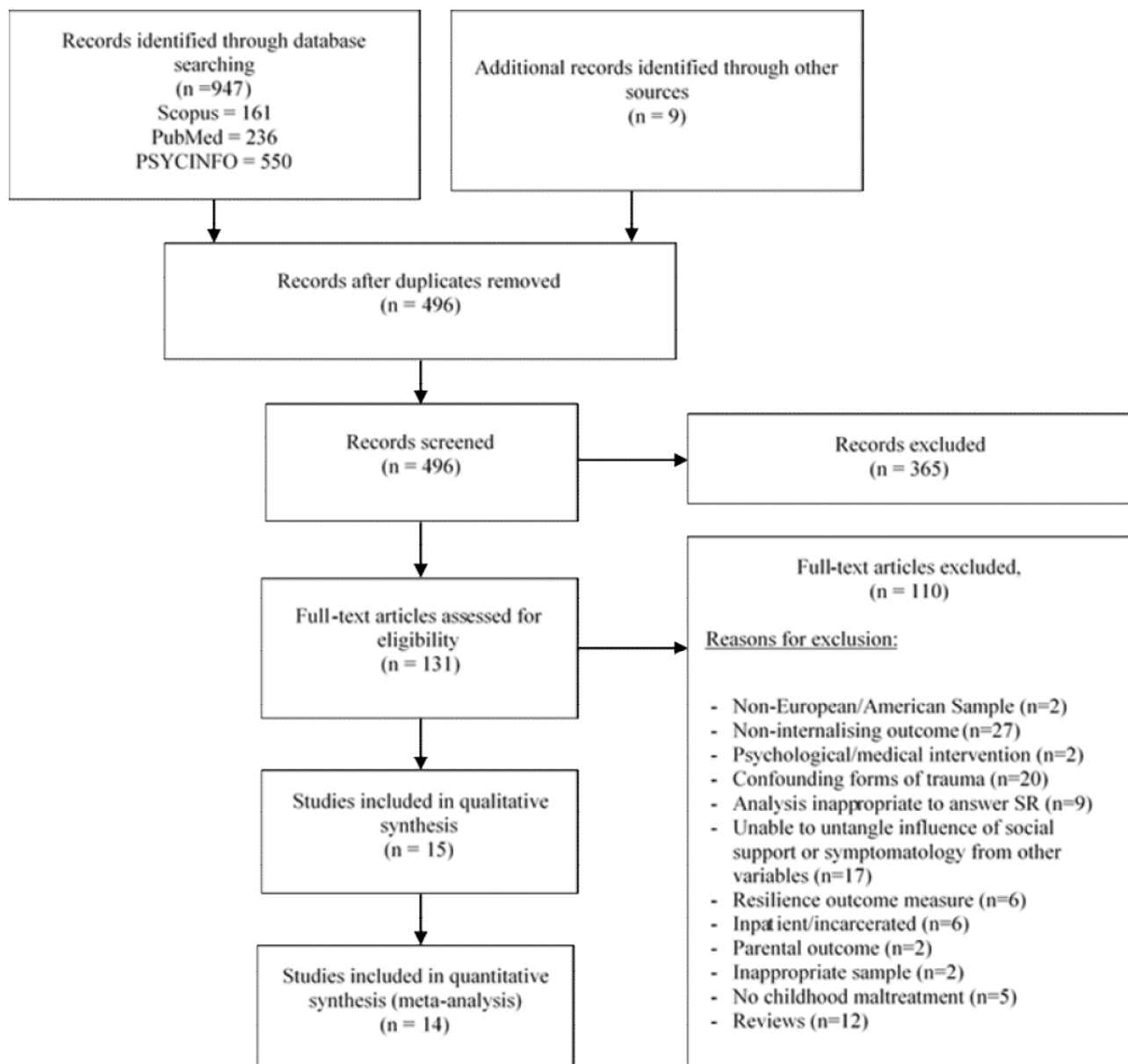
Due to the variability in the reporting of standardised and unstandardized beta coefficients, of the indirect path in mediation analyses, the variability in the inclusion of covariates across studies, and lack of some studies to report direct path statistics, it

would be problematic and potentially invalid to directly compare the beta coefficients across studies via meta-analysis. Therefore, a narrative approach to synthesis was taken for mediation and moderation models, informed by guidance on the narrative synthesis in systematic reviews by Popay and colleagues (Popay et al., 2006). The overarching guidance identifies four main elements of narrative synthesis: 1) developing a theoretical model for the role of a variable or intervention (as shown by separating the three hypotheses of social support), 2) developing a preliminary synthesis which describes strength and direction of effects, 3) exploring relationships within and between studies, 4) assessing the robustness of the synthesis which may include assessment of quality, study methodology, and strength of evidence to draw conclusions (provided within the results and discussion).

## Results

756 studies were identified through electronic databases using the search terms specified in **Table 1**. Nine additional articles were identified through review of eligible articles reference lists and through other sources. Removal of duplicates condensed the pool of articles to 496, of which the titles and abstracts were subsequently screened. The full text of 131 articles were then assessed using the eligibility criteria set out in the methods section. Following this step, 110 articles were excluded, with reasons provided in **Figure 4**. Two of the studies (Kennedy, Bybee, Sullivan, & Greeson, 2009, 2010) undertook similar analyses looking at separate outcomes, depression and PTSD, on the same sample, therefore the data from these two articles has been combined for the purposes of the systematic review and will be referred to as one study (Kennedy et al, 2009, 2010).





*Figure 4. PRISMA flow diagram*

### Quality assessment

The quality ratings of the included studies are presented in **Table 2**. 11 of the included studies received an overall quality rating of ‘strong’, indicating that they met criteria within all areas of the tool. While nine studies received an overall rating of ‘moderate’, only one of these received a ‘no’ rating (indicating that it did not satisfy

criteria for adequate quality) in more than one domain. One study received a ‘weak’ rating (Miller, VanZomeren-Dohm, Howell, Hunter, & Graham-Bermann, 2014). However, on agreement with my supervisor, this was included in the systematic review due to the relative strength within the other domains (i.e. sampling and maltreatment measurement).

*Table 2. Quality ratings for included studies*

	Sampling strategy	Internalising measurement	Maltreatment measurement	Social support measurement	Confounders	Relevant analytic protocol	Overall quality rating
1. Hyman 2003	Yes	Yes	Yes	Yes	No	Yes	Moderate
2. Miller 2014	Yes	Yes	No	Yes	No	No	Weak
3. Steine 2017	Yes	Yes	Yes	Yes	Yes	Yes	Strong
4. Munzer 2017	Yes	Yes	Yes	Yes	Yes	Yes	Strong
5. Ezzell 2000	Yes	Yes	Yes	Yes	Yes	Yes	Strong
6. Pinto 2017	Yes	Yes	Yes	Yes	Yes	Yes	Strong
7. Lagdon 2018	Yes	Yes	No	Yes	Yes	Yes	Moderate
8. Vranceanu 2007	Yes	Yes	Yes	Yes	No	Yes	Moderate
9. Tremblay 1999	Yes	Yes	Yes	Yes	No	Yes	Moderate
10. Stevens 2013	Yes	Yes	Yes	Yes	No	Yes	Moderate
11. Negriff 2018	Yes	Yes	Yes	Yes	Yes	Yes	Strong
12. Owen 2008	Yes	Yes	Yes	Yes	No	Yes	Strong
13. Salazar 2011	Yes	Yes	Yes	Yes	Yes	Yes	Strong
14. Sperry 2013	Yes	Yes	Yes	Yes	Yes	Yes	Strong
15. Folger 2013	Yes	Yes	Yes	Yes	No	Yes	Moderate
16. Jaffee 2017	Yes	Yes	Yes	Yes	Yes	Yes	Strong
17. Wilson 2014	Yes	Yes	Yes	Yes	No	Yes	Moderate
18. Powers 2009	Yes	Yes	Yes	Yes	No	Yes	Moderate
19. Gagne 2013	No	Yes	Yes	Yes	No	Yes	Moderate
20. Kennedy 2009, 2010	Yes	Yes	Yes	Yes	Yes	Yes	Strong

## Study overview

Data was extracted from the 20 studies and synthesised into two tables; study sampling and participant characteristics (**Table 3**), and study characteristics and outcomes (**Table 4**). For ease of reporting the first author and year of publication were used in the tables and relevant figures, full references are provided in the main text, and are asterisked in the reference list.

### Sample characteristics

**Table 3** provides an overview of the sample characteristics of the included studies. Half of the studies were undertaken with adult samples, and half with child and adolescent samples. The mean age of the adult samples ranged from 20.36 – 40.1 years, whereas the child and adolescent sample mean ages ranged from 4.94 – 21.09 years, as one of the studies followed individuals longitudinally from late adolescent into early adulthood (Salazar, Keller, & Courtney, 2011). The majority of the child and adolescent samples recruited from pre-adolescent (7-12 years) and adolescent populations (12-18 years), with only one recruiting a sample of young children (<6 years; Miller, Adams, Esposito-Smythers, Thompson, & Proctor, 2014). However, no study reported pubertal status, therefore such developmental designations may be variable across the studies. Most of the studies were carried out in the United States of America and Canada (70%) with the remaining studies being carried out in Europe (30%). 65% of the studies were carried out within the last ten years, whilst the remaining 35% were undertaken in the last twenty years. Four of the studies recruited only female participants, while six studies recruited between 40-60% female participants (mean across studies = 66.2% female, SD = 23.9%).

Sample size varied greatly from  $n=50$  (Tremblay, Hébert, & Piché, 1999) to 885 (Jaffee, Takizawa, & Arseneault, 2017), with a mean of  $n=295$  ( $SD=232.4$ ). 65% of the studies could be classified as taking a judgemental non-probabilistic approach to sampling (MT is an inclusion criteria from the convenience population), and 25% taking a consecutive non-probabilistic approach (all sampled from a convenience population), whereas the remaining 10% (two studies) used judgemental stratified sampling methods from a larger longitudinal cohort. Only four of the studies explicitly recruited a control group (Jaffee et al., 2017; Lagdon et al., 2018), and of these, two reported matching between the groups on socio-demographic variables (Negriff, Cederbaum, & Lee, 2018; Sperry & Widom, 2013). The remaining 16 studies undertook either within-group correlational analyses (13 studies) or constructed groups based on the responses to the measures of maltreatment experience post-recruitment (5 studies). When socio-economic status was reported (in 11 studies), it indicated that the samples were broadly from lower socio-economic groups; the mean rate of unemployment or receipt of benefits of the seven studies who reported the data was 68.7%, while education was predominantly at a high/secondary school level ( $\leq 13$  years of education), and income was generally in lower brackets (between \$500-1400 monthly income). When socioeconomic status was indicated to be elevated within the sample (Folger & Wright, 2013), individuals were found to be sampled from a university student pool, and thus there may be a greater likelihood of reflecting a higher socioeconomic group. 15 studies reported information on ethnicity. Across these studies, mean percentages indicated that 41.1% of individuals were from African-American backgrounds, 39.8% Caucasian, 9.4% Hispanic, and 13% were from other ethnic backgrounds. Only one study recruited an exclusively African-American population (Owen et al., 2008).

*Table 3. Study sampling and participant characteristics*

Study	Design, sampling, sample Location	Inclusion/exclusion, within/between group	Developmental group, age range and mean, N, sex ratio,	Ethnicity	Socioeconomic
1. Hyman 2003	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Clinical sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> historical MT; Exclusion: current MT</li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Age range</i> =17 - 55 (mean = 33.36; SD=8.98)</li> <li>• N=172</li> <li>• 100% female</li> </ul>	<i>African-American</i> 6.3%; <i>Caucasian</i> 82.4%; <i>Hispanic</i> 6.3%; <i>Asian-American</i> 0.6%; <i>Native-American</i> 0.6%; <i>Multiracial</i> 1.9%; <i>Other</i> 0.9%	<i>Unemployment:</i> 44.7%  <i>Annual income:</i> 28.2% '\$20,000 or more', 19.5% 'below \$5,000'.  <i>Education:</i> range of 4 to 20 years (mean 12.59).
2. Miller 2014	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling from a larger RCT</li> <li>• Community</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> MT</li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Child</li> <li>• <i>mean age</i>=4.94 (SD=0.85)</li> <li>• N=120</li> <li>• 47.5% female</li> </ul>	<i>African-American</i> 37%; <i>Caucasian</i> 48%; <i>Hispanic</i> 6%; <i>Asian-American</i> 1%; <i>Biracial</i> 8%	<i>Monthly income:</i> mean = \$1,347.57 (SD = \$1,376.90).  <i>Education:</i> 40% high school degree or less, 60% college education.
3. Steine 2017	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Clinical sample</li> <li>• Norway</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Age range</i> =19-71 (mean = 42.9, SD=11.6)</li> <li>• N=138</li> <li>• 96.4% female</li> </ul>	DNR	<i>Education:</i> 18.9% college, 27.4% school, 18.5% University, 10.4% primary school,  <i>Employment:</i> 47.4% disability benefits/pension, 24.8% full-time employed,

4. Munzer 2017	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• Germany</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: MT</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Children and adolescents</li> <li>• Age range =8 -17 (mean = 12.23, SD=2.47)</li> <li>• N=200</li> <li>• 44% female</li> </ul>	DNR	DNR
5. Ezzell 2000	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: MT</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Children and adolescents</li> <li>• Age range =6-14 (mean = 9.5)</li> <li>• N=100</li> <li>• 39% female</li> </ul>	African-American 62%; Caucasian 32%; other 6%;	SES measure (Hollingshead,1975): mean=26.2; “machine operators, semiskilled workers” category)
6. Pinto 2017	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• Portugal</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: MT; Exclusion: Neurodevelopmental, psychosis</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adolescent</li> <li>• Age range =13-17 (mean = 15.71, SD=1.31)</li> <li>• N=183</li> <li>• 51.4% female</li> </ul>	DNR	DNR
7. Lagdon 2018	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Community sample</li> <li>• Northern Ireland</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Control group (n=473), MT group (n=167)</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• Median ages, MT = 22, NMT = 21 (ranges or SD not reported)</li> <li>• N=640</li> <li>• 75.62% female</li> </ul>	DNR	DNR
8. Vranceanu 2007	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• Mean = 28.92 (SD=10.52)</li> <li>• N=100</li> <li>• 100% female</li> </ul>	African-American 47%; Caucasian 48%; Other ethnicity 5%	Unemployment: 73% Annual income: 67% less than \$15000
9. Tremblay 1999	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Community sample</li> <li>• France</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: MT; Exclusion: neurodevelopmental, language</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Child</li> <li>• Age range =7 - 12 (mean = 9.2)</li> <li>• N=50</li> <li>• 78% female</li> </ul>	Caucasian 96%	Education: range of 10 to 11 years

10. Stevens 2013	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> physically healthy</li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Mean age</i>=28.46 (SD=7.76)</li> <li>• N=130</li> <li>• 100% female</li> </ul>	<i>African-American</i> 83.5%; <i>Caucasian</i> 5.8%; <i>Hispanic</i> 5.8%; <i>Other</i> 6.4%;	<i>Unemployment:</i> 48.2%  <i>Annual income:</i> mean=\$9,455.00 (SD=14,442.00).
11. Negriff 2018	<ul style="list-style-type: none"> <li>• Retrospective cohort study. Convenience sampling.</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> MT</li> <li>• <i>Matched control:</i> age, sex, ethnicity, neighbourhood SES.</li> <li>• <i>Control group</i> (n=151), MT group (n=303)</li> </ul>	<ul style="list-style-type: none"> <li>• Child</li> <li>• <i>Mean age at T1:</i> 11.11(SD=1.15)</li> <li>• <i>Mean age at T2:</i> 12.28(SD=1.26)</li> <li>• N=454</li> <li>• 40% female in NMT group; 50% female in MT group</li> </ul>	<i>African-American</i> 38%; <i>Caucasian</i> 11%; <i>Latino</i> 39%; <i>Biracial</i> 11%;	DNR
12. Owen 2008	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> current relationship; <i>Exclusion:</i> physical health, neurodevelopmental, psychosis</li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Child</li> <li>• <i>Age range</i> =8-10 (mean = 10, SD=1.43)</li> <li>• N=148</li> <li>• 69.5% female</li> </ul>	<i>African-American</i> 100%	<i>Unemployment:</i> 65%  <i>Monthly income:</i> 38% - \$500 to \$999
13. Salazar 2011	<ul style="list-style-type: none"> <li>• Retrospective cohort study. Convenience sampling from within a larger retrospective cohort study</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion:</i> fostered;</li> <li>• <i>Exclusion:</i> neurodevelopmental, incarcerated/inpatient</li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adolescent</li> <li>• <i>Mean age at T1:</i> 17.39 (SD=.49), <i>Mean age at T2:</i> 19.03 (SD=.19), <i>Mean age at T3:</i> 21.09 (SD=.30)</li> <li>• N=513</li> <li>• 54.8% female</li> </ul>	<i>African-American</i> 54.6%; <i>Caucasian</i> 33.3%; <i>Mixed race</i> 2%; <i>America/Alaskan Native or Asian/Pacific Islander</i> 2%	DNR



14. Sperry 2013	<ul style="list-style-type: none"> <li>• Retrospective cohort study. Convenience sampling</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Exclusion</i>: adoption of child, 'involuntary' neglect, placement only, failure to pay child support</li> <li>• <i>Matched control</i>: sex, race, age, hospital of birth at time of initial screening.</li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Mean ages at T1</i>=29.1 (SD=3.77). <i>T2</i>=39.5 (SD=3.51) <i>T3</i>=41.2 (SD=3.53)</li> <li>• N=696; Control (n=308), MT (n=388)</li> <li>• 51.3% female in NMT group; 54.4% female in MT group</li> </ul>	Hispanic non-white 64.5%	DNR
15. Folger 2013	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling.</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Mean age</i>=20.36 (SD=4.58)</li> <li>• N=344</li> <li>• 53.49% female</li> </ul>	African-American 3.4%; Caucasian 91.9%; Hispanic 2.6%; Asian-American 0.9%; Native-American 0.6%	Annual income: family income between \$50,000 and \$74,999.
16. Jaffee 2017	<ul style="list-style-type: none"> <li>• Retrospective cohort study. Stratified sampling</li> <li>• Community sample</li> <li>• United Kingdom</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion</i>: current intimate relationship</li> <li>• Control group (n=683), MT group (n=202)</li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Mean ages at T1</i>=33, <i>T2</i>=35, <i>T3</i>=38, <i>T4</i>=40</li> <li>• N=885</li> <li>• 100% female</li> </ul>	DNR	DNR
17. Wilson 2014	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience and systematic sampling from a larger pool.</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• <i>Age range</i> =18 - 26 (mean = 19.06, SD=1.15)</li> <li>• N=265</li> <li>• 100% female</li> </ul>	African-American 4.9%; Caucasian 75.8%; Hispanic 4.5%; Asian 10.2%; Other 4.6%	DNR

18. Powers 2009	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>DNR Inclusion/exclusion</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adult</li> <li>• Mean age = 43.1 (SD=12.7)</li> <li>• N=378</li> <li>• 54% female</li> </ul>	<i>African-American 93%; Caucasian 3.7%; Hispanic 0.3; Mixed and other 3.7%</i>	<i>Unemployment: 73.2%</i>
19. Gagne 2013	<ul style="list-style-type: none"> <li>• Cross-sectional retrospective. Convenience sampling</li> <li>• Community sample</li> <li>• Canada</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: SES</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Adolescent</li> <li>• Age range =12-17 (mean = 14.2, SD=1.1)</li> <li>• N=278</li> <li>• 45.3% female</li> </ul>	<i>DNR</i>	<i>DNR</i>
20. Kennedy 2009, 2010	<ul style="list-style-type: none"> <li>• Retrospective cohort study. Convenience sampling</li> <li>• Community sample</li> <li>• United States</li> </ul>	<ul style="list-style-type: none"> <li>• <i>Inclusion: MT</i></li> <li>• <i>Within group correlational analyses</i></li> </ul>	<ul style="list-style-type: none"> <li>• Child</li> <li>• Age range =8 -12 (mean = 9.90, SD=1.48)</li> <li>• N=100</li> <li>• 39% female</li> </ul>	<i>African-American 45%; Caucasian 29%; Hispanic 3%; Multiracial 23%;</i>	<i>Unemployment:46%</i>  <i>Government assistance/benefits: 66%</i>

DNR= did not report; MT=maltreated; PA=physical abuse; SA=sexual abuse; EA=emotional abuse; NG=neglect; EN=emotional neglect; PN=physical neglect; DV=domestic violence

### Study characteristics

Cross-sectional designs were most prevalent (80%), whereas only four studies (20%) used longitudinal retrospective cohort designs to investigate the role of social support, and thus able to consider questions related to causality between the variables of interest (**Table 3**).

### *Maltreatment*

12 of the studies measured multiple subtypes of maltreatment (multi-type), whereas eight studies measured one to two subtypes of maltreatment (singular subtype; **Table 3**). Importantly only one of these singular subtype studies measured other subtypes, and controlled for them in subsequent analyses (Steine et al., 2017). There was a great amount of variability in the measures used to capture maltreatment experience, with 12 distinct measures employed across the studies. The Childhood Trauma Questionnaire (Bernstein, Stein, Newcomb, Walker, Pogge, Ahluvalia, Stokes, Handelsman, Medrano, & Desmond, 2003) was the most commonly used tool, employed in four studies, alongside idiosyncratic methods based on standardised forms for extracting relevant information from medical and court records (employed in three studies), and the life Experiences Questionnaire (Gibb et al., 2001), used in two studies. However, all studies reported adequate validity and reliability statistics related to the questionnaire measures. Information gleaned from these questionnaires characterising the maltreatment, and used within the studies analyses, primarily related to frequency (30% of studies), severity (30%), and dichotomous presence (40%) of maltreatment. As reporting of the characterisation of abuse varied depending on the dimension measured (frequency, severity, presence), direct comparison of

subtype prevalence across studies was not consistent, and therefore was not included in **Table 3**. However, when multi-type abuse was recorded emotional abuse was reported to be the most prevalent subtype of abuse in four studies, followed by physical abuse in three studies, and neglect in two studies.

### *Social support*

As with the measures employed to capture maltreatment experience, the social support measures were similarly diverse. 11 distinct measures were employed, with the ‘Multi-dimensional Scale of Perceived Social Support’ (Zimet, Dahlem, Zimet, & Farley, 1988), and the ‘Interpersonal Support Evaluation List’ (Cohen & Hoberman, 1983) being used most commonly, however these were still only employed in four and three studies respectively, highlighting the diversity in the measurement of this concept. Three studies used an idiosyncratic measure to extract information related to frequency and appraisal of social support, or significant in-home figures within the child’s life (Miller et al., 2014). Many studies (65%), while collecting information regarding the source of social support (i.e. family, friend, other), collapsed the data to provide a combined measure of social support. Seven studies (35%) undertook separate analyses for distinct sources of social support.

### *Internalising symptomatology*

The most commonly measured psychiatric diagnosis was depression (measured in ten studies), followed by post-traumatic stress symptomatology/disorder (eight studies), and anxiety (in six studies). Measures of PTSD were the most consistent across studies; three studies employed the PTSD Symptom Scale Interview (Foa, Riggs, Dancu, & Rothbaum, 1993), and three further studies used the Impact of Event Scale – Revised (Weiss & Marmar, 1997). Depression measures were similarly consistent,

with three studies employing the Diagnostic Interview Schedule (Robins, 2011), three studies using the Children's Depression Inventory (Kovacs, 1985), and the remaining studies using well established measures such as the Beck Depression Inventory (A. T. Beck, Steer, & Brown, 1996) and PHQ-9 (Kroenke, Spitzer, & Williams, 2001). Measures for anxiety, while varied across studies were reported with adequate reliability and validity statistics and noted to be well-established measures of anxiety symptomatology. Two separate studies created a combined anxiety/depression score from the Trauma Symptoms Checklist for Children, while four studies assessed 'internalising symptomatology' using the commonly used Youth Self report questionnaire (Achenbach, 1995) and Child Behaviour Checklist (Achenbach, 2001).

Only eight studies included covariates pertinent to the sample population within the analyses. Age and sex were the most common covariates included, both present in six studies, followed by comorbid psychopathology and ethnicity, both present in three studies. Other variables of interest were included in 12 studies, with externalising symptomatology present in six studies, coping strategies in four studies, and other 'violence and trauma' in four studies, amongst other less common variables of interest. All studies included were able to report on statistics controlling for, or independent, of these 'other variables of interest'. Reporting of outcome statistics was inconsistent across studies; unstandardized and standardised beta values were reported interchangeably; standard error was not reported in many studies, and non-significant results were commonly reported without relevant statistics.

**Table 4.** Study characteristics and outcomes

Study	Maltreatment subtypes measured, form of report,	Internalising symptomatology (self-report unless indicated otherwise)	Domain of social support (self-report unless indicated otherwise)	Covariates and other variables of interest (VOI)	Outcome in relation to the role of social support (effect size if available – unstandardized(B)/standardised( $\beta$ ) beta coefficient, F-statistic, or rho)
1. Hyman 2003	<ul style="list-style-type: none"> <li>• Presence and frequency: SA</li> <li>• Structured clinical interview</li> </ul>	<b>PTSS</b>	<b>Perceived social support</b> across four domains - Self-esteem, Appraisal, belonging and Tangible, and a total cumulative score.	<i>Covariates:</i> DNR <i>Other VOI:</i> DNR	<b>Protective factor</b> – Increased levels of SS were significantly predictive of lower levels of PTSD ( $F(4, 167) = 5.540, p < .01$ ).
2. Miller 2014	<ul style="list-style-type: none"> <li>• Frequency and severity: DV.</li> <li>• Parent report.</li> </ul>	<b>Internalising symptomatology</b>	<b>Size of social network</b> - Idiosyncratic parent measure of child's in-home social network	<i>Covariates:</i> DNR <i>Other VOI:</i> externalising symptomatology	<b>Protective factor</b> - Larger in-home networks were related to fewer Internalizing ( $r = -.16, p < .01$ )
3. Steine 2017	<ul style="list-style-type: none"> <li>• Severity score: PA, SA, PN, EN, EA.</li> <li>• Self-report</li> </ul>	<b>PTSS</b>	<b>Perceived Social Support</b> - combined	<i>Covariates:</i> MT subtypes other than SA <i>Other VOI:</i> relational difficulties	<b>Protective factor</b> – ‘high and slightly decreasing’ PTSS profiles reported lower levels of SS to ‘sub-clinical and decreasing’ (mean difference = 8.45, $p = 0.001$ ).
4. Munzer 2017	<ul style="list-style-type: none"> <li>• Presence: PA, SA, NG, EA</li> <li>• Child and parent report</li> </ul>	<b>PTSS</b>	<b>Perceived Social Support</b> - combined	<i>Covariates:</i> age, sex <i>Other VOI:</i> MT related cognitions	<b>Protective factor</b> - negative correlation between SS and PTSS ( $r = -.24, p < .01$ ).

5. Ezzell 2000	<ul style="list-style-type: none"> <li>• Presence: PA.</li> <li>• Court reports – idiosyncratic extraction</li> </ul>	<b>Depression, Anxiety, and combined Anxiety &amp; Depression</b>	<b>Perceived Social Support</b> from family and friends	<i>Covariates:</i> DNR  <i>Other VOI:</i> Anger and aggression	<b>Protective factor</b> – family ( $r=-.31, p>.05$ ) and teacher ( $r=-.23, p>.05$ ) SS did not, while friend ( $r=-.51, p<.01$ ) SS significantly negatively correlate with depression. family ( $r=-.00, p>.05$ ) and teacher ( $r=.21, p>.05$ ) SS did not, while friend ( $r=-.46, p<.01$ ) SS significantly negatively correlate with anxiety
6. Pinto 2017	<ul style="list-style-type: none"> <li>• Presence: EA, PA, SA, EN, PN.</li> <li>• Self-report</li> </ul>	<b>PTSD, Depression, Anxiety</b>	<b>Perceived social support</b> - combined	<i>Covariates:</i> age, sex, education, recruitment setting, SES, comorbid symptomatology, substance abuse, traumatic events  <i>Other VOI:</i> Depression and anxiety, coping strategies	<b>Protective factor</b> – SS was significantly negatively correlated with PTSD ( $r=-.37, p<.001$ ). SS did not significantly explain variance in PTSD after controlling for MT ( $\Delta R^2 = 0.00, \beta = -.02, p>.05$ )
7. Lagdon 2018	<ul style="list-style-type: none"> <li>• Dichotomous presence: PA, EA, SA, NG.</li> <li>• Self-report.</li> </ul>	<b>PTSD, Depression and Anxiety</b>	<b>Perceived social support</b> from family, friends and significant other	<i>Covariates:</i> Sex, age, relationship status  <i>Other VOI:</i> DNR	<b>Partial mediator:</b> <ul style="list-style-type: none"> <li>- family SS partially mediates the relationship between CM and PTSD, anxiety, &amp; depression (<math>\beta = .37, \beta = .25, \beta = .20</math>, respectively; <math>p&lt;.05</math>).</li> <li>- Friend SS partially mediates relationship between CM and anxiety (<math>\beta = .14, p&lt;.05</math>), no mediation effect for PTSD (<math>\beta = .07</math>) or depression (<math>\beta = .08</math>).</li> <li>- Other SS <i>did not</i> mediate between CM or outcomes (all <math>\beta = .01</math>).</li> </ul>
8. Vranceanu 2007	<ul style="list-style-type: none"> <li>• Cumulative frequency: PA, EA, NG, SA, DV.</li> <li>• Self-report.</li> </ul>	<b>PTSS and Depression</b>	<b>Perceived social support, and satisfaction with support network</b> – combined	<i>Covariates:</i> DNR  <i>Other VOI:</i> Perceived stress, Loss of resources	<b>Partial mediator</b> - Social support was a significant mediator, partially explaining the effects of CM on PTSD ( $\beta=.26, p<0.01$ ) but <i>not</i> depression (DNR). Direct path remained significant

8. Vranceanu 2007	(carried over from previous page)				<b>Protective factor</b> – negative correlation between SS and PTSD ( $r=-.38, p<.05$ ), but not between SS and depression (DNR).
9. Tremblay 1999	<ul style="list-style-type: none"> <li>• Identity, severity and duration: SA.</li> <li>• Medical reports – standardised form extraction</li> </ul>	<b>Internalising symptomatology</b>	<b>Perceived social support</b> from peers, family and teachers	<i>Covariates:</i> DNR  <i>Other VOI:</i> Perceived competence, Coping strategies, Externalising symptomatology	<b>Does not mediate</b> – family and friend SS <i>did not</i> mediate the association between SA and internalising symptomatology ( $\beta=-.02, \beta=-.10$ , respectively, $p>.05$ ).  <b>Protective factor</b> – <i>non-significant</i> correlation between family ( $r=-.13, p>.05$ ) and friend ( $r=-.03, p>.05$ ) SS and internalising
10. Stevens 2013	<ul style="list-style-type: none"> <li>• Presence: PA, EA, SA.</li> <li>• Self-report</li> </ul>	<b>PTSS</b>	<b>Perceived Social Support</b> - combined	<i>Covariates:</i> none reported  <i>Other VOI:</i> Recent interpersonal violence, emotion regulation difficulties	<b>Partial mediator</b> - Social support partially mediated the association between CM and PTS symptoms ( $B=.05, se=.02$ ). Direct path remained significant.  <b>Protective factor</b> – negative correlations between SS and PTS symptoms ( $r=-.48, p<.001$ ).
11. Negriff 2018	<ul style="list-style-type: none"> <li>• Cumulative instance: SA, PA, EA, NG.</li> <li>• Court reports – idiosyncratic extraction</li> </ul>	<b>Depression</b>	<b>Perceived social support</b> from family and friends	<i>Covariates:</i> placement stability, sex, age, ethnicity, no. of people in social network  <i>Other VOI:</i> DNR	<b>Does not mediate</b> - family and Friend SS <i>did not</i> act as a mediator between CM and depressive symptomatology (DNR indirect path).  <b>Protective factor</b> – negative correlations between family ( $r=-.12, p<.01$ ) and friend ( $r=-.10, p<.05$ ) SS and Depression at time 1, but not at time 2 ( $r=-.05, r=-.09$ , respectively, $p>.05$ ).



12. Owen 2008	<ul style="list-style-type: none"> <li>•Severity: DV.</li> <li>•Child and parent report</li> </ul>	<b>Internalising symptomatology</b>	<b>Perceived Social Support</b> - combined	<i>Covariates:</i> DNR  <i>Other VOI:</i> externalising symptomatology, mother's social support	<b>Partial mediator</b> - mediational role of children's perceived levels of social support in the relation between blame (B=.21, p<.05), conflict (B=.08, p<.05), and threat (B=.10, p<.05) in DV, and internalising problems (separate tests).  <b>Protective factor</b> – negative correlation between SS and internalising (r=-.34, p<.001).
13. Salazar 2011	<ul style="list-style-type: none"> <li>•Frequency: PA, SA, EA, NG</li> <li>•Self-report</li> </ul>	<b>Depression</b>	<b>Perceived social network sufficiency</b> - combined	<i>Covariates:</i> sex, age, ethnicity  <i>Other VOI:</i> DNR	<b>Partial mediator</b> - Partial mediating effects of SS for both pre-care MT (B=-.27 (95% CI= -.39 -.15) p<.001) and during care MT (B= -.27, 95% CI -.39 to -.15, p<.001) on depression.  <b>Moderator</b> –SS moderated the association between pre-care (B=0.119 (95% CI=0.01-0.23), p=0.027) and during-care (B=0.147 (CF=0.04-0.25), p<.01) MT on depression  <b>Protective factor</b> – SS was significantly negatively correlated with depression (r=-.12, p<.01)
14.Sperry 2013	<ul style="list-style-type: none"> <li>•Dichotomous presence: PA, SA, NG.</li> <li>•Court reports - idiosyncratic extraction</li> </ul>	<b>Anxiety and Depression</b>	<b>Perceived social support</b> across four domains - Self-esteem, Appraisal, belonging and Tangible, and a total cumulative score.	<i>Covariates:</i> Age, sex, ethnicity, prior psychiatric diagnosis. Lifetime symptoms of depression, anxiety, and drug use	<b>Partial mediator</b> – Total SS and belonging domain of SS mediated relationship between CM and depression (B=1.56, 95% CI .53-1.63) and anxiety (B=1.03, 95% CI .93-2.32) – direct path became NS.  <b>Did not moderate</b> - SS <i>did not</i> interact (moderate) with level of CM on outcomes (B=.16, 95% CI -.24-.55; Depression DNR).  <b>Protective factor</b> - Direct protective factor of total SS for all individuals regardless of level of stressor (MT vs. NON-MT) for anxiety (B=-.79, p<.001) and depression (B=-1.28, p<.001) only.

15. Folger 2013	<ul style="list-style-type: none"> <li>•Cumulative frequency: PA, SA, EA, EN, PN.</li> <li>•Self-report</li> </ul>	<b>Anxiety and depression</b> combined to create a measure of 'negative affect'	<b>Perceived social support</b> from family and friends	<i>Covariates:</i> DNR <i>Other VOI:</i> DNR	<p><b>Moderator</b> – friend SS moderated the relationship between MT and negative affect - effect only apparent within males. (<math>\beta = .16, p=.016</math>). Greater CM and low SS experienced greater Depression/anxiety</p> <p><b>Protective factor</b> – negative correlation between family and friend SS and 'negative affect' (<math>r = -.31, r = -.33</math>, respectively)</p>
16. Jaffee 2017	<ul style="list-style-type: none"> <li>•Dichotomous presence: PA, SA, EA, EN, PN.</li> <li>•Self-report.</li> </ul>	<b>Depression and Anxiety</b>	<b>Enacted financial support, twin support, emotional support</b> - from family, friends and significant other – combined	<i>Covariates:</i> socioeconomic status <i>Other VOI:</i> Psychosis spectrum disorder, physical health markers, health-risk behaviours	<p><b>Did not moderate</b> – SS <i>did not</i> moderate the association between CM and anxiety (<math>B=.67, p&gt;.05</math>), or depression (<math>B=.96, p&gt;.05</math>)</p> <p><b>No protective effect</b> – No difference between high vs low SS on depression and anxiety</p>
17. Wilson 2014	<ul style="list-style-type: none"> <li>•Dichotomous presence: PA, SA.</li> <li>•Self-report</li> </ul>	<b>PTSS</b>	<b>Perceived social support</b> from family, friends and significant other	<i>Covariates:</i> DNR <i>Other VOI:</i> DNR	<p><b>Moderator</b> - family and friends SS moderated PTSD symptoms in physical abuse survivors (<math>\beta = -.37, \beta = -.16, p &lt; .05</math>), but not for Other SS (<math>\beta =</math>) but not sexual abuse survivors (DNR).</p> <p><b>Protective factor</b> – negative correlation between family (<math>r = -.20, p &lt; .05</math>), and friend (<math>r = -.16, p &lt; .01</math>) SS and PTSD, but not significant other and PTSD (<math>r = -.01, p &gt; .05</math>).</p>
18. Powers 2009	<ul style="list-style-type: none"> <li>•Presence: PA, SA, EN, PN, EA.</li> <li>•Self-report</li> </ul>	<b>Depression</b>	<b>Perceived Social Support</b> from family and friends	<i>Covariates:</i> DNR <i>Other VOI:</i> Community and School violence	<p><b>Moderator</b> – friend SS moderated the relationship between MT and depression (<math>\beta = -.14, t = -3.01, p &lt; .01</math>); however this was only significant in females (<math>\beta = -.27, t = -4.10, p &lt; .001</math>) and not in males (<math>\beta = -.03, t = -.50, p = .63</math>). Family SS <i>did not</i> moderate (<math>\beta = -.03, t = -.61, p = .54</math>)</p>

18. Powers 2009	(carried over from previous page)				<b>Protective factor</b> – family ( $r=-.20$ , $p<.01$ ) and friend ( $r=-.22$ , $p<.01$ ) SS significantly negatively correlated with depression.
19. Gagne 2013	<ul style="list-style-type: none"> <li>•Frequency: EA.</li> <li>•Self-report</li> </ul>	<b>Internalising symptomatology</b>	<b>Perceived social support</b> - availability and satisfaction	<i>Covariates:</i> none reported  <i>Other VOI:</i> externalising symptomatology	<b>Did not moderate</b> – availability ( $\beta = -.07$ , $p>.05$ ) and satisfaction ( $\beta = .07$ , $p>.05$ ) SS <i>did not</i> moderate the association between EA and internalising.  <b>Protective factor</b> - availability ( $r=-.20$ , $p<.05$ ) and satisfaction ( $r=-.29$ , $p<.01$ ) SS significantly negatively correlated with internalising symptomatology.
20. Kennedy 2009, 2010	<ul style="list-style-type: none"> <li>•Frequency: DV.</li> <li>•Self-report</li> </ul>	<b>Anxiety and Depression</b>	<b>Perceived Social Support and satisfaction</b> , from family only	<i>Covariates:</i> DNR  <i>Other VOI:</i> Community and School violence	<b>Did not moderate</b> – family SS <i>did not</i> moderate the association between DV and anxiety initially or over time (DNR) or depression initially ( $\gamma_{07}=1.10$ , $p>.05$ ) or over time ( $\gamma_{17}=-.10$ , $p>.05$ ).  <b>No protective effect</b> – family SS <i>did not</i> significantly predict anxiety over time ( $\gamma_{06}=1.19$ , $p>.05$ ), or depression over time ( $\gamma_{06}=-.01$ , $p>.05$ ). MT sample
Notes: SS= social support; DNR= did not report; MT=maltreated/maltreatment; PA=physical abuse; SA=sexual abuse; EA=emotional abuse; NG=neglect; EN=emotional neglect; PN=physical neglect; DV=domestic violence					

## Salient findings

The studies in **Table 4** are ordered to reflect the grouping of main analysis type from direct protective factor, to mediator, and moderator models. If studies reported independent domains of social support, such as belonging or appraisal (Sperry & Widom, 2013), an available total score was examined if possible to provide equivalence in comparison across studies. Utilising the guidance on narrative synthesis by Popay and colleagues (Popay et al., 2006), the sections on the role of social support as a mediator and a moderator, will explore the strength and direction of reported effects, and whether these effects vary according to variable and study characteristics.

### *Direct protective factor*

All but one (Lagdon et al., 2018) of the included studies reported zero-order correlations or univariate group comparisons between social support and symptomatology. Nine of these nineteen studies recruited samples who all shared an experience of childhood maltreatment, while ten either recruited maltreated samples with concomitant control groups, or samples in which maltreatment experience was measured following sampling and thus represents a sample with both maltreated and non-maltreated individuals. Appropriate correlation statistics were available for 14 studies, the remaining five studies reported dichotomous group comparison statistics and for brevity were not included in the meta-analysis. These 14 studies reported 26 independent effect sizes. Initial models combined the source of social support, to provide a combined effect size per study. All but one study included in the meta-analysis reported correlations with one outcome measure of internalising

symptomatology, therefore a combined effect size of internalising symptomatology was pooled for the purposes of the meta-analysis (Ezzell, Swenson, & Brondino, 2000). Sample sizes ranged from  $n=50$  (Tremblay et al., 1999) to  $n= 513$  (Salazar, Keller, & Courtney, 2011).

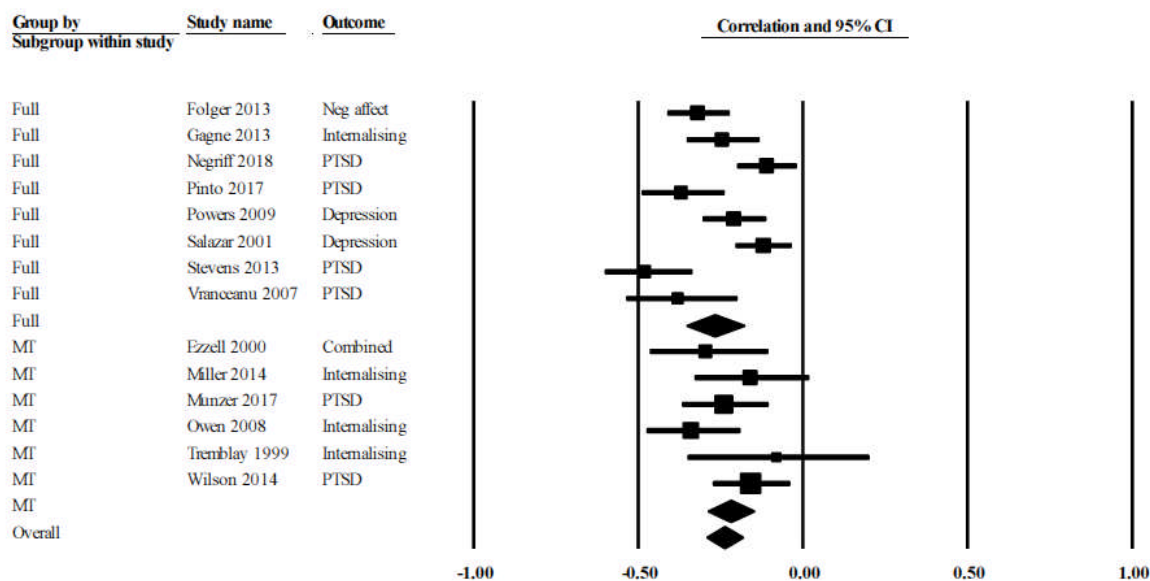
**Table 5.** Outcomes of the meta-analysis on the correlation between social support and psychopathology in mixed and MT only samples.

Sample	No. of studies	No. of effect sizes	N	Correlation (95% CI)	Z-value	p
Mixed	8	12	2380	-.27 (-.35, -.18)	-5.82	<.001
MT only	6	14	883	-.22 (-.29, -.15)	-6.11	<.001
Combined	14	26	3263	-.24 (-.29, -.19)	-8.39	<.001

Notes: Mixed = sample included participants with and without experiences of maltreatment.

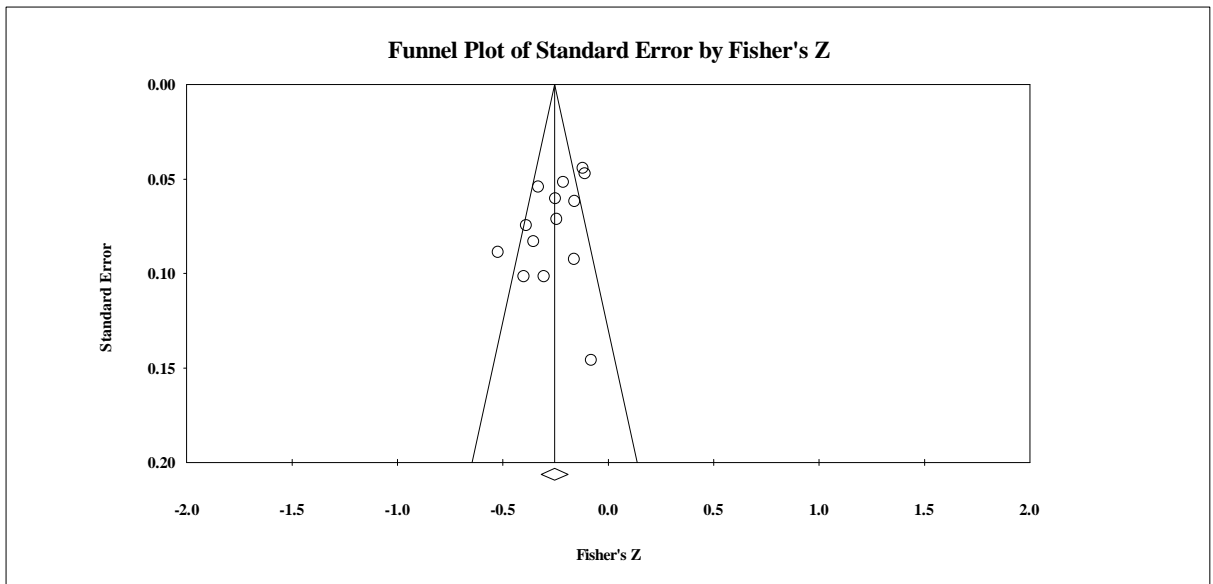
A significant association between at least one aspect of social support and internalising symptomatology was reported in 13 of the 14 identified studies. Pooled estimates using all studies suggested that there was a significant negative correlation between social support and internalising symptomatology ( $r=-.24$  [95% CI=-0.29, -0.19],  $p<.001$ , Table 5), of a small effect size (Cohen, 1992). A subgroup analysis to explore pooled effect sizes within mixed samples (those including both maltreated and non-maltreated individuals) and maltreatment only samples (only individuals who had experienced maltreatment) found that there was not a significance difference between the pooled estimates ( $Q(1)=0.74$ ,  $p=.39$ ). Table 5 summarises the pooled correlations between the subgroups and the combined correlation across all studies/subgroups.

Figure 5 displays the forest plot of all studies, including the cumulative correlations for mixed samples, MT-only samples and overall correlational effect size.



**Figure 5.** A cumulative meta-analysis on the relationship between social support in mixed and MT-only samples.

Mean age ranged from 4.94 (Miller et al., 2014) to 43.1 years (Powers, Ressler, & Bradley, 2009). Using meta-regression on the 14 included studies, the relationship between social support and internalising symptomatology was not found to be moderated by age ( $B=-.003$ ,  $SE=.003$  [95% CI=-0.01 - 0.004],  $Q(1)=0.78$ ,  $p=.38$ ). Source of social support was also explored as a potential moderator. Due to only two effect sizes being attributable to ‘teacher’ and ‘significant other’ within the included studies, comparison was restricted to within ‘combined’, ‘friend’ and ‘family’ sources of social support. The source of social support was found to not moderate the relationship between social support and internalising symptomatology ( $Q(2)=1.92$ ,  $p=.38$ ).



**Figure 6.** Funnel plot for all studies reporting correlational statistics between social support and internalising symptomatology.

The Orwin's fail-safe N suggested that an additional 65 studies would need to be located with non-significant results to bring the combined effect size to a correlation coefficient of  $r=.05$ . Kendall's Tau-b (corrected for any ties, if any) was  $-.25$  ( $p=.10$ ), suggesting that there is a lack of significant inverse correlation between study size and effect size. Duval and Tweedie's Trim and Fill approach suggested that under the random effects model that the value of the pooled effect across the studies are unchanged. A funnel plot is presented in **Figure 6** which depicts that there is relative symmetry about the combined effect size, suggestive of a lack of publication bias. Taken together, these statistics and funnel plot suggest the absence of publication bias.

### *Mediation*

Eight studies (four of ‘strong’ and four of ‘moderate’ quality) explicitly investigated a mediation effect of social support on the relationship between childhood maltreatment and internalising symptomatology (7-14 in **Table 4**; Lagdon et al., 2018; Negriff et al., 2018; Owen et al., 2008; Salazar, Keller, & Courtney, 2011; Sperry & Widom, 2013; Stevens et al., 2013; Tremblay et al., 1999; Vranceanu, Hobfoll, & Johnson, 2007). Of these eight studies, three recruited child and adolescent samples (12-14 in **Table 4**), and five were undertaken with adult samples (7-11 in **Table 4**). While six studies generally reported a significant mediation effect, there was variability in the sources of social support which were associated with these significant mediations which will be explored below. Exploring the mediation analyses by developmental period (child and adolescents vs adults), there did not appear a general trend for differences in the mediation effect as a function of developmental age grouping, with four of the five adult studies and two of the three child and adolescent studies reporting a mediation effect.

### *Strength of effects*

Within the studies that had reported a mediation effect, only one study (Sperry & Widom, 2013) reported mediation in which the direct path between maltreatment and the symptomatology outcome, depression, became non-significant when accounting for the indirect path (the association between maltreatment to depression *through* social support). This was not apparent with anxiety as an outcome of interest, which nonetheless demonstrated a significant partial mediation. While this result indicates a potentially substantial mediation effect, the term ‘full’ mediation should be used with caution as the direct path coefficient was not reduced to zero. The remaining five



studies reporting mediation, found a partial mediation effect, such that the direct path from maltreatment to internalising outcome remained significant when accounting for the indirect path, and therefore other unaccounted-for variables may also significantly mediate the direct pathway.

However, while reporting significant partial mediations, three studies indicated that there was still a substantial main effect after accounting for the indirect path (Salazar et al., 2011; Stevens et al., 2013; Vranceanu et al., 2007), through mechanisms other than social support (e.g. indirect path  $B=.04$ , direct pathway after accounting for indirect path  $B=.36$ ; Stevens et al., 2013). Therefore, while these partial mediations are significant, the degree to which they decrease variance in the direct pathway, and thus represent substantial mediators, appears modest. Both Lagdon et al (2018), and Owen et al (2008), did not report the direct path accounting for the indirect pathway, and thus the strength of the mediation effect cannot be fully understood for these studies.

#### *Non-significant effects*

The two studies that predominantly reported a non-significant mediation effect of social support (Negriff et al., 2018; Tremblay, Hebert, & Piche, 1999), described either small direct effects from maltreatment to internalising symptomatology ( $\beta=.11$ ; Negriff et al., 2018) or only a significant direct effect in one area of maltreatment characterisation (perpetrator identity only  $\beta=.33$ , and not within severity  $\beta=.01$  or duration of abuse  $\beta=-.01$ ; Tremblay et al., 1999). As such, lack of variance within the direct path may have precluded the detection of mediation effects. Negriff and colleagues, found that depression symptomatology and social support scores decreased over time in their longitudinal investigation, further suggesting that there

may have a lack of variance in the predictor variables. Tremblay et al (1999), recruited the fewest participants of all the studies in this review ( $n=50$ ), which begs a question of lack of power, given the larger required samples for mediation analyses than simple regression analysis. Given that the direct paths after accounting for indirect paths were still substantial in several studies, and thus there is a potentially small mediation effect, a lack of power due to small sample size may account for non-significant mediation in this case.

#### *Variability in sources and characteristics of social support*

While one study presented ‘headline’ significant partial mediation effects (Lagdon et al., 2018), it is important to note that for the majority of the other mediation models within the study (five of nine) which explored different sources of social support (family, friends, significant others) on three separate indices of symptomatology (PTSD, depression and anxiety) indicated a non-significant mediation effect. Within Lagdon and colleagues’ study, ‘significant other’ social support did not mediate the association between multi-type abuse and PTSD, anxiety, or depression. Social support from friends painted a similar picture, as it was found to only significantly partially mediate the association between maltreatment and anxiety, but not for the outcomes of depression and PTSD. Exploring the relative beta coefficients between the different sources of social support, the effects were considerably larger within the models exploring family social support ( $\beta=.20$  to  $.37$ ), than friends ( $\beta=.07$  to  $.14$ ), and significant others (all  $\beta=.01$ ; Lagdon et al., 2018). Two further studies explored social support by independently looking at the sources of social support, from family and friends (Negriff et al., 2018; Tremblay et al., 1999). In both studies social support was not a significant mediator. However, in the five studies in which the social support

variable was combined across sources social support, it was consistently found to mediate the relationship between maltreatment and internalising symptomatology. Together these results demonstrate that combined measures of social support exhibit greater mediation effects than measures that explore separable sources of social support.

#### *Variability in symptomatology measures*

Overall there was not an apparent trend for certain symptomatology clusters to show consistent patterns of significant mediations over others, nor were reported effects found to be stronger for one symptomatology over others (e.g. whether direct path was non-significant after considering the indirect path).

#### *Moderation*

The outcomes from the moderation analyses displayed greater variation than the outcomes of the mediation analyses. Of the eight studies (four of 'strong' and four of 'moderate' quality; **Table 2**) that investigated the moderation of social support on the association between maltreatment experience and internalising symptomatology (no. 13- 20 in **Table 4**; Folger & Wright, 2013; Gagné & Melançon, 2013; Jaffee, Takizawa, & Arseneault, 2017; Kennedy et al., 2009, 2010; Powers et al., 2009; Salazar et al., 2011; Sperry & Widom, 2013; Wilson & Scarpa, 2014), four studies (no. 13,15,17-18 in **Table 4**; Folger & Wright, 2013; Powers et al., 2009; Salazar et al., 2011; Wilson & Scarpa, 2014) reported significant main moderation effects. However, exploring the directionality of the moderation effects and the interaction with demographic variables painted an inconsistent picture of social support as a moderator of maltreatment experience on internalising symptomatology.

### *Direction and strength of moderation effect*

In the investigation by Salazar et al (2018), a significant moderation effect was demonstrated between cumulative number of maltreatment subtypes experienced (both pre-fostering care and during fostering care) and a combined measure of social support on depression symptomatology. However, the effect for pre-care maltreatment indicated that social support was more influential when maltreatment was less severe and diminished for individuals who experienced a greater number of maltreatment subtypes. An unexpected finding was also presented in relation to ‘during-care’ maltreatment, in which low levels of social support did not differentially impact depressive symptomatology while maltreatment subtype experienced increased, but at moderate and high levels of social support there was a positive relationship with number of maltreatment subtypes on depressive symptomatology, such that symptom levels were *greater* than those with low social support.

Wilson and colleagues undertook an individual approach to investigate the moderation effect of social support, by contrasting two separable subtypes. While the subtypes were represented conceptually independent maltreatment experiences, sexual abuse and physical abuse, there was a great amount of comorbidity, such that 85% of individuals who reported sexual abuse also reported to have experienced physical abuse. Nonetheless, moderation between maltreatment subtype and social support on PTSD was found to be significant in models exploring family, friend and ‘significant other’ social support. Examining the simple slopes significant negative correlational relationships were demonstrated between social support, for both friend and family, and PTSD for the physical abuse group *only*. Interestingly, for the ‘significant other’ test of simple slopes there was a significant positive correlation between social

support and PTSD for the sexual abuse group, while a non-significant negative correlation was found for the physical abuse group. As there was no control group/sub-sample who had not experienced maltreatment, or concrete basis to determine severity scaling between the two subtypes, moderation within this study is not comparable to the other studies included.

*Moderation effect varying by sex*

Of the two remaining studies that reported a significant moderation effect, these were found to vary by sex. Folger and colleagues (2013) found that friend social support moderated the impact of maltreatment on ‘negative affect’ (combined scale scores for anxiety and depression) only within males; such that at high levels of maltreatment those with low levels of friend social support presented with elevated levels of ‘negative affect’ when compared with males with high levels of friend social support. At low levels of maltreatment, this differential effect was not apparent, and ‘negative affect’ was similar regardless of friend social support. It is important to note that similar effects were not found to be significant for either friend or family social support for females, nor family social support in males. A similar interaction with sex was demonstrated by Powers and colleagues (2009), who found that friend social support buffered against adult depression, after maltreatment experience was accounted for in the model, however this effect was principally driven by the female participants ( $\beta = -.27, p < .001$ ). Friend social support for males was not significant in the model ( $\beta = -.27, p = .63$ ). Consistent with Folger and colleagues’ findings, Powers and colleagues also found that family social support did not significantly contribute to the variance in depression symptomatology after maltreatment experience was accounted for.

### *Non-significant effects*

Regarding the studies which reported non-significant moderation effects, Gagne and colleagues (2013) presented minimal and nonsignificant beta weights for the interaction term of emotional abuse and availability and satisfaction (both  $\beta = -.07$ ,  $p > .05$  for females;  $\beta = .02$  and  $\beta = .01$  respectively,  $p > .05$  for males) of social support, within both separate models for males and females. Sperry & Widom (2013), similarly reported minimal coefficients for anxiety (unstandardized  $B = .16$ ,  $p > .05$ ), but neglected to report coefficients statistics for the non-significant moderation between emotional abuse and total social support. The authors also reported a non-significant a three-way interaction with sex, social support, and maltreatment, indicating that the lack of moderation was consistent across males and females. Last, Jaffee et al (2017) described non-significant regression coefficients for depression and anxiety, in the opposite direction to what was predicted (social support found to increase the impact of maltreatment on outcomes). Within all these studies, sources of social support were combined, therefore one cannot untangle the potential variances in moderation effects based on family and friend support, highlighted previously.

### *Variability in symptomatology measures*

While the internalising symptomatology outcome measure used varied across the studies, there did not appear to be a trend for moderation effects within a specific internalising symptomatology cluster over others. Generally, depression was the most commonly studied symptomatology, present within five studies (Folger & Wright, 2013; Jaffee et al., 2017; Powers et al., 2009; Salazar et al., 2011; Sperry & Widom, 2013).

## Discussion

This study provides the most comprehensive (and sole) systematic review to date on the role of social support in the presence and emergence of internalising disorders in individuals who have experienced childhood maltreatment. It has been postulated that social support may influence the emergence of psychopathology following stress or trauma in three ways: as a direct protective factor, a mediator, or a moderator. These three hypotheses were used to structure the systematic review. Qualitative/narrative synthesis and a meta-analysis approach were used to synthesise 20 cross-sectional and longitudinal studies examining the role of social support in maltreated individuals in line with these hypotheses. There was consistent evidence for the role of social support as a direct protective factor. The evidence for its role as a partial mediator was relatively uniform but modest and appeared to vary by source of social support. The evidence for the role of social support as a moderator was inconsistent, varying in direction of effect and by sex, and thus providing no firm basis to infer that social support moderates the relationship between maltreatment experience and internalising symptomatology. The evidence for each hypothesis will now be considered in turn.

### Social support as a protective factor

Pooled correlation statistics indicated a homogeneous and significant small effect size for the relationship between social support and internalising symptomatology in samples of maltreated individuals, suggesting a consistent role of social support as a direct protective factor. This was presented alongside correlation statistics indicating statistically similar effect sizes within mixed samples of maltreated and non-maltreated individuals. These findings are consistent with the view that social support

is a direct protective factor for internalising symptomatology irrespective of maltreatment experience (**Figure 1**), and that the protective effect is similar for those who have experienced maltreatment. These findings add to previous meta-analytic evidence which demonstrate a protective role of social support against depression in the general population of western countries (Gariépy et al., 2016), and broader social support research (Sheldon Cohen, Gottlieb, & Underwood, 2000).

Gariépy and colleagues reported variance in the protective effect across life periods within a general population, and others have shown similar results in individuals who have experienced other forms of trauma, such as within war veterans (Weiner, Monin, Mota, & Pietrzak, 2016). However meta-regression indicated that age did not moderate the relationship. The findings also indicated that sources of social support did not moderate the relationship between social support and internalising symptomatology in contrast to previous studies (e.g. Stice, Ragan, & Randall, 2004). Overall these findings cement backing for the role of social support as a direct protective factor, particularly within maltreated individuals.

### Social support as a mediating factor

Synthesis of the mediation analyses indicated that a general trend for partial mediation of the relationship between maltreatment experience and internalising symptomatology by social support. Beta coefficients indicated a small to intermediate effect size, while many studies, bar one, reported partial mediation in which the direct path was still significant when accounting for the indirect path. Furthermore, several studies reported relatively substantial direct pathways from maltreatment to internalising symptomatology after accounting for the indirect pathway between



through social support. Taken together, these findings indicate evidence for social support as a mediator of small effect, with indirect pathways through other mechanisms accounting for considerable amount of the remaining variance.

It is perhaps unsurprising that the mediation effect of social support does not comprise a large share of the variance in an indirect pathway between maltreatment and psychopathology given the breadth of the concept (Vangelisti, 2009) and the variability of how it is measured (discussed later in this review; see page 76). Prior research indicates other mechanisms and factors that may play a substantial mediational roles in the relationship between maltreatment and psychopathology across the lifespan, such as emotional regulation (Kim & Cicchetti, 2010), impulsivity (Somers, Ginzburg, & Kramer, 2012), rumination (Raes & Hermans, 2008), and avoidance (Shenk, Putnam, Rausch, Peugh, & Noll, 2014) among others.

Findings indicated that combined measures of social support were more consistent in demonstrating a mediation role and were of generally greater effect than constituent domains. However, Lagdon and colleagues (2018) study demonstrated clear evidence, within one of the largest samples, for a stronger mediational effect for family social support than friend or significant other social support. The proximity and long-term nature of family relationships relative to peers may contribute to the greater mediational role here. The importance of the characteristics and sources in understanding the function of social support has been previously highlighted (Feeney & Collins, 2015; Taylor, 2012), and therefore represents an important candidate for further research. Considering the small mediation effect generally found, designs which confer greater power (i.e. the use of larger samples, control groups and

longitudinal testing), may be needed to tease apart the differential mediation roles related to the source of social support an individual receives, elicits, or seeks out.

### Social support as a moderating factor

The findings from the narrative synthesis indicate an inconsistent and variable picture regarding the role of social support as a moderator in the relationship between maltreatment and internalising symptomatology. Overall only half of the studies reported a significant moderation effect, and in the ones that did, these were found to vary as a function of sex, or indicate an opposite effect to those proposed in the stress-buffering model in which perceived social support attenuates the impact of stressful life events on the development of psychopathology (Sheldon Cohen & Wills, 1985). As such this review provides limited evidence as to the role of social support as a moderator.

Existing literature has detailed an association between sex and levels of perceived social support, with some finding that greater levels of social support in women than men (Turner & Lloyd, 1999), and substantially greater effects for the protective factor of social support against depression for women than men (Kendler, Myers, & Prescott, 2005). However, while there were apparent differences in the role of social support as a moderator as a function of sex within this review, these indicated opposing directionality for friend support as a moderator between abuse and internalising symptomatology for males and females. Importantly, other meta-analytic reviews have shown a sex/gender invariant effect of social support on its protection against PTSD within general populations (Prati & Pietrantonio, 2009). The role of sex on social support as a moderator therefore remains unclear.

It was striking that some findings indicated that high levels of social support were associated with greater levels of symptomatology in those who had experienced greater levels of maltreatment, in contrast to Cohen and Wills' stress-buffering hypothesis (1985). In such cases, social support may represent a risk factor for those who have experienced maltreatment. Two potential explanations for the lack of consistent and opposing findings of social support as a moderator are considered.

First, other authors have suggested that there may be a 'failing' of social support, or that the buffering effect is 'overpowered', at greater levels of trauma (Salazar et al., 2011; Sameroff, Gutman, & Peck, 2003). As such social support may not represent a protective *nor* a risk factor against symptomatology. Second, as shown by the synthesis of the mediational analyses, maltreatment may engender alterations in the way that individuals may utilise, elicit, or respond to social support. While the direct mechanisms by which this occurs are not within the remit of the review, these processes may alter the protective potential of social support at higher severities of maltreatment. In other words, these individuals are not able to utilise, elicit, or respond to social support in the same manner as those who have experienced less severe maltreatment. In some cases, individuals may potentially become despondent to available or perceived social support, or worse, act in ways that elicit negative social experiences. The subtle ways in which maltreatment may engender these differential alterations would be undetected by moderation analyses which are insensitive to the underlying mechanisms by which a person would interact with social support at high levels of stress and trauma and may result in lack of or opposing findings exhibited in this review. Therefore, mediation effects, and potential

differential underlying mechanism, may obscure or preclude the detection of moderation effects.

### Clinical implications

There are several clear clinical implications of these findings. First, the evidence demonstrating the role of social support as a direct protective factor highlights the importance of systemically focussed interventions that promote meaningful social interactions for maltreated individuals who may present with internalising symptoms. In light of the findings on the moderation and mediation effect of social support it appears that individuals with greater levels of maltreatment may respond in very different ways to social support with less severe maltreatment histories and may differ in their ability to utilise this support effectively. It is important to note the equivocal evidence for the effect of social support in the form of peers; this may have implications for interventions which rely heavily on social support and interaction (e.g. therapeutic groups), as these may have limited effect for some maltreated individuals who have experienced high levels of early trauma. Given the mediation findings, there is an impetus for researchers and clinicians to delineate the mechanistic process that underlie how maltreatment experience alters the subtle ways in which these individuals respond, elicit, or seek out social support. In doing so, we may be able to harness or focus in on these ‘adaptations’ through tailored therapeutic and systemically focussed interventions.

### Limitations of the assessed studies

Several limitations to the assessed studies should be acknowledged. Chiefly, there was noticeable substantial heterogeneity in the measures used to operationalise the concepts of maltreatment experience and social support. Previous meta-analyses have noted the disparate ways in which social support is measured (Gariépy et al., 2016), and within this review, 11 different measures were employed. Assuming consistency and validity across all measures to the concept of social support would be problematic. Additionally, for the most commonly used measure, the MSPSS (Zimet et al., 1988), confirmatory analysis has indicated that the significant other dimension also appears to measure both friend and family support at the same time and poses ‘serious conceptual and measurement problems’ (Cheng & Chan, 2004). This reflects the general social support literature which indicated very little agreement on how to operationalise social support (Vangelisti, 2009), and that many measures have poor or unreported psychometric properties (Uchino, Cacioppo, & Kiecolt-Glaser, 1996).

The challenges of measuring childhood have also been well documented (Fallon et al., 2010; Manly, Kim, Rogosch, & Cicchetti, 2001); attributable to the complexities of the construct which encompasses diverse physical and emotional experiences with multiple characteristics nested within each other. Similar, to social support, the ways in which maltreatment experience was measured by the studies was varied, with 11 different measures used across the included studies, with eight different ways characteristics were translated into an applied score. There was also a substantial weighting towards the use of retrospective and self-report measures of maltreatment, which have both been noted for psychometric weakness and potential inaccuracy (DiLillo et al., 2006; Widom, Raphael, & DuMont, 2004). Furthermore,

collapsing scores towards dichotomous presence variables may provide greater power towards identifying significant effects, but may in turn reduce the chance of identifying subtle effects relating to the characterisation of abuse that have been identified in other studies (e.g. Manly, Kim, Rogosch, & Hope, 2001). A recent systematic review highlighted the prevalence of inconsistencies in the measurement of maltreatment (Thornberry, Knight, & Lovegrove, 2012), and called for substantial changes in researchers approaches to measuring maltreatment.

An important limitation of the included studies was that many were cross-sectional, therefore prohibiting inference of the direction of associations between social support and internalising symptomatology. Last the recruitment of solely, or predominantly, female samples may not reflect the ‘gender symmetry’ in maltreatment prevalence that some researchers assert (Straus, 2011), and given the opposing moderation effects by sex, this factor should be included in future research.

### Strengths and limitations of this study

There are several strengths of the current study. This review represents the most comprehensive studies of the three proposed theories of social support in maltreatment research to date. The incorporation of both qualitative and meta-analytic approaches has allowed us to consider all three theories together to provide an inclusive and complementary understanding of social support. There is strength in the cautionary approach taken towards applying meta-analysis to the mediation and moderation outcomes, as doing so may have been inconsistent and potentially invalid, even if it was initially attractive. Furthermore, the inclusion and exclusion criteria can be recognised as a strength as it enabled us to identify a restricted number of articles,

while still representing generalisability across age, sex, maltreatment experience, and internalising symptomatology.

However, there are several limitations to this systematic review. First, there are numerous prevalence statistics and epidemiological studies that illustrate the comorbidity between internalising and externalising symptomatology (Angold, Costello, & Erkanli, 1999; Sallis et al., 2019). To look at one symptom cluster in isolation may be painting a biased picture of the role of social support but represents a clear candidate for future systematic reviews. Quantifying the pooled effect sizes for the moderation and mediation analyses would have represented a novel contribution to the existing literature, and its absence represents a prevailing limitation. However, without the use of more sophisticated approaches, such as meta-analytic structural equation modelling (MASEM), to do so with traditional methods would have misrepresented the data, inflating certain reported effect sizes over others.

There are several additional avenues to take for future studies within this area of research that have not already been highlighted. First, research into the underlying mechanisms by which social support mediates the pathway from maltreatment to internalising symptomatology is crucial, if clinicians are to operationalise such results into preventative interventions. Second, while the pervasive detrimental impact of childhood maltreatment has been extensively mapped across the lifespan, the impact of other socialised traumas is less well researched. Bullying, cyber bullying and community violence are only recently garnering attention in maltreatment research, but they represent significant risk factors that may interact with prior abuse and neglect in childhood, and equally social support within the community and school environments. Last, this systematic review did not consider how social support may

interact with concurrent therapeutic treatment for internalising disorders. Likelihood of treatment response is of considerable interest to managers and commissioners of therapeutic psychology services, more so now as services are under economic and political strain to demonstrate high rates of treatment response.

### Conclusion

In conclusion, this study represents the most comprehensive systematic review of the role of social support on the emergence and presence of internalising symptomatology in individuals who have experienced maltreatment. Social support was found to be a consistent protective factor, of small effect size, for these individuals across their lifespan and regardless of source of the support. The role of social support as a mediator was largely supported, however this appeared to vary by the source of social support and was of a small effect. This systematic review did not find reliable evidence to support the view that social support moderates the relationship between maltreatment experience and internalising symptomatology. It was suggested that the mediational processes may preclude the detection of moderation effects, or that social support may 'fail' or become 'overwhelmed' at the most severe levels of trauma. This research has implications for how clinicians may use systemically focussed interventions to utilise perceived social support and provides impetus for researchers to demonstrate the mechanistic processes that underlie the mediational effect.



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**Empirical Study:**  
**The sequelae of childhood maltreatment:  
a multi-level longitudinal investigation of  
brain structure, symptomatology and social  
support**

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## Abstract

- **Background:** Childhood maltreatment has been associated with a pattern of atypical cortical structure. However, extant literature has tended to be cross-sectional, with few studies examining the predictive nature of these differences on later symptomatology, or how they may be moderated by a resilience factor. The aim of the current study was to systematically investigate the relationship between regional differences in cortical structure (cortical thickness, local gyrification, and surface areas) associated with maltreatment and psychiatric symptomatology across two time points, two years apart, and how this relationship may be moderated by social support. **Method:** Thirty-three children with documented maltreatment and thirty-three matched controls underwent a structural MRI of their brain, alongside completing a battery of questionnaires relating to psychiatric symptomatology and current social support (T1). Two years following the MRI (T2), the participants completed the psychiatric symptomatology questionnaires again. **Results:** A broad pattern of reductions in local gyrification associated with maltreatment was exhibited, including in the superior frontal, superior parietal, fusiform and inferior frontal regions. In addition, the maltreated group displayed a relative increase in cortical thickness within the caudal middle frontal region compared to the non-maltreated group. There were no group differences in cortical surface area after correcting for multiple comparisons. Structural gyrification values within the superior parietal region were found to negatively correlate with T2 anxiety scores whilst controlling for T1 scores within the maltreated group, and was of small effect size. Furthermore, frequency of social support was found to moderate this relationship, however in a direction that was not predicted: such that social support benefitted those who exhibited reduced impact of maltreatment at a neural level. Of importance,

maltreatment severity was found to negatively correlate with social support importance and frequency.

- **Conclusions and implications:** Atypical gyrification within the superior parietal region associated with maltreatment may be predictive of later symptomatology. Furthermore, social support may be most beneficial in protecting against later symptomatology to those who present with the smallest cortical structural differences associated with maltreatment. Regional differences more broadly were not associated with symptomatology at T1 or T2, nor did social support present strong moderation effects. This suggests that examining a simple relationship between cortical structure, social support, and symptomatology in maltreated individuals may be a misplaced endeavor. Rather, it is proposed that a focus on intermediary processes may be more sensitive in delineating the functional importance of brain structural differences associated with maltreatment exposure.

## Introduction

### Childhood maltreatment

Childhood maltreatment continues to represent a pervasive and enduring public health and social-welfare concern (Gilbert et al., 2009). Notably conceptualized as a '*pathogenic relational experience*' (Cicchetti & Lynch, 1995), childhood maltreatment and its subtypes, physical, emotional, sexual abuse, and neglect, represents a robust predictor for a diverse range of mental health disorders in childhood and adulthood (Vachon, Krueger, Rogosch, & Cicchetti, 2015). Most notably a considerable body of evidence links maltreatment experience with depression (Anda et al., 2006), anxiety disorder (Scott, Smith, & Ellis, 2010), and borderline personality disorder (Widom, Czaja, & Paris, 2009) across the lifespan. Moreover, a myriad of physical health difficulties, including inflammation (Danese, Pariante, Caspi, Taylor, & Poulton, 2007) and heart diseases (Dong et al., 2004), have been associated with maltreatment. It is therefore unsurprising that maltreatment presents an enduring economic and societal burden (Conti, Morris, Melnychuk, & Pizzo, 2017), with difficulties in educational and economic attainment seen into adulthood (Nikulina, Widom, & Czaja, 2011).

Prevalence studies indicate that nearly one in five (18.6%) 11 to 17 year-olds in the UK have experienced some form of severe maltreatment in childhood (Radford et al., 2011). Third party organizations, such as the NSPCC and ChildLine, have reported large increases (between 21-55%) in contact and referrals for psychological interventions since 2010/11, specifically in relation to abuse and neglect (Bentley et al., 2017). In this context, understanding of the precise developmental pathways from

maltreatment experience to psychopathology is crucial if we are to identify and implement interventions that are effective, efficient and well targeted.

It is important to note that maltreated children follow a probabilistic developmental pathway, in which the early negative relational experiences serve to significantly increase the *likelihood* of poor outcomes (Cicchetti, 2013; McCrory & Viding, 2015). That is, adverse experiences are not deterministic. For those individuals where mental health difficulties do emerge, we are learning that psychiatric disorders in maltreated individuals emerge earlier, with greater severity, and a greater risk of comorbidity (Alvarez et al., 2011; Harkness & Wildes, 2002; Hovens et al., 2010). Moreover, individuals who have experienced maltreatment show a poorer treatment response compared to non-maltreated peers (Nanni, Uher, & Danese, 2012). Such evidence suggests that aetiology, and pathogenesis of psychopathology, are of relevance to the development of our approach to both prevention and intervention.

While the long-term impact of maltreatment is well documented, there is surprisingly little understanding of the specific and measurable underlying mechanisms of how early maltreatment elevates mental health risk in later life (McCrory, Gerin, & Viding, 2017). There is pressing need to understand these pathways at multiple levels of functioning, linking neurobiological, psychological, and social factors in ways that are meaningful and go beyond diagnostic categories (Cicchetti & Blender, 2006; Pollak, 2015).

## The neurobiology of maltreatment

At a neurobiological level, abusive experiences prompt a cascade of ‘stress-mediated effects on hormones and neurotransmitters’ that have an impact on the development of cortical structure and function (Teicher, Andersen, Polcari, Anderson, & Navalta, 2002). Extant literature has identified a number of localized differences in brain structure in maltreated individuals when compared to non-maltreated counterparts (McCrorry et al., 2017). A recent meta-analysis indicated a broad pattern of maltreatment associated grey matter volume reductions in orbitofrontal-limbic-temporal regions; areas that are implicated in top-down affect control (Lim, Radua, & Rubia, 2014). The most consistent findings arise in ventromedial and dorsolateral prefrontal cortex and lateral temporal areas, regions commonly implicated in the processing of fear and emotional regulation (Cha et al., 2014; Silvers, Wager, Weber, & Ochsner, 2015). Remarkably, these neural signatures are apparent in child and adolescent samples even in the absence of elevated psychiatric symptomatology (De Brito et al., 2012; Kelly et al., 2015). Moreover, these patterns of atypical cortical structure are distinctly similar to neural signatures indicated in adult populations with psychiatric diagnoses, such as anxiety and depression (Etkin & Wager, 2007; Kempton et al., 2011). As such these changes may reflect latent markers of vulnerability to later mental health problems (Kelly et al., 2015).

Grey matter volume, a metric that is investigated in the majority of these neuroimaging studies, is determined by two distinguishable cortical indices, cortical thickness (CT) and surface area (SA), which are purported to have distinct genetic influences and developmental trajectories (Hutton, Draganski, Ashburner, & Weiskopf, 2009; Panizzon et al., 2009). A limited number of studies have investigated

surface-based measures in relation to maltreatment experience, and have indicated specific and separable associations between maltreatment and CT and SA (Busso et al., 2017; Gold et al., 2016; Lim et al., 2017). In particular, a consistent and replicated finding across research labs, has been the identification of cortical thinning in the anterior cingulate cortex (Gold et al., 2016; Kelly et al., 2016; Lim et al., 2017), alongside an absence of SA differences within this region. Furthermore, localised patterns of maltreatment associated SA and CT reductions have not been found to display spatial overlap (Gold et al., 2016; Kelly et al., 2016). These findings would suggest that experiences of maltreatment might influence the development of separable cortical morphology in distinct ways. Therefore, there is a convincing rationale to investigate these cortical characteristics independently to gain greater precision to our understanding of the impact of maltreatment on underlying neurobiological systems.

### Latent Vulnerability and longitudinal investigations of maltreatment

The functional significance of patterns of neural differences may be conceptualized within the theory of latent vulnerability (McCrorry & Viding, 2015). The theory proposes that alterations across several neurocognitive systems may represent a calibration to abusive environments. It is proposed that these calibrations may incur long-term costs when the individual encounters subsequent normative environments, with such calibrations becoming maladaptive, increasing vulnerability to stressors across the life-span (McCrorry & Viding, 2015). These adaptations may not necessarily equate to symptomatology of psychiatric disorders, but contribute to the

emergence or pathogenesis of a disorder over time (McCrory & Viding, 2015). Understanding the pathogenesis of disorder following maltreatment is a necessary step if we are to inform models of preventative help for individuals at high-risk of poor outcomes.

However, currently we know surprisingly little about the functional significance of the observed structural differences in maltreated adolescents, and whether they represent true markers of latent vulnerability. The majority of prior studies have been cross-sectional in nature, thus limiting the directional and predictive inferences one is able to make. Current studies have highlighted the clinical significance of prospective imaging studies in predicting the onset of common mental health disorders (Foland-Ross et al., 2015). To our knowledge, there have only been four studies using maltreated samples of adolescents and adults (Busso et al., 2017; Gorka, Hanson, Radtke, & Hariri, 2014; Rao et al., 2010; Van Dam, Rando, Potenza, Tuit, & Sinha, 2014), that have examined prospective associations between cortical structure and psychopathology across all indices of cortical structure. Findings suggested that maltreatment related hippocampal volume predicted anxiety symptomatology (Gorka et al., 2014; Rao et al., 2010) and substance use relapse (Van Dam et al., 2014) at follow-up. Busso and colleagues (2017), represented the only prospective study to investigate surface-based measures (CT), finding that maltreatment-related thinning in a number of regions predicted later symptomatology, in particular demonstrating the predictive nature of middle-temporal gyrus thickness in the development of generalized anxiety disorder.

However, Busso and colleagues' (2017) investigation was characterized by several limitations. This study was restricted to a small sample of maltreated



individuals who experienced what the authors termed as ‘environmental threat’ (physical and sexual abuse), which may not reflect the interpersonal nature of the abuse, and overlooking what may be other important subtypes of maltreatment (neglect and emotional abuse). As such, this may not represent a particularly generalisable sample since maltreatment subtypes are highly interrelated and frequently co-occur (Cecil, Viding, Fearon, Glaser, & Mccrory, 2017). Furthermore, a selective region of interest approach on a sole measure of brain structure (CT) was used; this increases the likelihood that effects were not detected across the brain or in relation to other structural indices. To advance our understanding we need both (i) a larger longitudinal sample with documented experience of all common forms of maltreatment and (ii) a whole-brain approach across a number of surface-based indices.

### Resilience and social support

As mentioned previously, the relationship between experiences of maltreatment and psychopathology is probabilistic, such that it may serve to increase the *likelihood* of the development of psychiatric disorders. Therefore, not all individuals will go on to develop a psychiatric disorder. This indicates the importance of identifying internal and external factors that may represent resilience. Social support is one domain that has been consistently identified as a resilience factor to poor mental health outcomes in maltreated individuals (Jaffee, 2017). Social support is the perception and reality of other’s availability to provide emotional and material support within safe and nurturing relationships (Merrick, Leeb, & Lee, 2013). Socially supportive relationships may serve to promote positive outcomes as they ‘provide stability,

validate the individual's sense of self-worth, and help the individual avoid negative experiences' (Cohen & Wills, 1985).

Research has suggested that social support may operate in distinguishable ways for maltreated individuals in altering developmental trajectories of psychopathology. Some research indicates that social support can act as a *direct protective factor*, such that high levels of social support are associated with lower levels of clinical symptomatology regardless of maltreatment experience (Gagné & Melançon, 2013). However, further studies have indicated that social support may actually partially *mediate* the association between maltreatment and psychopathology, such that maltreatment experience impacts the manner by which individuals may elicit, utilize or respond to social support (Sperry & Widom, 2013; Vranceanu et al., 2007). Others suggest that social support *moderates* the relationship between maltreatment severity and clinical symptomatology, exerting a greater influence for those individuals who have experienced greater trauma, and less of an influence for those with lower levels or no trauma; described as the buffering hypothesis (Cohen & Wills, 1985). However, there is not a clear consensus as to the precise role that social support plays for maltreated individuals within the aforementioned hypotheses, or how it may interact at a neural level (Jaffee, 2017). Nonetheless, social support represents a tangible focus for preventive intervention in this population at high risk of the development of psychopathology. In integrating social support into a multi-level understanding of the neurobiological mechanisms by which maltreatment confers risk for later psychiatric disorders is an important step in helping to inform targeted and efficient preventative interventions that promote positive outcomes in this population of high-risk individuals.

## The current study

The aims of the study are twofold. The first aim is to systematically investigate the predictive nature of brain structural differences associated with maltreatment on future symptomatology in maltreated adolescents, compared to non-maltreated counterparts. Surface-based neuroimaging analyses on the structural scans of 33 children who have experience maltreatment and 39 non-maltreated peers matched on a range of demographic characteristics, and aged between 10-14 years, will identify group differences related to maltreatment experience, in specific and distinguishable cortical indices (cortical thickness, local gyrification, and surface area). The degree to which these structural brain indices predict clinical symptomatology two years later will then be explored. The second aim is to examine whether this relationship, between brain structure and future psychiatric symptomatology, is moderated by social support. All participant data has been collected from the 84 children and adolescents, recruited from London and the Southeast of England, and as such this empirical study uses secondary data collected by the author (Philip Kelly).

We hypothesise that structural differences associated with the experience of maltreatment will predict changes in symptomatology within maltreated individuals compared to non-maltreated individuals. Furthermore, we predict that the structural differences associated with maltreatment experience will be located primarily within cortical areas associated with emotional processing. Finally, we hypothesise that social support will moderate the relationship between structural differences associated with maltreatment and symptomatology, such that social support provides a greater

'protective' effect for those with more atypical cortical structure compared to non-maltreated individuals.

## Methods

### Participants

Seventy-three children and adolescents aged between 10-15 years were recruited from London and the South-East of England between September 2013 and November 2014. Children with documented exposure to maltreatment (MT;  $n=33$ ) were recruited via a London social services (SS) department and a post-adoption service. The process of recruitment involved identification and agreement of suitability of potential families by the SS team who would subsequently contact the family or foster family to introduce them to the research. The SS team would only put forward cases who were judged to be competent to assent and did not have a diagnosis of a learning disability. Families who expressed interest were then contacted by a member of the research team to arrange a home visit to describe the research, respond to questions and gain consent. In cases where the child was living with their biological parents, assent was obtained from the child (Appendix 2), and consent was obtained from one parent (Appendix 3). In shared parental responsibility cases, consent was gained from the biological parent of the child if contactable, and SS (Appendix 3). In adoptive cases, consent was obtained from one of the adoptive parents and assent from the adopted child (Appendix 3). All adopted children ( $n=9$ ) had been under the care of SS for maltreatment exposure but had now been placed in an adoptive (rather than foster) placement.

Non-maltreated comparison children (NMT;  $n=39$ ) were recruited from primary and secondary schools in the London area and via advertisement in Local and London wide newspapers. NMT children were matched on matched on age, self and

parent rated pubertal stage, sex ratio, handedness, cognitive ability, socio-economic status and ethnicity (Table 6). Children were excluded if there had been previous contact with SS regarding the maltreatment or quality of the care of the child. Consent was obtained from the child and their parents. The exclusion criteria for all participants included a diagnosis of learning disability, pervasive developmental disorder, neurological abnormalities, standard MRI contraindications (e.g. ferromagnetic implants, neurological disorder) and  $IQ < 70$ .

### Power analysis

Power analysis using the G Power program (Faul, Erdfelder, Lang, & Buchner, 2007) was used to compute the sensitivity for the between-group (ANCOVA) and multiple regression analyses. With group sizes of  $n=33$  and  $n=39$ ,  $\alpha=0.05$ , two groups and four covariates, there is sufficient power (set at the recommended  $1 - \beta=0.80$  level; Cohen, 1992) to detect large ( $f=.40$ ) effect sizes (outputted effect size  $f=.33$ ) for the between group analyses. For the multiple linear regression analyses, power to determine effect sizes was calculated for the proposed models with the greatest number of predictors. With a sample size of  $N=72$ , an  $\alpha=0.05$ , a total of two tested predictors, and six total predictors, there is sufficient power (set at the recommended  $1 - \beta=0.80$  level; Cohen, 1992) to detect medium ( $f^2=.15$ ) and large ( $f^2=.35$ ) effect sizes (outputted effect size  $f^2=.14$ , equal to partial  $R^2=.13$ ).

This community sample represents an inherently hard to recruit population and therefore statistical power is restricted by the number of participants that were able to be recruited within the initial two-year window of Time Point 1. However, this

sample constitutes one of the largest longitudinal studies to date into the impact of maltreatment on cortical structure.

## Measures

### *Maltreatment history*

The SS case files for the maltreated group were independently rated by the social worker on Kaufman's four-point scale (Adapted from scale; Appendix 4; Kaufman, Jones, Stieglitz, Vitulano, & Mannarino, 1994). Response were rated from 0 = 'no abuse present' to 4= 'evidence of severe abuse' in relation to neglect, physical abuse, sexual abuse, emotional abuse, and domestic violence. Only four (12.2%) individuals experienced one form of maltreatment, whereas a large proportion (15 individuals; 45.5%) of adolescents had experienced two forms of maltreatment. Furthermore, 14 (42.5%) adolescents had experienced three or more forms of maltreatment. The sample was largely characterized by a preponderance of histories of emotional abuse (96.97%) and neglect (75.76%). Severity scores also indicated that while emotional abuse was the most prominent, the mean level of neglect was the greatest across the sample (Appendix 11). Instances of physical and sexual abuse were low (9.10% for both subtypes) within this sample, and severity statistics indicated that when these experiences had occurred, the severity of the abuse was low.

### *Self-reported experience of maltreatment*

The Childhood Trauma Questionnaire (CTQ; Appendix 5; Bernstein & Fink, 1998) is a 28 item self-report inventory that assesses perceptions of five types of childhood abuse and maltreatment (i.e. emotional abuse, physical abuse, sexual abuse, physical

neglect and emotional neglect) was collected as an adjunct to the Kaufmann measure. Responses are provided on a 5-point scale from 'never true' to 'very often true'. The CTQ has high internal consistency and high overall convergent and discriminant validity (Bernstein et al., 2003). A composite total score was created from the summation of the five subscales to indicate a cumulative perceived experience of maltreatment and indicated high internal consistency ( $\alpha = .88$ ). Severity scores from the CTQ indicated a similar trend to the Kaufman responses with greater scores within the categories of neglect (physical and emotional) and emotional abuse, and lower scores within the physical abuse and sexual abuse subtypes (Appendix 11). Total severity on the Kaufman and CTQ scales was significantly positively correlated, to a moderate effect ( $r=.41, p>.05$ ).

#### Cognitive ability

All participants were administered the Vocabulary and Matrix reasoning subtests of the Wechsler Abbreviated Scale of Intelligence – Second Edition (WASI) to estimate full scale IQ (FSIQ; Wechsler, 2011). None of the participants in the combined sample scored below 70 or above 130 on the WASI.

#### Socio-economic status

Socio-economic status was assessed using information collected from the parent or care giver, including highest level of education and household income. Highest level of education was rated on a 6-point scale from 0 = 'no formal qualifications' to 5 = 'postgraduate qualification'. Household income was rated on an 8-point scale from 1 = '£0-10,000' to 8 = '£60,000 – 70,000+'.



### Pubertal status

Pubertal development was assessed with the five-item self-report and parent rated Puberty Development Scale (PDS; Girls version shown in Appendix 6; Petersen et al, 1988). The scale shows good internal consistency reliability ( $\alpha=.82$ ; Petersen, Crockett, Richards, & Boxer, 1988). The intra-class correlation between the parent and child responses was ICC=.83 suggesting ‘good reliability’ (Koo & Li, 2016).

### Psychiatric symptomatology

Measures of psychiatric symptomatology were taken at both time points, as shown in Figure 7. The analytic protocol is described extensively in the ‘Procedure’ section on page 99.

### *TSCC*

The Trauma Symptoms Checklist for Children (TSCC; Appendix 7) is a 44-item self-report measure of affective and trauma-related symptomatology (Briere, 1996), therefore relevant for the recruited sample. The measure has five clinical subscales, of which only three were included in this study due to clear analogues with diagnostic clusters (Depression, Anxiety, Post-traumatic stress), and two were excluded (Anger, Dissociation). Reliability and convergent validity of the scale has been previously investigated, and has been found to be ‘good’ in both domains (Crouch, Smith, Ezzell, & Saunders, 1999).

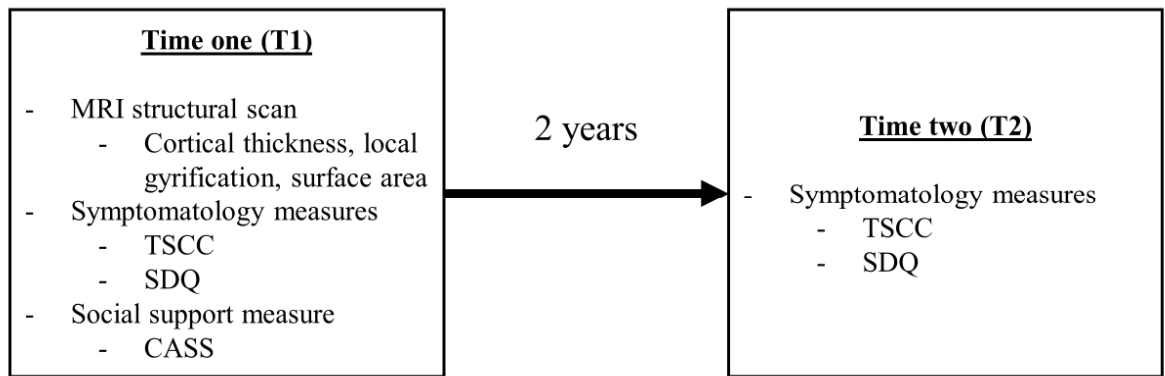
### *SDQ*

The Strengths and Difficulties Questionnaire (SDQ; Appendix 8) is a 25-item parent report measure that assesses general psychological and behavioural functioning

(Goodman, 1997). The SDQ includes five behavioural scales (Emotional symptoms, Conduct problems, Hyperactivity, Peer problems & Prosocial behaviour) and provides a composite score of total difficulties, which is utilised within this study. A recent systematic review has provided good evidence for discriminant validity, convergent validity, and internal consistency of the total difficulties scale (Kersten et al., 2016). The SDQ was selected due to its wide-spread international use, and its prevalence as a standard outcome measure within Child and Adolescent Mental Health services within the NHS.

### *Social support*

Social support was measured using the 40-item multi-dimensional Child and Adolescent Social Support Scale (CASS; Appendix 9; Malecki & Demary, 2002), which measures perceived social support from four sources: parents, teachers, classmates, and friends; and on two scales: frequency and importance. Frequency is rated on a 6-point Likert scale ranging from 1 (never) to 6 (always). Importance is rated on a 3-point Likert scale from 1 (not important) to 3 (very important). Subscale scores are calculated by summing the frequency or importance ratings on the 10 items for each source (e.g. parent, classmate). Total frequency and importance scores can be calculated by summing all four subscale scores. Test-retest reliability and internal consistency reliability was found to be 'good' to 'excellent' (Malecki & Demary, 2002). Social support was only measured at time one as indicated by Figure 7.



*Figure 7. Longitudinal process of collecting data*

### Structural image acquisition

Participants were scanned with a 1.5 Tesla Siemens (Siemens Medical Systems, Munich, Germany) Avanto MRI scanner with a 32-channel head coil. A high-resolution, three-dimensional T1-weighted structural scan was acquired with a magnetization prepared rapid gradient echo sequence. Imaging parameters are provided in Appendix 12.

### Procedure

Children and their families were invited to attend a magnetic imaging suite (BUCNI) at University College London to undertake a structural image of their brain and complete a battery of questionnaire, which will be referred to as time one (T1; Figure 7). As this study represents data collected from a larger longitudinal study, participants also completed several functional imaging paradigms, behavioural experiments and questionnaires not covered within this study. Participants and their families were remunerated for the cost of their travel and lunch and provided with a £20 Amazon voucher related to one of the behavioural tasks, a t-shirt, book, and an image of their brain on a CD. Participants were contacted approximately two years

subsequent to the initial testing to invite them to complete the battery of questionnaires again (T2; Figure 7). At T2, participants also undertook the aforementioned functional and behavioural tasks, as well as a second structural scan, however these will not be explored within this study. The study was approved by University College London Ethics Committee (0895/002). Royal Holloway ethics were obtained through the self-certification route on 31/01/18 (Appendix 10) for this research project.

### Structural image pre-processing

All images were initially manually inspected for any deformations that may impede its processing such as ‘drop-out’, movement artefacts or structural abnormalities. No participants within the sample were excluded due to deformations in the MRI image. The FreeSurfer surface based pipeline (Dale, & Fischl, 1999; Dale, Fischl, & Sereno, 1999; Fischl et al., 2004; Fischl, Sereno, & Dale, 1999) was used to process the T1 structural images into a standard space from which cortical thickness values could be derived on a participant by participant basis. The estimated total intracranial volume (eTIV) was calculated within FreeSurfer for each participant and no differences were found between the maltreated and non-maltreated groups (Table 6). The steps in this surface based morphometric pipeline have been described extensively and are well-validated (Dale et al., 1999; Fischl et al., 2004; Han et al., 2006) and are provided in Appendix 13.

### Cortical thickness and surface area measures

Cortical thickness (CT) at each vertex was measured by calculating the shortest distance from the white matter to the pial surface (in millimetres). The surface area (SA) was calculated at the pial level and represents the area of vertex on the gray matter surface, calculated as the average of the area of the tessellated triangles touching that vertex (Dale & Fischl, 1999). Gyral parcellation was based on the Desikan-Killiany atlas (Desikan et al., 2006) from which each participant's average surface area was calculated.

### Local gyrification index

The local gyrification index (IGI; Schaer et al., 2008) is an accompanying measure incorporated within the FreeSurfer analysis suite, provides a metric of gyrification of the cortical surface, and has been used to identify atypical gyrification across a number of psychiatric disorders (e.g Hyatt, Haney-Caron, & Stevens, 2012; Palaniyappan & Liddle, 2012). The IGI method uses the pial and white matter surface identification against an additional outer hull layer that tightly wraps the pial surface. The IGI value at each vertex is computed within 25mm circular regions of interest and represents the ratio of pial surface to outer hull surface, an indication of sulcal cortex buried in its locality and thus the extent of cortical folding. Schaer and Colleagues (2008) provide a comprehensive description of the analytic approach.

### Structural analysis

Regionally specific between group differences in cortical thickness and IGI were investigated on a whole-brain scale within the QDEC application of FreeSurfer using a general linear model. Cortical thickness measurements were smoothed with a full-width-at-half-maximum kernel of 15mm. Local gyrification index measurements were smoothed at 0mm, due to IGI maps being inherently smooth (given that GI is calculated in a radius of 25mm). Excessive smoothing of the IGI data can contribute to the failure of multiple comparisons. Between group differences were corrected for multiple comparisons with a Monte Carlo z-field simulation at  $p < 0.01$  (two-tailed).

Statistical analyses subsequent to the structural analysis were undertaken within SPSS 21 (IBM, 2016). The PROCESS toolbox for SPSS was used for moderation analyses (Hayes, 2012) where appropriate.

**Table 6.** Demographic and questionnaire data for the non-maltreated and maltreated groups

	<i>Non-maltreated (n = 39)</i>		<i>Maltreated (n = 33)</i>		<i>X<sup>2</sup></i>	<i>p</i>
Sex, N. of males (%)	15 (38.46)		17 (51.15)		1.23	0.27
Ethnicity, N. of Caucasian (%)	16 (41.03)		11 (33.33)		0.45	0.50
	<i>Mean (SD)</i>	<i>SD</i>	<i>Mean (SD)</i>	<i>SD</i>	<i>t-value</i>	<i>p</i>
Age (in years; T1)	12.62	1.17	12.22	1.63	1.15	0.25
PDS (T1) <sup>a</sup>	2.31	0.68	2.24	0.68	0.47	0.64
PDS (T2) <sup>a</sup>	3.10	0.48	2.92	0.65	1.18	0.24
<b>SES</b>						
Highest level of education	2.73	1.76	3.28	1.26	1.56	0.14
Income	3.88	2.1	2.87	2.34	1.69	0.10
Full Scale IQ	111.21	10.45	106.15	14.30	1.69	0.10
eTIV <sup>b</sup>	1486081.71	127065.22	1468277.34	132106.68	0.58	0.56
Global mean thickness	2.65	0.09	2.68	0.10	-1.18	0.24
<b>CASS <sup>c</sup></b>						
Total Frequency	243.11	30.20	224.90	36.18	2.28	0.03
Total Importance	118.08	20.73	113.00	20.53	1.00	0.32
	<i>Median</i>	<i>IQR</i>	<i>Median</i>	<i>IQR</i>	<i>t-value</i>	<i>p</i>
<b>Time 1</b>						
<b>TSCC <sup>d</sup></b>						
Anxiety	44.00	15	47.50	17	-1.88	0.06
Depression	41.00	15	44.50	12	-2.66	0.01
Post-traumatic Stress	42.00	11	47.00	10	-2.42	0.02
<b>SDQ <sup>e</sup></b>						
Total score	5.00	6	11.00	14	-4.64	0.00*
<b>Time 2</b>						
<b>TSCC <sup>d</sup></b>						
Anxiety	44.00	6	46.00	12	-0.90	0.34
Depression	44.00	9	45.50	8	-0.74	0.46
Post-traumatic Stress	42.00	12	46.00	10	-0.40	0.69
<b>SDQ <sup>e</sup></b>						
Total score	6.00	5	10.50	11	-2.09	0.04

<sup>a</sup> Puberty development scale time one (T1) and time two (T2); <sup>b</sup> Estimated Total Intracranial Volume; <sup>c</sup> Child and Adolescent Social Support Scale; <sup>d</sup> Trauma Symptom Checklist for Children; <sup>e</sup> Strengths and difficulties questionnaire.

Independent Samples t-test performed on transformed data. Untransformed medians and IQR presented for comparison with other samples and population. \* p<.001

All p values derived from t-tests except for sex and ethnicity comparisons which used Pearson's chi-square test. Separate variance estimates were used when homogeneity of variance assumptions was not met.

## Analytic protocol

The analytic protocol has five steps, detailed below:

1. Identification of regions of cortical structure associated with maltreatment experience.
  - a. *A general linear model will investigate localised differences associated with maltreatment by comparing MT and NMT groups, controlling for age, sex and FSIQ given their associations with the dynamic changes in CT and IGI (Giedd & Rapoport, 2010; Raznahan et al., 2011). eTIV and mean cortical thickness were included as covariates for IGI and SA, and CT respectively.*
    - i. *Mean structural value will be extracted from the significant cluster for subsequent analyses.*
2. Investigation of the association between cortical structure and maltreatment severity.
  - a. *Bivariate and partial correlations will explore correlations between regions of atypical structure and maltreatment severity (file report and self-report).*
3. Investigation of the relationship between cortical structure and symptomatology at T1.
  - a. *Partial correlations between cortical structure and symptomatology will explore relationships within the full sample.*
  - b. *A hierarchical linear regression with an interaction term of maltreatment experience and cortical structure will explore how relationship with symptomatology varies between the two groups.*



4. Investigation of the predictive nature of cortical structure on symptomatology change.
  - a. *Symptomatology change between T1 and T2 will be explored with repeated measure ANOVAS*
  - b. *Hierarchical linear regression will explore the amount of variance in T2 symptomatology explained by cortical structure associated with maltreatment experience, controlling for T1 symptomatology, and age, sex, FSIQ at the first level.*
    - i. *An interaction term between cortical structure and maltreatment will be included at the next level to explore how the relationship may vary by group.*
    - ii. *A theoretical approach will be employed to selection of candidate cortical regions given the potential for a large number of models and an increase in Type 1 error.*
5. Investigation of the role of social support in the relationship between cortical structure and symptomatology change
  - a. *Bivariate correlations will be undertaken between social support and symptomatology within the full sample and MT sample, and between maltreatment severity and social support*
  - b. *Moderation analysis of the relationship between cortical structure and symptomatology change, contingent on findings in analytic step 4.*

## Results

### Demographic characteristics

There were no statistically significant differences between the maltreated and non-maltreated groups in relation to age, sex ratio, ethnicity, FSIQ, pubertal stage, SES, and handedness (Table 6). As expected, measures of depression, anxiety and post-traumatic symptoms on the TSCC, and total SDQ scores, were found to differ significantly between the two groups, with the maltreated group reported higher symptomatology scores across the three indices (Table 6). However, group differences were only observed at T1 for TSCC, with T2 scores showing group differences only for total SDQ score.

### Exploration of data – outliers and normality of distribution

For all measures that were included in the analysis, the data were explored *by group* for detection of outliers and normality of distribution, given these assumptions are needed to be met for hierarchical linear regression and moderation analyses. Boxplots were created to identify outliers, and histograms, alongside analysis of skewness, and Shapiro-Wilk statistics were used to test for normality.

No outliers were detected that were greater than 2.2 times the inter-quartile range (IQR), therefore no scores were removed from the data set (Hoaglin & Iglewicz, 1987). Shapiro-Wilk tests indicated non-normal distributions within *both* NMT and MT groups on the measures of TSCC Anxiety ( $W=.94$ ,  $W=.90$  respectively;  $p<.01$ ), Depression ( $W=.93$ ,  $W=.92$ ;  $p<.01$ ), and PTSD ( $W=.88$ ,  $W=.89$ ;  $p<.01$ ), and on the Total SDQ score ( $W=.95$ ,  $W=.94$ ;  $p<.05$ ). Investigation of the histograms and

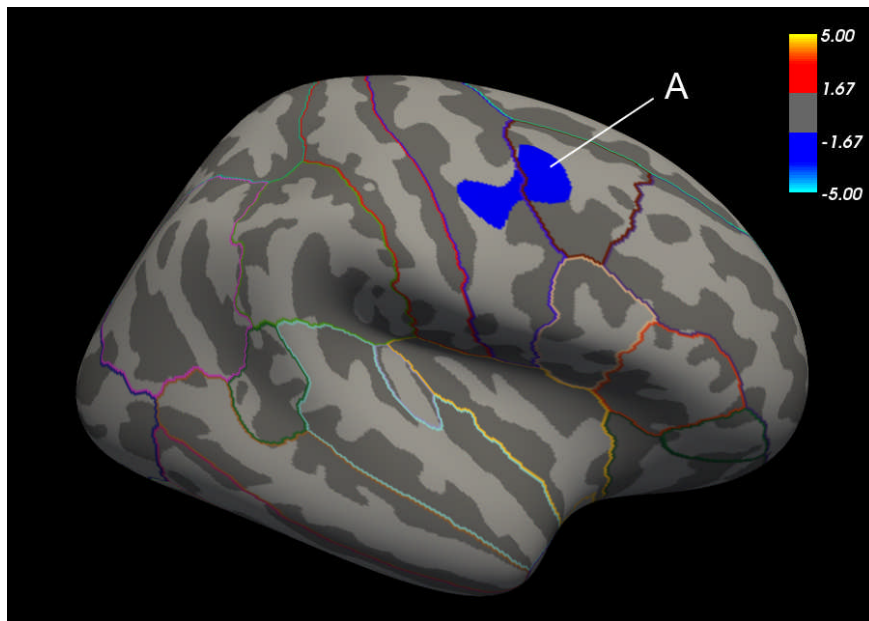
skewness statistics indicated a slight non-significant positive skew within all four measures. A log transformation was applied to these measures, which was successful in bringing the measures to normal distribution in both groups for the measures of Anxiety ( $W=.96$ ,  $W=.95$ ;  $p>.05$ ), Depression ( $W=.95$ ,  $W=.96$ ;  $p>.05$ ), and PTSD ( $W=.99$ ,  $W=.96$ ;  $p>.05$ ), and Total SDQ score ( $W=.98$ ,  $W=.97$ ;  $p>.05$ ). Total Frequency and Importance were found to have normal distributions and therefore were not transformed.

*The subsequent sections follow the analytic protocol set out in the methods.*

1. Identification of regions of cortical structure associated with maltreatment experience.

*Cortical Thickness*

The cortical thickness analysis identified one significant cluster, in the right hemisphere, that indicated increased cortical thickness within the maltreated group compared to the controls (cluster A, Figure 8; Table 7, Monte Carlo null-z simulation (MCZ) corrected  $p < .01$ ). Annotation based on the Desikan-Killiany parcellation Atlas (Desikan et al., 2006), of the group structural data indicated that the peak coordinate fell within the Caudal Middle Frontal region, with cluster extending across regions of the Precentral gyrus. No other significant clusters were detected that survived whole brain cluster correction within either the right or left hemisphere. Table 7 indicates the statistics for cluster A, including the coordinates, areal spread in  $\text{mm}^2$ , cluster-wide probability (p-cluster), calculated peak F value, and the maximum  $\log_{10}(\text{p-value})$  of the cluster.



**Figure 8.** Cluster-corrected region of increased cortical thickness in the caudal middle frontal and precentral gyri (A) within the maltreated group compared to the non-maltreated group.

Note: cluster was corrected at an  $\alpha=0.01$ . Colour bar depicts the direction (relative increase in blue or decrease in red of MT to NMT) and log-10 of the p-value of the cluster.

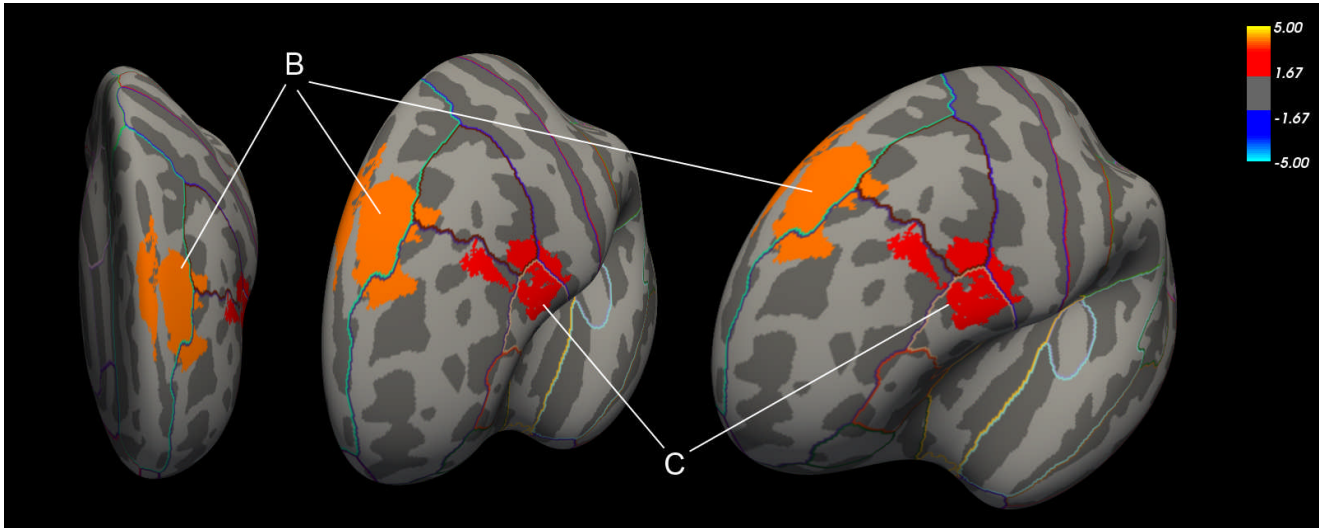
### Surface Area

Surface area values were extracted from gyri delineated using the Desikan-Killiany parcellation Atlas (Desikan et al., 2006) within FreeSurfer, and inputted into SPSS. A multivariate analysis was performed on the values to identify regions that differed in their average surface values between groups. Sex, age, FSIQ, and estimated intracranial volume were included as covariates. Four regions were identified as showing decreased surface area within the maltreated sample compared to the non-maltreated sample. Within the left hemisphere, the Isthmus cingulate ( $F(6,65)=5.01$ ,  $p=.03$ ) and within the right hemisphere, the Isthmus Cingulate ( $F(6,65)=8.32$ ,  $p=.01$ ), right parahippocampal ( $F(6,65)=5.64$ ,  $p=.02$ ) and right post central regions ( $F(6,65)=6.49$ ,  $p=.01$ ).

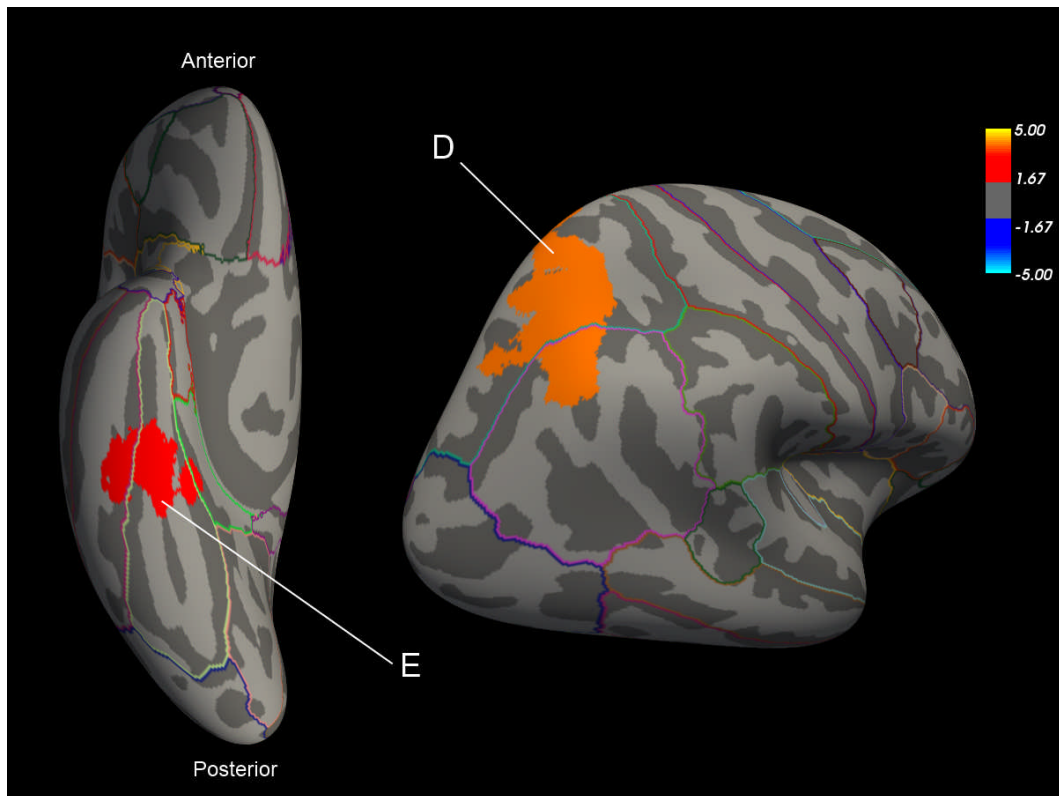
Given the number of multiple comparisons undertaken (34 per hemisphere), a step-up FDR correction was applied to the alpha value, providing a corrected significance value  $q^*=0.0015$  (Benjamini & Hochberg, 1995). None of the gyral region differences remained significant following this correction.

### Local Gyrfication Index

The local gyrfication analysis identified four significant clusters indicating reduced local gyrfication within the maltreated group compared to the non-maltreated group. Two significant clusters lay within the left hemisphere, with the first cluster's peak coordinate falling within the superior frontal region (cluster B, Figure 9; Table 7, MCZ corrected  $p < .01$ ) and the second cluster's peak coordinate falling within the Inferior Frontal Gyrus region, including aspects extending across Pars Opercularis, rostral middle frontal and caudal middle frontal regions (cluster C, Figure 9; Table 7, MCZ corrected  $p < .05$ ). Within the right hemisphere, the first cluster's peak fell within the superior parietal region, with the cluster extending into inferior parietal areas (cluster D, Figure 10; Table 7, MCZ corrected  $p < .01$ ), and the second cluster's peak falling within the fusiform gyrus, with the cluster primarily extending into inferior temporal regions, and slightly into parahippocampal regions (cluster E, Figure 10; Table 7, MCZ corrected  $p < .01$ ). All clusters survived cluster correction at a corrected level of  $p < .05$ , however clusters B and D also survived at a more stringent correction level of  $p < .01$ .



**Figure 9.** Cluster-corrected region of decreased local gyrification in clusters of the superior frontal gyrus (B) and inferior frontal gyrus (C) within the maltreated group compared to the non-maltreated group.



**Figure 10.** Cluster-corrected region of decreased local gyrification in clusters of the superior parietal gyrus (D) and fusiform/parahippocampal region (E) within the maltreated group compared to the non-maltreated group.

**Table 7.** Clusters of significant group differences for cortical thickness and local gyrification index, corrected for multiple comparisons

	Cluster no.	L/R	Peak F value	Max-log <sub>10</sub> (p-val)	P <sub>cluster</sub> <sup>a</sup>	Area (mm <sup>2</sup> )	Local Maxima (X, Y, Z)		
<b>Cortical thickness</b>									
<i>Control &gt; Maltreated</i>									
Caudal Middle Frontal/Precentral	A	R	6.79	-3.053	0.03	649.32	38.9	5.4	45.8
<b>Local gyrification</b>									
<i>Control &lt; Maltreated</i>									
Superior frontal	B	L	7.19	4.450	<.001	455.89	-20	30.8	48.0
Inferior Frontal Gyrus	C	L	5.09	2.148	<.001	664.48	-45.2	26.1	31.0
Superior parietal	D	R	6.86	3.184	<.001	731.97	30	-57.2	44
Fusiform /parahippocampal	E	R	6.78	3.675	<.001	595.52	41	-30.4	-20.6

L=left; R=right; <sup>a</sup> Cluster probability, All clusters were corrected for multiple comparisons using a monte-carlo null-z simulation at initial threshold of p<0.05 and adjusted for interhemispheric comparison

### Group differences on symptomatology at T1

A robust case has been suggested that when participants are not randomly assigned to groups, it is unsuitable to co-vary for variables inherently related to group assignment (Miller and Chapman, 2001). Consequently, anxiety, depression and PTSD symptomatology, known to be strongly associated with maltreatment experience, were not co-varied in the main analysis. Also given that these constitute a variable of interest at a later step in the analytic protocol, in which ‘baseline’ symptomatology (at time point one) would be controlled for, these variables were not included as covariates at this initial step.

## 2. Investigation of the association between cortical structure and maltreatment severity.

The Inferior Frontal Gyrus was negatively correlated with the CTQ measure of Emotional Neglect at a trend level only ( $r=-.31$  [CI 95% -0.56, -0.03],  $p=.07$ ), and the superior parietal region was positively correlated with Kaufman Neglect ( $r=.45$  [CI 95% -0.03, 0.71],  $p=.02$ ). However, the confidence interval for the superior parietal region passed through 0 thus we assume that this is not a significant effect.

Furthermore, neither correlations survived correction for multiple comparisons ( $q^*=0.002$ ; Benjamini-Hochberg approach for 30 correlations; Benjamini & Hochberg, 1995). No other structural region was found to be significantly correlated with the Kaufman total severity score (Appendix 14) nor the CTQ total score (Appendix 15).

## 3. Investigation of the relationship between cortical structure and symptomatology at T1

### *a. Partial correlations*

Table 8 displays the partial correlations within the full sample, indicating that there was significant negative correlation between structural values from the superior frontal cluster (cluster B) with depression symptomatology ( $r=-.28$ , (95% CI-0.45,-0.08),  $p=.02$ ). A negative partial correlation was also detected between structural values from the Fusiform gyrus cluster (cluster E) and total score on the SDQ ( $r=-.25$ , (95% CI-0.46,-0.10),  $p=.04$ ). Trend level ( $p<.07$ ) negative correlations were detected between structural values within the Fusiform gyrus and depression, the superior parietal cluster and total SDQ score, and the Superior Frontal cluster and total SDQ score. Zero-order correlations were also explored, due to the conservative nature of



the partial correlations. All correlational significance and direction remained the same, and r values were slightly larger within the partial correlations.

*Table 8. Partial correlations between structural values and symptomatology*

Structural Index	Structural cluster	TSCC			SDQ
		Anxiety	Depression	PTSD	Total
CT	<b>Caudal middle frontal (A)</b>	0.01	-0.01	0.06	0.06
		(-0.19,0.29)	(-0.21,0.23)	(-0.16,0.34)	(-0.14,0.28)
IGI	<b>Superior frontal (B)</b>	-0.07	-0.28*	-0.20	-0.23
		(-0.27,0.17)	(-0.45,-0.08)	(-0.35,-0.01)	(-0.43,0.02)
	<b>Pars Opercularis (C)</b>	-0.16	-0.186	-0.16	-0.14
		(-0.38,0.83)	(-0.41,0.04)	(-0.31,0.10)	(-0.31,0.10)
	<b>Superior parietal (D)</b>	-0.08	-0.21	-0.13	-0.23
	(-0.27,0.17)	(-0.43,0.04)	(-0.44,0.01)	(-0.01,0.12)	
	<b>Fusiform (E)</b>	-0.17	-0.23	-0.16	-0.25*
		(-0.39,0.08)	(-0.44,-0.02)	(-0.39,0.07)	(-0.46,-0.10)

CT = Cortical Thickness, IGI = local gyrification index, TSCC = Trauma Symptom Checklist for Children, SDQ = Strengths and Difficulties Questionnaire, \*p<.05; Bias corrected and accelerated (95% CI) bootstrapping applied and reported

*b. Interaction terms*

Hierarchical linear models were used to explore the interaction term between maltreatment experience (MT vs. NMT) and structural values that showed significant correlations in the full sample. The interaction term with the superior frontal values did not significantly contribute to an increase in the variance of TSCC Depression explained ( $\Delta R^2 = .001$ ,  $\Delta F(1,67)=0.54$ ,  $\beta^1=-.03$ ,  $t(66)=-0.23$ ,  $p=.82$ ). Furthermore, the interaction term between for the fusiform gyrus values did not significantly contribute

<sup>1</sup> Standardised Beta coefficients ( $\beta$ ) are reported throughout this study

to an increase in the variance of the total SDQ score explained ( $\Delta R^2 = .003$ ,  $\Delta F(1,67) = 0.24$ ,  $\beta = -0.07$ ,  $t(66) = 0.49$ ,  $p = .63$ )<sup>2</sup>.

#### 4. Investigation of the relationship between cortical structure and symptomatology at T2

##### a. Symptomatology change across time

Exploratory repeated measures ANOVAs were also used to explore whether there was significant change in symptomatology scores across time, or an interaction of time by group. By doing so, we may be able to identify candidate measures for this stage in the analysis and reduce the number of comparisons used. Only the measure of TSCC anxiety scale indicated a significant main effect of time ( $F(1,70) = 4.58$ ,  $p = .04$ ), with a trend of decreasing scores over time, however a non-significant interaction ( $F(1,70) = 2.13$ ,  $p = .15$ ). Whereas, TSCC PTSD showed an interaction between time and group ( $F(1,70) = 5.16$ ,  $p = .03$ ) and no main effect of time ( $F(1,70) = 2.80$ ,  $p = .10$ ). Exploring the profile plot of this interaction indicated that the MT group were reporting lower levels of symptomatology between T1 to T2, while the NMT group displayed similar levels across time.

##### b. Association with cortical structure

Lack of an interaction between structure and maltreatment experience at time one, does not preclude that these structural variables may be associated with T2 symptomatology. Therefore, T2 symptomatology was firstly explored within the two

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<sup>2</sup> The assumptions for absence of multi-collinearity (via VIF scores), homoscedasticity (via scatter plots), normality of residuals (via a histogram), and linearity (via scatter plots) were all met for all hierarchical linear models described in the results section.

regions that indicated significant correlations at time one, superior frontal and fusiform gyrus.

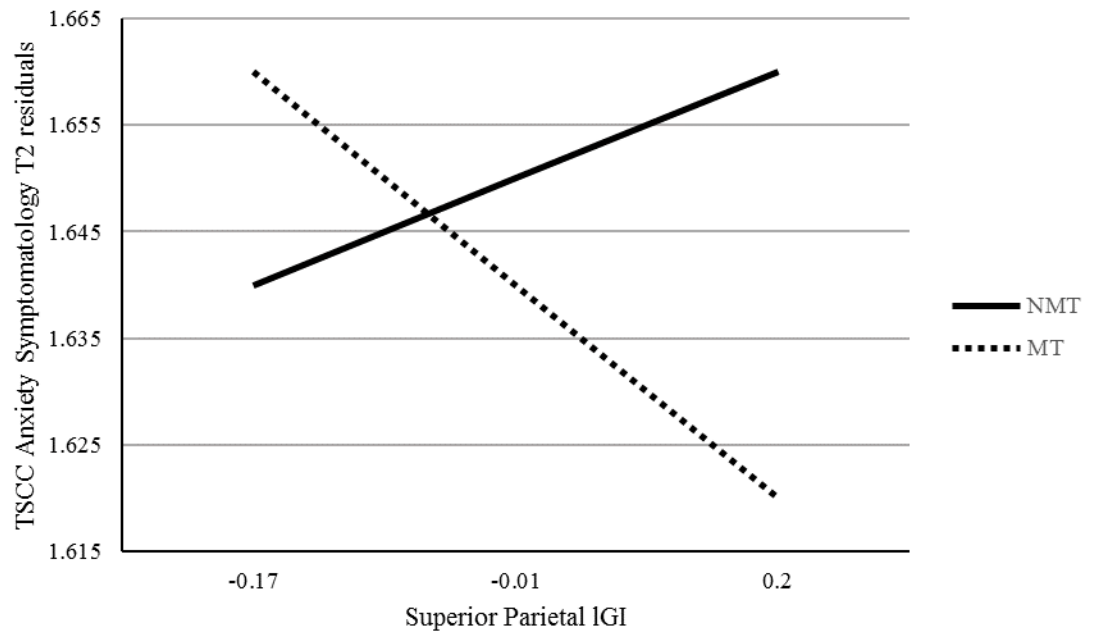
- Within the model exploring the association between superior frontal and T2 symptomatology in TSCC depression, inclusion of the superior frontal variable into the model did not significantly increase the amount of variance explained by the model ( $\Delta R^2 = .003$ ,  $F(1,64)=0.37$ ,  $p=.55$ ,  $\beta =.06$ ,  $t(64)=.61$ ,  $p=.55$ ). The inclusion of the interaction term similarly did not significantly contribute to the variance of symptomatology change ( $\Delta R^2 = .023$ ,  $\Delta F(1,65)=2.62$ ,  $\beta =-.16$ ,  $t(64)=-1.62$ ,  $p=.11$ ).
- Structure of the fusiform did not significantly contribute to an increase in variance explained in the model of T2 symptomatology within the total SDQ score ( $\Delta R^2 = .008$ ,  $\Delta F(1,65)=.52$ ,  $p=.47$ ,  $\beta =.09$ ,  $t(64)=.72$ ,  $p=.47$ ). Including the interaction term at the next level, similarly did not contribute an increase in the variance explained by the model ( $\Delta R^2 = .007$ ,  $\Delta F(1,65)=.49$ ,  $p=.49$ ,  $\beta =.14$ ,  $t(64)=.70$ ,  $p=.49$ ).

Given that there was a main effect of time for TSCC anxiety and a significant interaction term for TSCC depression, these variables were investigated as to whether cortical structure at T1 may be predictive of T2 symptomatology. A theoretical approach was taken to identify candidate structural regions that may be associated with T2 anxiety and PTSD, thus restricting the number of models implemented and limiting the possibility of type 1 errors, and ‘p-hacking’. The region of superior frontal cortex has shown altered functional activation (Garfinkel & Liberzon, 2009), and significant volumetric reductions within individuals with PTSD (O’Doherty et al.,

2017), and was therefore selected as a candidate region. Aspects of the superior parietal region have been associated with atypical activation in those with anxiety disorders (Bruder et al., 1997) and atypical cortical maturation within this region in adolescents has been associated with trait anxiety (Newman et al., 2016), thus this region was chosen as a candidate.

- Exploring symptomatology change in PTSD, the inclusion of superior frontal values at T1 did not significantly contribute to the variance explained ( $\Delta R^2 = .02$ ,  $\Delta F(5,66) = .92$ ,  $p = .41$ ,  $\beta = -.06$ ,  $t(66) = -.64$ ,  $p = .53$ ), nor did the interaction term with maltreatment experience ( $\Delta R^2 = .01$ ,  $\Delta F(6,65) = 1.29$ ,  $p = .26$ ,  $\beta = -.11$ ,  $t(65) = -1.34$ ,  $p = .26$ ).
  - However, within the model exploring T2 TSCC anxiety, while the inclusion of superior parietal values did not significantly contribute to the variance explained in the model ( $\Delta R^2 = .00$ ,  $\Delta F(5,66) = .03$ ,  $p = .87$ ,  $\beta = .02$ ,  $t(66) = .16$ ,  $p = .87$ ), there was a significant interaction term (adjusted  $R^2 = .38$ ,  $\Delta R^2 = 0.04$ ,  $\Delta F(6,65) = 5.00$ ,  $p = .03$ ,  $\beta = -.27$ ,  $t(65) = -2.24$ ,  $p = .03$ ). Nevertheless, this was a small effect, and represented only a 4% increase in the variance explained. Examining the slopes for the MT and NMT group, indicated that only within the maltreated group was the association between cortical structure and Anxiety symptomatology change marginally significant ( $b = -.17$ ,  $t(66) = -2.3$ ,  $p = .04$ ). Figure 11 displays the direction of the interaction, such that a lower local gyrification index was associated with greater amount of symptomatology at T2. However as this was not a particularly strong

interaction effect, it did not survive multiple comparison correction ( $q^* = 0.013$ ; Benjamini & Hochberg, 1995) when correcting for the four models run in this step of the analytic protocol.



**Figure 11.** Chart representing the interaction between maltreatment experience and cortical structure on T2 anxiety symptomatology

Together these findings indicate that cortical structure differences associated with maltreatment experience do not significantly correlate with symptomatology at T1, within the full sample or as function of maltreatment experience, or at T2 *after* controlling for multiple comparisons.

## 5. Investigation of the role of social support in the relationship between cortical structure and symptomatology at T2

### a. Bivariate correlations between social support and symptomatology, and maltreatment severity and social support

Bivariate correlations were undertaken to explore the correlation between total frequency and importance scales of the CASS and symptomatology scales at T1 and T2 within the full sample. At T1, only total frequency was significantly negatively correlated with total difficulties on the SDQ ( $r=-.31$  [95% CI -0.56,-0.02],  $p<.05$ ), but this relationship was not apparent at T2. However, at T2 total frequency was negatively correlated with Anxiety and PTSD scales of the TSCC, but this effect was not present at T1 (Table 9). Total importance was not significantly correlated with any of the symptomatology scales.

**Table 9.** Correlations between social support and symptomatology at T1 and T2

CASS	TSCC T1			TSCC T2			SDQ T1	SDQ T2
	Anxiety	Depression	PTSD	Anxiety	Depression	PTSD	Total	Total
<b>Total Frequency</b>	-0.24 [-0.51,0.06]	-0.17 [-0.43,0.10]	-0.24 [-0.45,0.01]	-0.40* [-0.60,-0.16]	-0.24 [-0.48,0.09]	-0.32* [-0.54,-0.07]	-0.31* [-0.56,-0.02]	0.12 [-0.16,0.38]
<b>Total Importance</b>	0.11 [-0.15,0.34]	0.19 [-0.07,0.41]	0.15 [-0.12,-0.39]	-0.05 [-0.34,0.23]	-0.09 [-0.22,0.43]	-0.02 [-0.29,0.27]	-0.06 [-0.30,0.17]	0.08 [-0.12,0.26]

TSCC = Trauma Symptom Checklist for Children, SDQ = Strengths and Difficulties Questionnaire, CASS= Child and Adolescent Social Support Scale; \* $p<.05$ , \*\* $p<.001$ ; Bias corrected and accelerated (95% CI) bootstrapping applied and reported

The relationship between maltreatment severity and social support within the maltreated group was investigated. Table 10 displays the correlation table for total severity scores. Total maltreatment severity as rated by the file-report Kaufman measure was significantly negatively correlated with both total frequency and

importance of social support. Self-report total severity as measured by the CTQ was significantly negatively correlated with total frequency of social support, but not with total importance of social support (**Table 10**).

Exploring correlations by maltreatment subtype (

Appendix 16 & 17) indicated that neglect exhibited the most consistent and strongest negative correlations with social support. Self-reported emotional abuse was also found to exhibit a moderate negative correlation with social support frequency ( $r=-.48$ , [95% CI -0.78, -0.03],  $p<.05$ ). The strongest negative correlation was observed between self-reported emotional neglect and social support frequency ( $r=-.63$ , [95% CI -0.81, -0.31],  $p<.05$ ), of large effect size. Physical abuse, sexual abuse and domestic violence were not consistently found to significantly correlate with either dimension of social support.

Exploring the correlation between maltreatment severity and subscales of the social support scale (

Appendix 16 & 17), indicated that Maltreatment severity (primarily driven by neglect) appeared to have the greatest impact on classmate and close friend social support frequency and importance than other sources of social support, for both self-report and file-report measures. The CTQ also demonstrated that maltreatment severity broadly negatively correlated with frequency of parental social support. Overall, these findings suggest that more severe experiences of independently documented maltreatment are associated with lower reported experiences of social

support. Furthermore, it suggests that higher levels of maltreatment within the home (particularly of neglect) may have the greatest impact on the social relationships with peers outside of the home/in school.

**Table 10.** Correlations between self-report and file report measures of maltreatment severity and social support

<b>Social support</b>	Kaufman Total Severity	CTQ Total Severity
<b>Total Frequency</b>	-0.41*	-0.61*
	[-0.74,-0.03]	[-0.80,-0.35]
<b>Total Importance</b>	-0.42*	-0.24
	[-0.65,-0.18]	[-0.59,0.05]

\*p<.05; Bias corrected and accelerated (95% CI) bootstrapping applied and reported

*b. Moderation effect of social support on cortical structures relationship to symptomatology change*

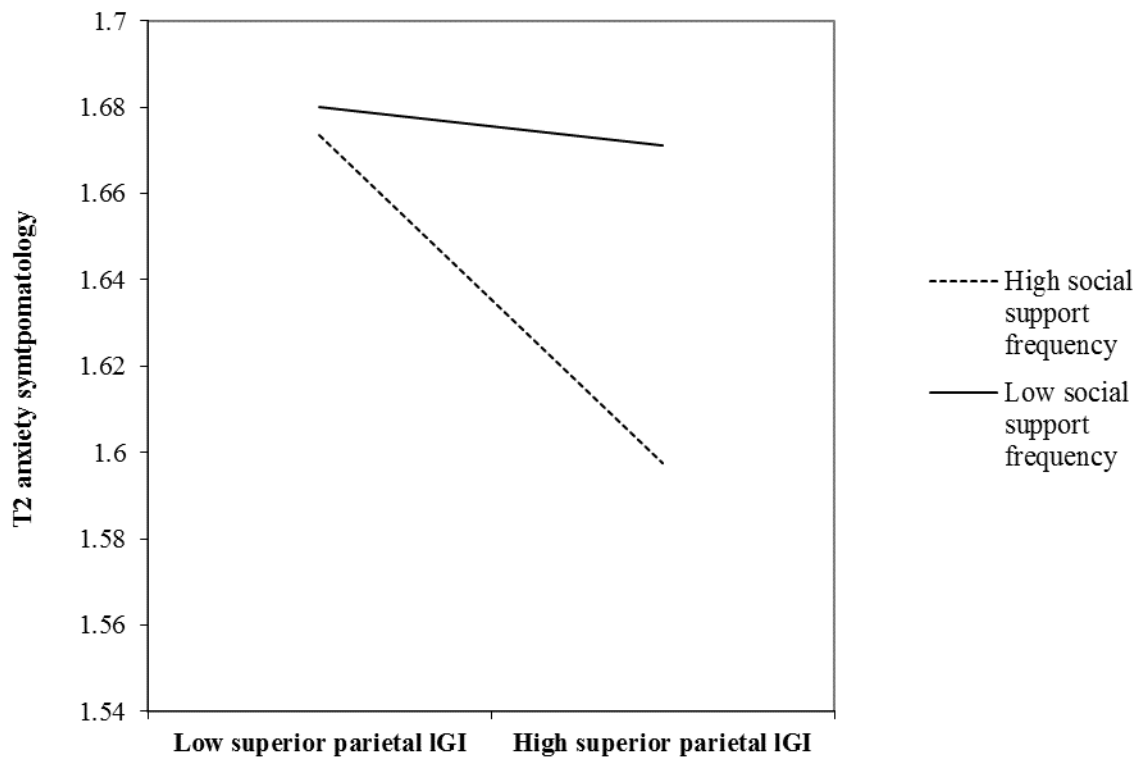
In the next step, an exploratory approach was taken to focus in on those individuals in the maltreated group who receive high levels of social support, and to discern on a neural level whether they benefit from a ‘protective’ related to social support. As the association between superior parietal IGI and Anxiety symptomatology change was found to be significant within the maltreated group, analysis was undertaken as to whether social support moderates this association. As there are two total domains of social support, total importance and total frequency, two models were run within the maltreated group.

Total importance was not found to significantly moderate the association between IGI in the Superior Parietal region and symptomatology change in anxiety ( $\Delta R^2 < .001$ ,  $\Delta F(1,25)=0.01$ ,  $\beta = < .001$ ,  $t(25)=0.08$ ,  $p=.94$ ). However, total frequency was found to moderate the association between cortical structure and symptomatology



change in anxiety ( $\Delta R^2 = .10$ ,  $\Delta F(1,25) = 7.80$ ,  $\beta = -0.21$ ,  $t(25) = -2.79$ ,  $p = .01$ ). Plotting the conditional effects of the superior parietal IGI at tertile values (low, average, high) of social support indicated that only at high ( $\beta = -0.12$ ,  $t(25) = -2.50$ ,  $p = .02$ ) and average ( $\beta = -0.22$ ,  $t(25) = -3.03$ ,  $p = .01$ ) levels of social support was IGI negatively associated with anxiety symptomatology change. At low levels of social support there was a lack of significant association between cortical structure and symptomatology change ( $\beta = -0.05$ ,  $t(25) = -1.11$ ,  $p = .28$ ). Figure 12 visualizes the moderation of the relationship between IGI and T2 anxiety symptomatology at high and low levels of support, such that the high levels of social support appear to be beneficial only for those with the smallest differences in cortical structure associated with maltreatment. Greater levels of social support frequency do not appear to have an impact on the association between IGI and T2 anxiety for those with the greatest group cortical differences (low superior IGI).

**Moderation of the effect of superior parietal IGI on T2 anxiety symptomatology at values of the moderator social support frequency**



*Figure 12. Moderation of superior parietal LGI on T2 anxiety symptomatology by social support, at conditional effects of high and low support*

## Discussion

The current study sought to systematically investigate the relationship between regional differences in cortical structure (CT, IGI, and SA) associated with maltreatment and psychiatric symptomatology. Subsequent analyses explored whether regional differences associated with maltreatment were associated with change in symptomatology across a two-year period. Furthermore, the study sought to investigate the role of social support in moderating this relationship.

### *1. Identification of regions of cortical structure associated with maltreatment experience.*

We found that maltreatment exposure was associated with an increase in CT within a caudal middle frontal cluster, IGI decreases within regions of the superior frontal gyrus, inferior frontal, superior parietal region and the fusiform gyrus/parahippocampal regions, but an absence of significant associations with the index of SA after correction for multiple comparisons. These findings were generally consistent with our predictions. However, the direction of the CT findings was unexpected, given that there is a general trend towards a pattern of structural decreases associated with maltreatment exposure (Lim et al., 2014). Nonetheless, relative increases within right middle frontal regions have been consistently noted within volumetric studies, as evidenced by a recent meta-analysis (Lim et al., 2014), and within CT studies of individuals who have experienced traumatic events (Lyo et al., 2011). Studies within clinical populations have additionally found regional increases in CT within youth with major depressive disorder (MDD; Reynolds et al., 2014), and gyrification abnormalities associated with MDD within adult clinical samples (Depping et al., 2018). Functionally the structural differences within this

region have been linked with adaptive emotion regulation strategies during adolescence (Vijayakumar et al., 2014) and within clinical samples (Bruehl et al., 2013). Therefore, such findings may be relevant for understanding the role of this region in regulating emotion within high-risk samples, such as those who have been exposed to maltreatment.

Regional decreases in IGI were detected in both hemispheres and may reflect existing findings of reductions in cortical structure associated with maltreatment in superior frontal, parahippocampal, and parietal regions, consistently reported in regards to the index of grey matter volume (Lim et al., 2014). As such, these regions may represent a reliable correlate of maltreatment experience across both the structural measures of cortical volume and local gyrification. Furthermore, the structural decreases detected in the parahippocampal region are of significance given recent findings which demonstrate the mediational role of the region on the relationship between childhood abuse and antisocial behaviour (Busso et al., 2017)

The detection of structural decreases within the fusiform gyrus and the superior parietal region, may also be of interest. Existing literature has consistently supported a proposed association between the fusiform region and facial recognition (Furl, Garrido, Dolan, Driver, & Duchaine, 2011; Kanwisher, McDermott, & Chun, 1997), and studies have detailed the association of the superior parietal region with the decoding of high level features of faces and facial representation (Sarkheil, Goebel, Schneider, & Mathiak, 2013). Functional connectivity studies have similarly highlighted the positive correlation between fusiform areas and parietal regions during face-matching tasks (Bokde et al., 2006). It has been shown that maltreatment experience is associated with an increase in perceptual information required to

recognize sad facial expressions (Pollak & Sinha, 2002), selective attention to facial emotion (Pollak & Tolley-Schell, 2003), and enhanced reactivity to emotional faces in the parietal region (van Harmelen et al., 2012). Moreover, maltreatment severity has been found to positively correlate with fusiform activity during novel face processing (Edmiston & Blackford, 2013). Considering these findings, one might speculate that the observed structural differences represent correlates of atypical facial processing in maltreated samples. However, further studies explicitly exploring structural-functional correlations in facial processing is needed to support any such inferences.

IGI decreases within the superior frontal region are of significance given prior research showing a greater recruitment of superior prefrontal regions during cognitive control of emotion among maltreated youth (McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015), and structural deficits within this region in individuals with PTSD (O'Doherty et al., 2017), a disorder in which cognitive control, especially of emotion, is significantly impaired (Aupperle, Melrose, Stein, & Paulus, 2012). Furthermore, severity of maltreatment has been found to positively correlate with Inferior Frontal Gyrus during mentalization (van Schie et al., 2017), a process by which individuals mirror others' thoughts and emotions vital for effective interpersonal functioning. As such, these findings are in line with our predictions that regional differences associated with maltreatment would lie within regions associated with emotional processing; although it appears that these regions are associated with higher order emotional processing. The lack of findings for the SA index after applying multiple comparisons correction was surprising given previous findings of SA decreases associated with maltreatment within a subset of this sample (Kelly et al., 2013). However the current knowledge regarding the impact of maltreatment on

surface area is relatively sparse and inconsistent compared to volumetric techniques (Lansing, Virk, Notestine, Plante, & Fennema-Notestine, 2016; Opel et al., 2019), thus further research is needed to provide an understanding to the factors that may contribute to the presence or absence of atypical surface area in maltreated individuals.

## 2. Cortical structure and maltreatment severity.

Structural decreases within right parietal and inferior frontal regions have previously been associated with increases in CTQ scores in adult and adolescent samples (Dannowski et al., 2012; Edmiston et al., 2011), however similar correlations with the superior parietal cluster, nor with other clusters, were not reflected within this sample. It may be interpreted that structural differences are only detected at a group level, and do not provide subtle differentiation between characteristics of the maltreatment experience. A recent systematic review of structural findings associated with maltreatment highlighted the lack of investigations into the moderating effect of maltreatment severity on structure and function in maltreated samples (Cassiers et al., 2018), consequently future studies would be needed to differentiate group-level vs. dose-response effects of maltreatment on structure within the same sample.

## 3. Structure and symptomatology at T1.

The findings regarding the associations between cortical structure and symptomatology, suggest that maltreatment related cortical differences were not reliably associated with symptomatology at T1. While there were two significant correlations, between the superior frontal cluster and depression, and between the fusiform cluster and total SDQ, the interaction terms were not found to be significant,

such that there wasn't a differential relationship between cortical structure and symptomatology as a factor of maltreatment exposure. Similar studies have also found relatively few associations between CT and symptomatology scales (Busso et al., 2017; Whittle et al., 2013). Busso and colleagues (2017) undertook 96 separate correlations between regions of interest sensitive to abuse and symptoms of psychopathology but found only six correlations reached significance after correcting for multiple comparisons (17 uncorrected). This is consistent with the view that there is unlikely to be a direct linear relationship between cortical structure and symptomatology severity. It is perhaps more plausible to assume that structural differences associated with maltreatment experience reflect alterations in common underlying cognitive mechanisms rather than disorder specific processes. This highlights an important theoretical consideration for the role of cortical structure in understanding trajectories towards psychiatric symptomatology, which will be explored further in the 'Key considerations' section on page 130.

#### 4. Structure and T2 symptomatology.

The detection of a significant interaction between maltreatment exposure and superior parietal gyrification on T2 anxiety symptomatology suggested that this structural difference associated with maltreatment may be predictive of later symptomatology within the maltreated group. The parietal lobe plays a vital role in visuospatial processing, especially during threat detection (Bremner, 2004), a salient process within anxiety disorders (Cisler & Koster, 2010). Prior findings have also indicated towards atypical cortical maturation within this region in adolescents with trait anxiety (Newman et al., 2016). Atypical cortical structure within this region *may* represent a neural correlate of atypical visuospatial processing and threat detection,

and thus relevant in the emergence and maintenance of anxiety symptomatology within the maltreated sample. Lack of findings at T1 may tentatively indicate that underlying visuospatial process exert a protracted and gradual influence on the emergence of anxiety symptomatology, only detectible through psychometric measures after several years. Nonetheless the size of this effect was small and the regression slope within the maltreated group was marginally significant, not surviving multiple comparisons correction. Therefore, caution should be taken in generalizing from this result to wider populations of maltreated individuals.

#### 5. Social support

Lack of consistent associations between social support and symptomatology within the full sample was surprising, given the existing evidence of social supports role as a protective factor against depression in the general population (Gariépy, Honkaniemi, & Quesnel-Vallée, 2016), and in maltreated samples, as demonstrated in the systematic review component of this thesis. Lack of significant associations may reflect a general trend for the full sample to present with low levels of symptomatology, as indicated by the median t-scores for the TSCC scales sitting within an ‘average’ range for both groups. Importantly, while there were expected group differences on the social support scales, reflecting numerous findings demonstrating lower levels of social support in individuals with exposure to maltreatment (Horan & Widom, 2015; Lamis, Wilson, King, & Kaslow, 2014; Sperry & Widom, 2013), the findings of negative correlations between maltreatment severity and social support importance, and more-so frequency, have noteworthy implications. First this implies that greater severity of maltreatment may alter the ways in which these individuals elicit or utilize social support. Neglect appeared to engender the



greatest negative impact on social support, however as the sample were largely characterized by neglect and emotional abuse, one cannot make firm inferences about the specific roles of maltreatment subtypes within this relationship. Second, the findings suggests that maltreatment experience within the home has an impact on non-family relationships, outside of the home and within school to the greatest extent, which makes it less likely that atypical behaviour by the child's family is driving the potential difficulties in eliciting or utilizing social support. These findings provide support for the theory that potential short-term calibrations to available relationships and socialized support within the context of maltreatment may represent maladaptive processes in normative environments to other forms of socialized support, such as in the classroom and out of the home (McCrory & Viding, 2015).

The exploratory findings of a significant moderation by social support frequency of the predicative association between superior parietal IGI and later anxiety symptomology at T2 within the maltreated group, were unpredicted given the direction of the effect opposite to the proposed role of social support in the 'buffering hypothesis' (Cohen & Wills, 1985). It appeared that greater frequency of social support exerted greater benefits only for those with the smallest differences in cortical structure associated with maltreatment. One interpretation of this finding is that alterations within the superior parietal region may represent certain cognitive or affective processes that interfere with the ability to utilize available social support resources. As previously described, superior parietal regions have been associated with higher level facial feature processing (Sarkheil et al., 2013) and the visuospatial detection of threat (Bremner, 2004); both processes likely to impair iterative social interactions and mentalizing abilities if attuned to subtle facial predictors of threat

(Chanes, Baumann Wormwood, Betz, & Feldman Barrett, 2018; Fonagy & Bateman, 2016). This is also supported by the aforementioned link between maltreatment severity and frequency of social support; that those who have the greatest level of trauma may be less able to elicit or utilize social support. These changes may therefore represent a calibration that is advantageous in abusive and threatening environments but confers a disadvantage in normative social situations. As with the finding of associations between cortical structure and longitudinal change, the size of this effect was small (the interaction term represented 10% of the variance explained in T2 symptomatology), and the previous inferences should be taken with caution without further systematic investigations into relationships between superior parietal structure and iterative social interaction in maltreated individuals.

### Key considerations

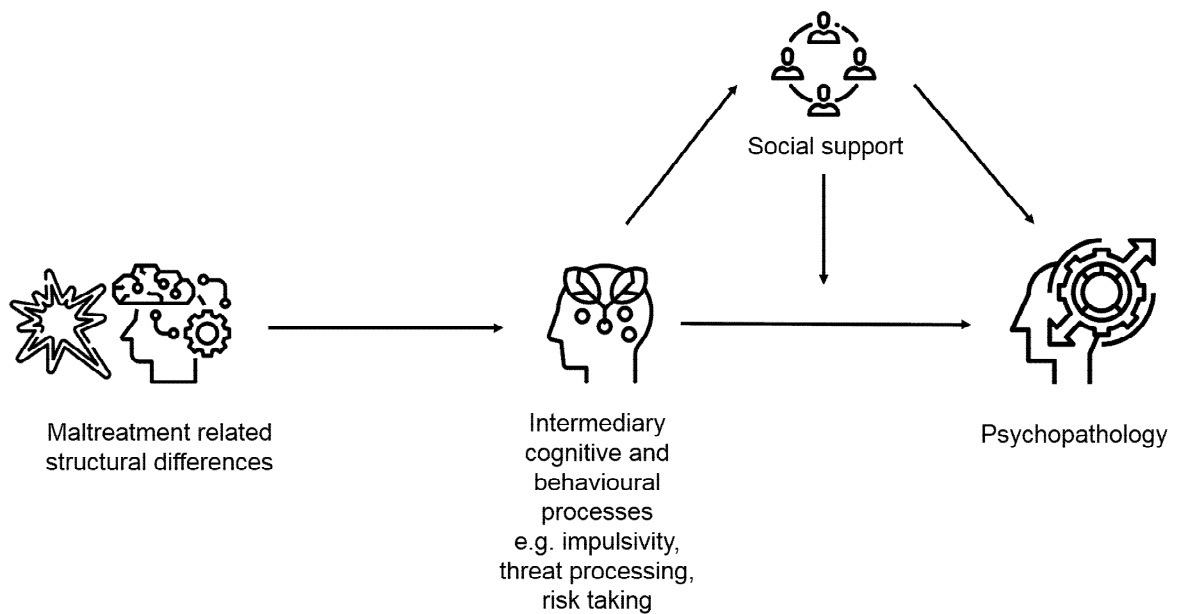
Three key considerations, based on the outcomes and process of investigation within this study, will be discussed. First, social support appears to be a poorly operationalised and measured construct. Researchers in the wider social support literature have highlighted the lack of consensus on how to operationalize social support (Vangelisti, 2009), the heterogeneity in which it is measured (Jaffee, 2017), the poor psychometric properties of many measures (Uchino, Cacioppo, & Kiecolt-Glaser, 1996) or do not have appropriate discriminant validity within the domains of the questionnaire (Cheng & Chan, 2004). One may even question the validity of self-report questionnaires in capturing the complex iterative and dynamic processes that occur within social interactions, and therefore adjunct behavioural measures which code the two-way nature of these interactions are needed to accurately represent 'social support'. A common framework is needed to accurately reflect social support,

and aid in the comparison between studies, and populations, and untangle the distinct influence of separable domains of social support.

Second, it is not clear how structural differences can be interpreted. There is not a simple relationship between static levels of cortical structure and behaviour, nor relative cross-sectional levels of structure between two different groups and observable behaviour and function. One could view decreases in cortical structure within a neurotoxicity hypothesis, such that prolonged cortisol exposure associated with stress atrophies susceptible regions of the brain (Gould & Tanapat, 1999), or that underlying functions are underdeveloped/used less, or equally decreases in structure may represent synaptic pruning towards neural efficiency due to greater recruitment of the region (Rypma et al., 2006). Lyoo and colleagues (2011) demonstrated a pattern of regional cortical differences in trauma-exposed individuals compared to controls, however these differences were subsequently found to correlate with greater posttraumatic stress disorder symptom reductions, and recovery. It is possible within the current study, that region structural differences represent protective adaptations to maltreatment exposure, rather than reflecting maladaptive calibrations that confer risk or reflect symptomatology. Given that the t-scores on the symptomatology scales did not indicate a general trend of elevated scores within the clinical range, one could assume that the maltreated sample were ostensibly 'well-functioning'. Furthermore, it is a potentially invalid presumption that the cortical structure of resilient individuals would resemble that of non-maltreated or 'healthy' individuals. Teicher and colleagues (2016) identified a substantive number of studies in which maltreatment-related atypical structure were 'by and large, independent of the presence or absence of psychopathology' (Teicher, Samson, Anderson, & Ohashi, 2016). It is more likely

that these individuals use compensatory processes, represented by cortical differences, rather than remain unaffected. This is supported by studies that show that maltreated individuals without psychopathology present with differences in how they regulate their emotions on an hour by hour basis, when compared to non-maltreated controls, even if there was no difference in average scores (Teicher, Ohashi, Lowen, Polcari, & Fitzmaurice, 2015). It is therefore important for subsequent multi-level investigations on the neurobiological impact of maltreatment to integrate resilience and coping factors as a potential mediator and outcome in delineating the longitudinal pathways from maltreatment.

Third, we may be asking the wrong questions regarding the relationship between maltreatment, social support and psychopathology. All three concepts are broad, diverse and often poorly defined in many research and clinical contexts; to propose that there are direct and linear associations between them may be misguided. Figure 13 visualizes an alternative theoretical model; as stated previously regional differences associated with maltreatment may better represent calibrations in underlying cognitive and affective processes. These calibrations may increase risk to psychopathology, but in themselves may not be symptomatic of common psychiatric disorders, as proposed by the theory of latent vulnerability (McCrory & Viding, 2015). In turn, social support may closely interact with these calibrations, in both a moderation and mediation role, in conferring risk to psychopathology, more so than cortical architecture.



**Figure 13.** Theoretical model of the trajectory from cortical structure to psychopathology

Existing research already points to potential candidate intermediary processes that are found to be atypical in maltreated populations and associated with the maintenance of many common psychiatric disorders, such as threat processing (McCrorry et al., 2011), emotional regulation (Kim & Cicchetti, 2010), impulsivity (Somers, Ginzburg, & Kramer, 2012), rumination (Raes & Hermans, 2008), and avoidance (Shenk, Putnam, Rausch, Peugh, & Noll, 2014). Future multi-level studies investigating the impact of maltreatment on neurodevelopmental pathways may therefore benefit from including intermediary cognitive, behavioural and affective processes, to better map the subtle relationships between different levels of functioning and provide clearer targets for preventative interventions.

These key considerations highlight the direction for future studies. Namely, the inclusion of intermediary cognitive, affective and behaviour processes that may better characterize multi-level developmental pathways from maltreatment; the

inclusion of longitudinal testing of brain structure alongside psychopathology and resilience/recovery measures that will allow researchers to capture cortical change that may more accurately represent the neurological underpinning of risk and resilience following maltreatment; and use of multidimensional constructs of social support that allow researchers to precisely delineate moderation and mediation roles (e.g. measurement of dyadic reciprocity, conversational turn-taking, gaze following, mentalization).

### Strengths and limitations

Several strengths of this study should be acknowledged. Use of stringent group matching and inclusion of appropriate covariates supported our proposal that the observed structural differences were associated with maltreatment experience, and not confounded by socio-demographic factors that have frequently been associated with cortical structure. Measurement of multiple subtypes of maltreatment and use of both file-report and self-report measures allowed a comprehensive characterisation of the maltreated sample, thus aiding in the comparison of these outcomes with prior studies. Undertaking a whole brain approach to the structural analyses with stringent correction for multiple comparisons meant that we were able to detect durable structure differences associated with maltreatment. This study also represents one of the largest longitudinal samples exploring the predictive nature of cortical structure on later symptomatology.

However, it is important to note several limitations of the current study. While this study is longitudinal in regard to symptomatology, the use of cross-sectional analysis of cortical structure limits the inferences we are able to make between the

importance of cortical regions on future symptomatology. Furthermore, a relatively short two-year period between measurement time points may limit the variance in symptomatology change and thus the detection of meaningful effects. Last the symptomatology clusters included in the study may appear limited, given that several other psychiatric disorders are associated with maltreatment exposure, such as borderline personality disorder (Ibrahim, Cosgrave, & Woolgar, 2018). While this may impede the generalizability of the results presented, reducing the number of implemented models and correlations provided some protection against Type 1 errors.

### Conclusions

In conclusion, this study sought to systematically investigate the relationship between regional differences in cortical structure (CT, IGI, and SA) associated with maltreatment and psychiatric symptomatology across two time points, and how this relationship may be moderated by social support. A pattern of significant relative decreases in IGI and an increase in CT was observed within the maltreated sample compared to non-maltreated peers, while gyral SA did not significantly differ between the two groups after correcting for multiple comparisons. The regional differences were found with a broad range of areas, associated with emotional regulation and facial processing. While these differences were apparent at a group-level, severity of the maltreatment experience did not exert a significant impact on the structure of these regions. Furthermore, these regional differences broadly were not associated with symptomatology at T1, and when they were, the association was not distinct within the two group. The predictive nature of the cortical differences was limited to IGI in the superior parietal region which exhibited a negative correlation with later symptomatology, but was of a small effect, and did not survive multiple comparisons

correction. Social support was found to moderate this relationship, but in the opposite direction to hypothesized, such that social support benefitted those who exhibited reduced impact of maltreatment at a neural level. Importantly, maltreatment severity was negatively correlated with social support frequency and importance, predominantly for peer support out of the home and in the classroom. Future longitudinal studies should incorporate cortical change, multi-dimensional measures of social support, and intermediary process to delineate the functional importance of the reported structural differences associated with maltreatment exposure.



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## **Integration, Impact and Dissemination**

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## Integration

The overarching objective of this thesis was to investigate the sequelae of childhood maltreatment and explore the factors that characterise the pathways towards poor mental health outcomes. The systematic review proposed to understand how one of these factors, social support, may characterise the probabilistic pathways to internalising disorders. In doing so, it provided a clear rationale for the empirical study, which explored pathways to psychiatric symptomatology but on a neurological scale, to include social support as resilience factor in the multi-level investigation. There were several central themes that ran through both the systematic review and the empirical study; resilience factors in maltreatment research, the role of social support, and a need for greater understanding of the underlying mechanisms and processes that characterise the pathways from maltreatment to symptomatology. As such, four unified inferences and implications have been made from this thesis, which will be explored below.

### *Neural underpinning of resilience following maltreatment*

The systematic review highlighted the expansive literature regarding social support as one of many resilience factors for individuals with an experience of maltreatment and emphasized the importance of social support in protecting maltreated individuals from poor outcomes. Importantly, the review indicated that the way social support interacts with maltreatment experience is complex, beyond its role as a direct protective factor. I suggested that further research mapping out the underlying mechanistic processes of this interaction that traverses multiple levels of functioning was crucial in future research. However, much of the existing neurobiological research, particularly within

structural neuroimaging, predominantly explores the relationship between neurobiology and risk or symptomatology, and neglects to investigate the neural correlates of resilience (Teicher et al., 2016). Within the empirical study it was postulated that the structural differences may equally represent effective and protective adaptations as they do maladaptive calibrations that represent risk. This was due to the limited associations between structure and symptomatology, and a finding that social support moderated the relationship between structure and function for those individuals with the smallest cortical structural differences associated with maltreatment. This is particularly pertinent given that the majority of maltreated individuals do not go on to develop psychiatric disorders (McGloin & Widom, 2001) and many structural differences associated with maltreatment are ‘largely independent’ of the presence of psychopathology (Teicher et al., 2016). The integration of these two chapters suggests that greater focus should be directed towards the neurological underpinning of adaptive coping and resilience factors in maltreated individuals. In doing so, researchers and clinicians will have tangible indicators of certain cognitive and emotional processes within resilient individuals that could guide effective interventions for those maltreated individuals who are at a greater risk for psychiatric difficulties.

#### *Lack of evidence for the buffering hypothesis*

Importantly, the systematic review highlighted that there was a lack of sufficient evidence for the role of social support as a moderator of the impact of maltreatment experience on the emergence and presence of internalising symptomatology. Notably, within the review there were studies which indicated that the directionality of the moderation was contradictory from that proposed within the ‘buffering hypothesis’

(Cohen & Wills, 1985), and that there was inconsistency how sex influenced the moderation effect. These findings were mirrored in the empirical study where high levels of social support was found to moderate the relationship between cortical structure and later symptomatology *only* for individuals with the smallest structural differences associated with maltreatment, counter to the proposed buffering hypothesis. Furthermore, maltreatment severity was negatively correlated with social support. Therefore, it is plausible that the mere presence of social support will not engender a protective factor as maltreatment severity may cause calibrations across levels of functioning that alter the way in which these individuals elicit and utilise social support. Taken together, this suggests the stress buffering hypothesis may not sufficiently capture the complex and subtle ways in which social support interacts with maltreatment experience and stimulates resilience against poor outcomes.

*Investigations into mediators and intermediary processes at a social and neural level*

The review largely supported the role of social support as a mediator in the relationship between maltreatment experience and internalising symptomatology. However, it was apparent that the exact ways in which maltreatment altered how social support protected individuals from the emergence of internalising symptomatology were not clear, nor which specific characteristics of social support engendered the greatest protective effect. The empirical study similarly highlighted, through 1) the findings of negative correlations between maltreatment severity and social support, 2) the relative lack of associations between the structural findings, symptomatology, and social support, 3) the ambiguity in whether the structural differences may represent protective adaptations or maladaptive calibrations, that we

may have not been asking the right questions. I suggested that a focus on the underlying intermediary processes that reflect the association between maltreatment severity and social support, and a functional understanding of the observed structural differences may provide a clearer picture to the developmental trajectories from maltreatment towards resilience *and* poor outcomes.

While there is extraordinary precision by which neuroimaging can capture variations in cortical structure associated with maltreatment, the mapping of structure with underlying function is less clear. Furthermore, the accuracy of neuroimaging techniques stands in stark comparison to the vague manner by which social support is captured. Researchers and clinicians should therefore focus on ecologically valid measure of social functioning, that may bring better accuracy to concepts of social support and may have a greater mapping to variations in cortical structure. There are several processes that would be attractive candidates. First, ‘epistemic trust’, the trust a person has in the authenticity and personal relevance of interpersonally transmitted knowledge, has been suggested to enable social learning (Fonagy, Luyten, Allison, & Campbell, 2017), and is proposed to atypically develop in the context of attachment difficulties, common within maltreated individuals. Second, ‘mentalizing’, the capacity to understand others’ and one’s own behaviour in terms of mental states, has been found to be recruited at a neural level during social interaction (Alkire, Levitas, Warnell, & Redcay, 2018), but how this may be effected by maltreatment is currently unclear. Last, emotional regulation, represent another ecologically valid measure of social interaction given that research has indicated that effective emotion regulation strategies may be similar to those associated with eliciting social support (d’Arbeloff et al., 2018).

### *Social support as a poorly operationalised construct*

Last, in both sections of the thesis it appeared that social support is a poorly conceptualised and operationalised construct. The variance in the findings of the synthesis of the role of social support as a moderator, the diversity of the measures used by the included studies, and the variance of social support as a protective factor in the empirical study, may highlight the poor convergent validity of social support measures. It has been noted previously that the psychometric properties of many measures of social support are weak (Uchino et al., 1996). This limitation further contributes to the proposal that the underlying mechanisms of social support, and other intermediary and resilience factors, should be delineated. Consequently, these investigations into these underlying mechanisms would feedback into a better common framework of social support as measurable construct. This would subsequently aid in the comparison between studies, and populations, and untangle the distinct influence of separable domains of social support (e.g. frequency, source of support).

### Reflections on methodology and the process of the thesis

#### *Service-user involvement*

Service-user involvement (SUI) within this thesis was limited. Primarily, this was a result of the use of secondary data used in the empirical study, thus inhibiting SUI in many steps of the conception and running of the study. It is important to note the ‘Young People in Care Islington Advisory Group’, a group of individuals who have been in care or have experienced maltreatment, were involved in the initial consultation for information sheets, consent forms, and the language used when

recruiting and testing (e.g. maltreatment and resilience). However, there are clear opportunities for the further involvement of service-users at this later stage, specifically in the dissemination of the outcomes. Due to the sensitive nature of the subject matter, it would be important to involve appropriate service-user groups in the precise use of language, translation of results into lay understanding, and advice on opportune forums for dissemination. Feedback from service-users and service-user groups following dissemination would be advantageous to provide qualitative information on how people may have experienced social support in the wake of maltreatment and across their lifespan. This feedback would also be especially important in the formulation of future studies, as it will help guide the research team to identify the most salient ‘intermediary process’ to investigate. Going forward in consequent research in this area, I can see clear opportunities for SUI in the co-creation and selection of questionnaires and in the undertaking of the research (e.g. involving previously recruited participants).

#### *Pre-existing data*

The use of secondary data allowed for greater scope to be taken within the empirical study. This community sample represents an inherently hard to recruit population who had taken over a year and a half to recruit and had undertaken multiple testing sessions (sometimes taking ~3hrs). Due to the time limitations of the current thesis, this would have not been possible alongside the extensive structural imaging pre-processing and analysis. On the other hand, the use of secondary data limited the capacity to which I was able to adapt the empirical study based on the outcomes of the systematic review (e.g. what social support scales were used), and restricted the analyses I was able to undertake based on the sample size that had been collected (e.g.

mediational analysis would have been underpowered). As such, the influence of the two chapters on each other was heavily skewed towards the empirical study, in some ways governing the direction of the systematic review. Nonetheless, I had been instrumental in the initial collection of the data, involved in the recruitment, testing, and design of the battery, and as such I was able to see the clinical and academic value in the data that had not been analysed, and therefore this has been a great aid in constructing the questions and hypotheses of this study.

## Impact

There are several key beneficiaries from the outcomes of the thesis, including services users, academic researchers, social workers, the general public, clinicians, governmental policy, and teachers. The impact of this research will be expanded on below for each of these beneficiaries.

### Service users

Individuals who have experienced childhood maltreatment should be primarily considered as one of the beneficiaries of this research. Namely this research underlines that the trajectories from childhood maltreatment are **not** deterministic, and there are a great number of factors that may serve to protect these individuals from poor outcomes, such as social support, as well as factors that may increase their risk of mental health difficulties in certain contexts. The way in which this is disseminated is vital to ensure that this research maximises the potential benefits, and attenuates stigma, or mistrust. Ways in which this is done is through use of appropriate language, lay explanations of brain development, and suitable dissemination routes, amongst others, which will be expanded upon in the dissemination section. Prior



research which have described cortical differences associated with maltreatment as ‘limbic scars’ (Dannowski et al., 2012), is a prime example of language that may serve as detrimental and stigmatising to service-users and individuals who have experienced maltreatment.

### Academic

There are several clear implications for academic researchers, that have been touched on in the two chapters. First, it demonstrates the value of investigations into fine-grained cortical indices that characterise more general and blunt measure grey matter volume, and represent distinct genetic influence and developmental trajectories (Panizzon et al., 2009; Schaer et al., 2008). The empirical study detailed distinguishable differences across cortical thickness and local gyrification, adding to the recently emerging literature indicating that maltreatment may impact on these indices in distinct ways (Busso et al., 2017; Kelly et al., 2016; Lim et al., 2017). Future studies should therefore make explicit efforts to incorporate multiple indices of cortical structure to delineate how these may translate to function and behaviour. Second, both the review and empirical study emphasized social support as a salient and appropriate resilience factor for maltreated individuals. However, the way maltreatment may interact with social support is currently unclear, with both chapters indicating that the exploration of intermediary processes between structure and symptomatology and ecologically valid measures of social interaction in maltreated individuals are vital steps in delineating the multifinality of maltreatment. Several candidate processes, such as vigilance to threat, impulsivity, and risk-taking, and measures of social interaction, including trust, mentalizing, and emotional regulation, were set out as prime candidates for future multi-level investigations across brain,

cognition, and behaviour. Last the longitudinal element of the empirical study highlights the strength of such design in understanding the emergence of mental health symptomatology following maltreatment, given that the only significant relationships between cortical structure and symptomatology were predictive from T1 to T2; however these were of small effect and did not survive multiple comparisons correction. Longitudinal designs provide a better grounding to infer about causal relationships between the variables we have set out but do come with greater barriers related to cost and time. Nonetheless maltreatment investigations that use such designs have recently attracted a large amount of funding, from organisations such as the NSPCC and ESRC who awarded £1.7million in 2016 to four distinct studies that use RCTs and cohort studies within maltreated populations.

### Social care

Social care professionals are key beneficiaries to this work. The findings demonstrating a negative correlation between maltreatment severity and social support have significance for social care assessments. For those who have experienced maltreatment, the social workers should consider in what ways does the child need support. The evidence indicated that social support is not simply a deficit to fill, but there is an active process that is going on between prior trauma and the way that these individuals may elicit and respond to social support. As such social workers should consider the subtle ways in which these individuals may respond to support and their relationship to different forms of socialised 'help'.

## General population

The impact of this research on the general population should also be considered. The interest in neuroimaging and ‘pictures of the brain’ in the press has been clearly noticeable (Racine, Bar-Ilan, & Illes, 2006). However, there appears to be an the limited public understanding of neuroscientific data (Herculano-Houzel, 2002) even though many have greater confidence and optimism in this biological data regardless of whether it reflects poorly designed studies with dubious outcomes (Beck, 2010; Weisberg, Keil, Goodstein, Rawson, & Gray, 2008). While the potential interest of the neuroscientific findings provides a clear opportunity for this research to reach a greater audience, highlighting the importance of maltreatment research, probabilistic trajectories and resilience factors, and an understanding of the plasticity of the brain to environmental factors during development, the dissemination of the findings requires an appropriate and proportional translation to lay understanding which is mindful of underlying biases.

## Clinicians

While the findings from both chapters illustrated the importance of social systems characterising the multifinality of childhood maltreatment, and consequently the value of holding systemic principles and approaches, there are several additional implications and impact that these findings have. First, childhood maltreatment has been associated with poorer psychological treatment response, greater numbers of drop-out and early termination from therapy before receiving any therapeutic benefits in child and adolescent populations (DeLorenzi, Daire, & Bloom, 2016; Lau & Weisz, 2003). The research supports the consideration of an individual’s relationship to help

(Reder & Fredman, 1996), especially in the context of childhood maltreatment, and highlights that these individuals may elicit and respond to available help in distinct ways. Clinicians thinking about the interacting beliefs about the treatment process particularly for individuals who have experienced maltreatment, and how they may respond differently to forms of socialised support may have a marked effect on the rates of engagement and treatment adherence.

Second, interventions that are focussed on strengthening and expanding an individual's relational network given our findings of maltreated individuals relatively less frequent social contact may be particularly salient. Inter-personal therapy (IPT), which help to develop skills in developing relationships within a person's network, and utilise relational skills to identify, express, and regulate one's own emotions and meet their interpersonal needs, is a evident target therapy which has been shown to have favourable outcomes for individuals with maltreatment histories in adult populations (Duberstein et al., 2018; Talbot et al., 2011). With recent developments in an adolescent analogue to IPT, there are clear implications for the use of this therapy in this high-risk population as a preventative intervention.

Last, the findings of the empirical study highlight the sensitivity and caution with how clinicians utilise and translate neuroimaging into clinical practice. It was shown that there are unlikely to be simple linear relationships between brain structure and symptomatology, and that we are unable to infer the functional importance of atypical structure associated with maltreatment. Therefore, these findings impact clinicians by motivating their questioning of the direct utility of neuroimaging to clinical practice and provide greater consideration to the pathways by which neuroimaging and clinical practice may be mapped. Moreover, it would not be valid

to give each child a brain scan to determine probabilistic cortical factors that suggest emergence of poor outcomes in the future given that the reported findings indicate average differences across a group, and that individual differences in brain structure are typically greater than group differences.

### Policy

A recent government green paper, a document that sets out discussions and proposals at a formative stage, on transforming child and young people's mental health provision (*Transforming Children and Young People's Mental Health Provision: a Green Paper*, 2017) provides a considerable amount of examination to the link between adverse childhood maltreatment and mental health difficulties. These included setting out research priorities that included establishing the mechanisms through which adverse childhood experiences may impact negative outcomes in later life and determining the relative risk of abuse severity. The current research has provided evidence for why the mechanisms underlying the multifinality of maltreatment are needed in multi-level investigations and what mechanisms may represent opportune candidates for future investigations (e.g. ecologically valid measures of social support and interaction), and has provided initial evidence of the relative risks of maltreatment severity within the home on the frequency of peer social support out of the home, and within the classroom. As such the research provides further evidence within a domain that has been shown to have an impact on the government's plans for the progression and transformation of children and young people's service and is likely to support further improvements within these services based on statements of policy.

## Schools

Last, given the findings that in-home maltreatment impacts social support with peers out-of-home and within classrooms, there are implications for teaching professionals and schools as key environments for educational, social and emotional development. Studies have detailed a link between childhood maltreatment and poor educational outcomes and concomitant mental health difficulties (Gilbert et al., 2009; Romano, Babchishin, Marquis, & Fréchette, 2015). There have been calls for coordinated plans that address these mental health and educational difficulties for individuals who have experienced maltreatment in a school environment (Romano et al., 2015), and social support may represent a clear factor to consider given that maltreatment may impact the ways these individuals elicit and respond social support most out of the home and within schools with peers. Some existing educational frameworks specifically designed for maltreated individuals neglect the inclusion or consideration of social resources (Cole et al., 2005), whereas others include social skills training and explicit support for the development of classroom friendships (Lowenthal, 2001). This research clearly advocates for teaching interventions to consider how maltreatment, and the severity of the adverse experience, may interact with peer social support, in ways that it is simply not a ‘deficit to fill’.

## **Dissemination**

The two chapters of the thesis will be prepared for submission to high impact academic journals. The journals, *Journal of Child Psychology and Psychiatry* (impact factor – 6.62), *Social Cognitive and Affective Neuroscience* (impact factor – 3.50), and *Neuroimage Clinical* (impact factor – 2.16) have been selected as appropriate

journals for submission of the empirical study. These journals have published several multi-level investigations that incorporate neuroimaging with clinical markers of mental health outcomes and have shown prior interest in publishing studies within maltreated populations. The systematic review will be prepared for submission to *Child Abuse and Neglect* (impact factor – 2.899) or *Development and Psychopathology* (impact factor – 4.357), due to the explicit focus of these journals in adverse childhood experiences, and the longer form to the articles that they regularly publish.

Further dissemination of the two chapters to academic community will be through spoken and poster presentations at upcoming conferences. International conferences identified include the International Society for the Prevention of Child Abuse and Neglect (ISPACN) in Oman (September 15-17), and the Society for the Research in Child Development to be confirmed for 2020 (2019 conference has passed). National conferences identified are the yearly MQ Mental Health Science meetings (held in February), and The BPS cognitive psychology section and developmental psychology section joint conference 2019 in Stoke on Trent (September 4th-6th). These conferences provided a range of clinical and academic focussed presentations and attendees, thus will be well placed to maximise the impact of the research,

Dissemination to groups outside of the research community will also be undertaken. Service-user involvement (SUI) at this stage will be crucial for several reasons. First, the language used to describe the sample, methods, and findings is essential to limit stigma associated with the experience of maltreatment and mental health difficulties. Second, SUI will help to establish which findings may elicit

greatest interest and to help construct an easily understandable lay description of the research and its outcomes. There should be focus directed to the tentative nature of the findings, and consideration of brain plasticity; that these cortical differences are not deterministic and structure changes across the lifespan in response to the environment and other factors. Last, SUI will be vital in defining the dissemination pathways so that the research is able to reach individuals who have experienced maltreatment. As mentioned previously the 'Young People in Care Islington Advisory Group' is one organisation that have previously worked in the initial stages of the overarching longitudinal project and are a clear candidate organisation to approach again.

It would also be beneficial disseminate the findings to social care teams, who will have regular contact with maltreated individuals, and undertake social care assessments. The inner-London social care teams involved in the recruitment of the participants represent an initial candidate to present the findings to, following interpretation and translation through SUI. Furthermore, the schools who were vital in the recruitment of the control participants also serve as a channel to maximise the impact of this research, given the previously discussed impact of at-home maltreatment on in-school peer social support.

Dissemination to other clinical disciplines will allow us to maximise the impact of the research within settings in which psychologists will work alongside other healthcare professionals, such as within hospitals and CMHTs. Presentations in health settings, such as within my current placement at the Paediatric Liaison Team at the Royal London Hospital and attendance and presentation at psychiatry conferences, such as the Royal College of Psychiatrists Faculty of Child and Adolescent Psychiatry



Annual Conference 2019 (26th September 2019) represent two avenues for interdisciplinary dissemination.

Dissemination to the general public will also be important, and the use of social media has been reported to be a key way in engaging with diverse audiences and increasing public visibility both within public and academic communities (Harrison, Hayes, Woollard, & Tracy, 2019), with 'retweets' within the first few days of publication correlating with how highly cited an article is (Eysenbach, 2011). Two avenues of social media dissemination will be through personal and collaborator's twitter and approaching mental health blogs (e.g. MentalElf) for summary articles. Furthermore, the feedback and public discussions through social media outlets, will be beneficial in influencing future studies. Liaison with the press communications team at Royal Holloway would be an additional route to pursue to review draft press releases, determine which outlets to approach, and to release through their own university channels (e.g. email newsletters, websites).

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**Note:** *Articles included in the systematic review are denoted by an asterisk*

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# Appendix

## Appendix 1. Idiosyncratic quality assessment tool

### **Quality assessment tool**

- Adapted from the Mixed Methods Appraisal Tool (MMAT) and Quality Assessment Tool for Quantitative Studies (QATQS)
- Responses: yes/no

### **Sampling strategy**

#### **1. Is the sampling strategy relevant to address the research question?**

Criteria

1. *Is the sample relevant to investigate social support and internalizing symptomatology in individuals who have experienced childhood maltreatment?*
  - o *Have the authors sampled everyone from within a population (e.g. community sample).*
  - o *If the authors have used an opportunistic sample, have group matched between a maltreated sample and non-maltreated samples?*
  - o *If the authors without a 'control group' have recruited through an opportunistic or clinical sample (e.g. through a clinic for abuse survivors), are all participants recruited from the same population, and are maltreatment characteristics of the sample reported?*
2. *Have the authors reported sufficient inclusion/exclusion criteria relevant to maltreatment experience, sampling procedures, using demographics, location, and time period?*

*Yes = Both criteria.*

*No = Only one or none of the criteria*

### **Data measurement**

#### **2. Are measurements appropriate to characterise internalising symptomatology (clear origin, or validity known, or standard instrument)?**

Criteria:

1. *Authors have used a standardised or well validated and reliable questionnaire measure ( $\geq 0.7$  on reliability or internal consistency) or diagnostic interview to determine symptomatology within an internalising disorder (e.g. anxiety, depression, PTSD, panic, OCD).*
2. *A composite measure of internalizing symptomatology is used but clear and comprehensive contributory symptomatology is detailed, including internal consistency statistics.*

*Yes = Either of the above criteria*

*No = Neither of the above criteria. Idiosyncratic or measure, without prior use in studies or has no reliability or validity statistics.*

#### **3. Are measurements appropriate to operationalise maltreatment within the study (validity known, or standard instrument)?**

Criteria:

1. *Presence of maltreatment is appropriately captured via substantiated file-report, to provide a dichotomous variable of presence of child maltreatment (experienced/not experienced).*

2. *The experience of maltreatment is captured with a standardised or well validated and reliable questionnaire measure ( $\Rightarrow 0.7$  on reliability or internal consistency), to provide a measure of presence or severity of maltreatment.*

*Yes= One of the above two criteria met.*

*No= Measure has not been used in previous studies or can report sufficient ( $\Rightarrow .70$ ) reliability or validity statistics.*

**4. Are measurements appropriate to characterise social support (clear origin, or validity known, or standard instrument)?**

*Criteria:*

1. *Authors have used a standardised or well validated and reliable questionnaire measure ( $\Rightarrow 0.7$  on reliability or internal consistency) to capture one or many dimensions of social support (social network, perceived social support, enacted social support).*

*Yes= Above criteria met*

*No= Idiosyncratic scale used, or scale that has been used in previous studies but does not report reliability and validity statistics.*

**Confounders**

**5. Did the study control for potentially confounding variables?**

*Criteria:*

1. *Authors have included potential confounders/covariates within the analytic model (e.g. Ethnicity, sex, age, cognitive ability, other psychiatric comorbidities other than the variable of interest).*
2. *If covariates or potential confounders were not included in the analytic model, was appropriate justification provided?*

*Yes= One of the above criteria*

*No= Potential confounders or covariates are not identified, or their absence is not justified.*

**Analyses**

**6. Was the process for analysing the quantitative data relevant to address the research question?**

*Criteria:*

1. *Were the appropriate statistical analyses used to address the research question?*
2. *Did the authors clearly state the analytic protocol, adequate statistical output, whether assumptions were met/ adjustments to data (e.g transformations) or significance values made (e.g. multiple comparisons correction)?*

*Yes= Both above criteria met*

*No= Inappropriate statistical procedure used to address question, protocol is unclear, absence of or inappropriate adjustments to data made*

## INFORMATION SHEET FOR CHILDREN

### What is the study about and what is its goal?

- ☺ We are interested in how our brains deal with emotions and what helps children succeed.
- ☺ How the brain reacts to faces showing different emotions can help us understand how people behave in real life. We want to find out how people cope best with different things that happened to us in the past, both good and bad.
- ☺ If we know about how the brain deals with challenges we may be able to help more children do well and succeed even if they have had difficult experiences in the past.



### What does the study involve?

- ☺ You would come to UCL (a University) and meet with Amy & Philip for around 2 and a half hours. We will ask you some questions about faces showing emotions and give you some simple word and shape puzzles to do. We will also ask you some simple questions about your life at home (where you are living now).
- ☺ We would also like to take a saliva sample of your DNA, which is very quick and easy. It is important for you to know that no one else apart from the researchers in this study will have access to your DNA.
- ☺ We will invite you back in a couple of years to see how your brain has changed. We will also keep in touch with you during this time.
- ☺ We will also be asking your parent / carer and social worker (if you have one) to complete some questionnaires for us.



### How do we study what happens in the brain?

- ☺ We would like to take a picture of your brain with a special camera while you are playing some games.
- ☺ The games you will be playing are not very hard and should be fun. We might ask you to make decisions about different faces or cartoons.
- ☺ The camera to take the picture of your brain looks like a huge donut. The inside of the donut is where the camera is - it will never touch you and you won't feel anything, but it does make a few funny sounds when it is taking the picture. You can wear headphones to protect your ears from the sounds.



- ☺ If you want, your parent or carer can stay with you while we take the picture of your brain. You can also practice lying in the scanner and have a 'mock scan' if you would like to try it out first.

### Can I have a picture of my brain?

- ☺ Yes, if you decide to take part in the study, we will give you a CD with pictures of your brain to take home and a t-shirt.



### What will we do with our findings?

- ☺ The picture of your brain, information about your behaviour and any information we collect about your home or school is confidential. Your private information will never be reported on its own.

### Do I have to take part?

- ☺ No, it is up to you. Have a chat with your parent or carer about taking part in this study, as well as other grown-ups you trust. You can also call us if you have any questions.
- ☺ If you decide that you would like to take part, please sign the consent form that is provided.
- ☺ If you do not want to take part, let your parents or carers know and they will tell us. Even if you decide to take part, you can stop at any time. (Please remember to say if you are afraid of small spaces as this might mean that it is best for you not to take part in the first place).



*Thank you for taking the time to read this information sheet.  
You and your family's help make our research possible.*





## Child Consent Form

Please tick (✓) the box:

**YES**, I would like to participate in this study

I agree for a DNA saliva sample to be taken (it is okay to say no and still take part):

**Yes**

**No**

**NO**, I do not want to participate in this study

If you are **NOT SURE** you can speak to your parent, carer or Amy Palmer on 02076 795367 or by email on amy.palmer@ucl.ac.uk

**If Yes, please tick (✓) the following:**

- I have read the Information Sheet or someone has read it to me.
- I understand that I do not have to take part in this study if I do not want to
- I understand I can leave from a session at any time without giving a reason
- I have had the opportunity to ask any questions I wish to ask.
- I have the names and telephone numbers of the research team in case I have any queries in the future.

My School: \_\_\_\_\_

My Name: \_\_\_\_\_

My Signature: \_\_\_\_\_ Date: \_\_\_\_\_

**Investigator's Statement (to be completed by the research team).**

I .....

confirm that I have carefully explained the purpose of the study to the participant and outlined any reasonably foreseeable risks or benefits (where applicable).

Signed:

Date:

***Thank you***

### Appendix 3. Parents information sheet and consent form



Amy Palmer, PhD  
Department of Psychology  
University College London  
26 Bedford Way, London  
WC1H 0AP  
Email: amy.palmer@ucl.ac.uk  
Tel: 02076 795367

## Information Sheet for Parents & Carers

Dear Parent / Guardian,

**We would like to invite you to take part in an exciting new research project that is being carried out at University College London.** We are a team of researchers interested in child development and in particular childhood resilience – essentially the ability to bounce back from difficult experiences.

The main focus of our work is to better understand how early experiences might influence the way that children develop coping skills. We wish to invite your child to be part of our control group of children about whom there have been no concerns. We will look at the influence of positive factors during development (e.g. having a good set of peer friendships) and the influence of negative factors (e.g. being treated poorly at times by adults). Our goal is to understand how children about whom there have been serious professional concerns show brain changes that help them manage adversity in a positive way. If we can understand what contributes to this kind of resilience we will be better placed to help other children who may be experiencing adversity.

### Do I have to take part?

No. You should only participate if you wish to; while we would encourage you to participate, choosing not to will not disadvantage you or your child in any way. Before you decide, it is important you read the following information carefully and discuss it with others if you wish. Please feel free to contact us if there is anything that is not clear or you would like more information. Even if you decide to take part, you can withdraw your child at any time, without giving a reason.

### What does the study involve for me?

We are especially interested in how the brain develops over time, and so in this study we are inviting families to take part in scanning on two separate visits. The second visit will take place two years after the first visit. You will receive similar levels of reimbursement for your time and travel for each of the two visits. We are recruiting 100 children aged between 10 and 18 years old. If you agree to participate, for each visit we will organize a time for you to visit us with your child - your visit should take around 2.5 hours in total. For each visit, in the first part of the session we ask you to complete some questionnaires, for example, about your child's behaviour, experience and personality. We also ask some questions about any difficult or upsetting events that may have happened in your family. This kind of information is important as it can help us to better understand children's resilience and coping strategies. Any information you give during this session should be entirely voluntary and will remain strictly confidential unless required by law (e.g. if your child is at current risk of harm).

### What does the study involve for my child?

The study involves your child having a scan using a brain scanner. Scanning involves your child doing a few short tasks such as looking at facial expressions that help us understand their emotional processing. Please see the attached FAQ sheet for further details. Your child will also complete a set of tasks, puzzles and questionnaires that look at their strengths and abilities, behavior, and negative experiences if they have been treated badly by adults. We would also ask permission for your child's teacher to complete a short rating scale questionnaire about their behaviour at school. Having a DNA swab taken is an optional part of the study.

### What about collecting DNA?

Sometimes differences in genes make some children more reactive or resilient to negative experiences than others. We also know that negative experiences can switch genes 'on' or 'off'. We are keen to collect DNA to study the influence of genes and this would help us to better understand how negative experiences have their effect. DNA would be collected for research purposes only and no one apart from our team would have access to this information. DNA can be collected quickly and non-invasively with a saliva sample. If you want to take part in the study, but would not like your child to provide DNA, that is fine.

**Will arranging a visit be difficult?**

We hope not. If you wish to participate we will call you and do our best to make your trip to UCL (UCL is near both Euston train and underground stations and Russell Square underground station) as straightforward and convenient as possible. For example, we will reimburse your travel costs, and talk you through directions. If you feel uncomfortable to travel within London, we are happy to send staff to meet you at the station. We will also contribute to the costs of lunch and snacks on the day for you and your family. We want to make taking part an enjoyable experience – and will give your child (if they would like it) a CD with pictures of their brain to take away (and show off at school!) and a t-shirt with a picture of a brain on it. You will also receive an honorarium of £20 for your time and effort in completing these questionnaires. Generally the visit will last about 2.5 hours.

**What happens during the brain scan exactly?**

We use magnetic resonance imaging (MRI) brain scans to measure brain activity. Children are given a few tasks that assess decision-making, memory and how the brain pays attention to emotion. The tasks are designed for kids and involve (for example) making decisions about faces and pictures. These tasks last approximately 10 minutes each. We may also provide a short DVD to keep your child occupied for 10 minutes while we take a picture of their brain structure. In order to ensure that the scanning experience will not be too tiring for your child we have a minimum 15 minute break in the middle of the scanning session and are happy for your child to have a longer break if needed. Your child will be offered the opportunity to have a ‘mock scan’ to practice what the scan is like – for example, see how much space there is and hear how noisy it is. *You are also welcome to stay in the scanner room with your child if you wish.*

**Is there a reason my child may not be able to take part?**

Yes. Because the scanner uses a magnetic field, children who have *any metallic implants or braces cannot participate*. *If your child is on prescription medication, or is afraid of small, enclosed spaces or loud noises*, MRI is unlikely to be suitable. Please discuss any concerns you might have before participating.

**Are there any side effects involved with MRI scanning?**

MRI has been in use in medicine for about 20 years, and has shown to be safe. It does not involve any radiation. People who have metal in their bodies (e.g. a pace maker) should not be scanned. We enclose a safety questionnaire with this information sheet so that we can be sure that it is completely safe for your child to be scanned. Please review this questionnaire, and tell us if your child has any of the conditions, which are listed. We will be happy to discuss this with you. The people who operate the scanner will also check that your child is safe to be scanned when you come to see us.

**What are the possible disadvantages & risks of taking part?**

There are none. However, because we will be taking pictures of your child’s brain there is a remote possibility we will have unexpected findings. If this happens, our neuroradiologist (who specializes in looking at pictures of the brain) will write to your GP in the first instance. The GP will then contact you if further assessments are required.

**What are the possible benefits of taking part in this research?**

There are no immediate benefits. We do hope that taking part will stimulate your child’s interest about how the brain works. Also, in the longer term you will help research that has potential to help other children in the future

**Will the information be kept confidential?**

Yes. All information that we collected is kept very private and secure. We remove your name and address so that you or your children cannot be recognized from it. All data will be collected and stored in accordance with the Data Protection Act. We are by law required to notify relevant agencies if your child is at risk in any way. When we report the study findings we do not identify individuals – instead our results report how participants do on average and do not refer to any personal information.

**What should I do now?**

Please contact us if there is anything that is not clear or you would like more information. You can call Amy Palmer on 02076 795367 or email: amy.palmer@ucl.ac.uk we would be very happy to help. If wish to participate please complete the consent form and the safety questionnaire provided and return in the stamped addressed envelope. We will then be in touch.

**We very much appreciate your time in considering whether to participate in this study.**

## Magnetic Resonance Imaging (MRI): Frequently Asked Questions

1. **Is an MRI or functional MRI (fMRI) safe? Are there any risks because of my child's age and developing brain?**

There is very little risk and discomfort when safety guidelines are closely followed. MRI has been performed on children of all ages for over 20 years without ill effect or discomfort (fMRI for 10 years). We have scanned over 150 children aged between 10 and 13, with great success. Instead of using radiation to form an image (as with X-rays), MRI uses magnetic fields and radio waves. Both magnetic field and radio wave exposure is safe for most people, unless your child has metallic implants or devices. This is because the MRI machine generates a very powerful magnetic field, which may draw metallic items or implants towards it or cause medical devices to malfunction. If your child has any metallic implants or medical devices, he/she will not be allowed to participate. Some people are claustrophobic (i.e. afraid of small spaces) which makes MRI an unpleasant experience for them. Claustrophobia is more likely to occur with adults than children. This is because adults are bigger and they have less space inside the MRI machine.

2. **I'm not sure my child will be able to sit still or tolerate being in a confined space. How do you handle this?**

Your child will undergo systematic familiarization with the MRI environment to decrease anxiety about being in the scanner. This will involve exposing your child to the noises, space, tasks, and sights of the MRI environment. If your child does not feel comfortable during the training, then scanning will not be attempted. If your child is uncomfortable during scanning, then scanning will be discontinued immediately. You can sit with your child throughout the scan if you wish.

3. **If my child moves while he is being scanned, will he be injured?**

If your child does not stay still there is no risk of injury, but the images we take will be blurred. If your child has a tantrum, there is no more danger of the child becoming injured than if he/she had a tantrum in a room with furniture.

4. **If you find something concerning on the scan, will you let me know? Can I pass on the images from the scan to my child's pediatrician/neurologist?**

The neurologist liaising with our study will review all the images we collect and will let your child's physician know if we notice any abnormalities. However, because the scans are being done for research, they are not optimized to detect abnormalities.

5. **Can I stay in the room with my child while the scan is being done?**

Yes. We encourage you to stay in the room with your child if it will help reduce anxiety. However, you, like your child, will have to wear hearing protection and remove anything metal from your body or clothing.

6. **Will a doctor be there if anything goes wrong? Or, will one be available to answer questions and address concerns I have?**

There may not be a doctor present during the scan, but there will always be MRI-trained personnel on site. These personnel have received extensive training on the MRI machine and MRI safety, as well as training for emergency medical intervention. There is immediate access to University College Hospital in the unlikely event that anything goes wrong. The MRI operators can answer any questions about the MRI. If you would rather speak with a physician, we can arrange for you to discuss with one liaised with our study.

## Parent Consent Form

Please tick appropriate box:

**Yes**, I would like my child to participate in this study and wish to know more.

I give consent for a DNA saliva sample to be taken (this is entirely optional):

- Yes**  
 **No**

**No**, I do not want my child to participate in this study.

If you are **NOT SURE** you can contact Amy Palmer on 02076 795367 or by email on amy.palmer@ucl.ac.uk

---

If Yes, please complete the following:

- I have read the Information Sheet.  
 I understand that I may withdraw my children from the study at any time without giving a reason.  
 I understand that my children may withdraw from the study at any time without giving a reason.  
 I understand I will be contacted directly by the research team in the future.  
 I have had the opportunity to ask any questions I wish to ask.  
 I have the names and telephone numbers of the research team in case I have any queries in the future.

---

Parent's Name: \_\_\_\_\_ Signature: \_\_\_\_\_

Phone Number: \_\_\_\_\_ E-mail Address: \_\_\_\_\_

Child's Name & Date of Birth: \_\_\_\_\_

Child's School: \_\_\_\_\_

**Investigator's Statement (to be completed by the research team)**

I .....

confirm that I have carefully explained the purpose of the study to the participant and outlined any reasonably foreseeable risks or benefits (where applicable).

Signed:

Date:

**Please return this form in the SAE provided.**

***Thank you***

## Child Maltreatment ratings

---

### PHYSICAL ABUSE

---

**Severity** (Please check box)

- |                                                               |                          |     |
|---------------------------------------------------------------|--------------------------|-----|
| No evidence of physical abuse.....                            | <input type="checkbox"/> | (0) |
| <b>If no evidence</b> , is there suspicion of physical abuse? | <input type="checkbox"/> | (0) |
| Evidence of physical abuse.....                               | <input type="checkbox"/> | (1) |
| Evidence of bruises from physical abuse .....                 | <input type="checkbox"/> | (2) |
| Injuries requiring medical care.....                          | <input type="checkbox"/> | (3) |
| Severe Injuries requiring hospitalisation .....               | <input type="checkbox"/> | (4) |

**Frequency**

- Age of onset (months/years):..... Duration of abuse (months/years):.....
- Abuse occurred throughout child's life: **Y / N**
- Abuse isolated incident: **Y / N**

### PHYSICAL ABUSE CONTINUED

---

**Frequency**

- Injuries inflicted more than 5 years ago: **Y / N**
- Is there current experience of abuse? **Y / N**

**Perpetrator**

- Who was the perpetrator? .....
- Multiple perpetrators? **Y / N**
- Child has not live with the perpetrator for more than 5 years? **Y / N**

### NEGLECT

---

**Instances of neglect** (please check box)

- |                                                              |                          |  |
|--------------------------------------------------------------|--------------------------|--|
| No evidence of neglect.....                                  | <input type="checkbox"/> |  |
| <b>If no evidence</b> , is there suspicion of neglect?       | <input type="checkbox"/> |  |
| <b>PLEASE TICK ALL THOSE THAT APPLY</b>                      |                          |  |
| Occasionally not fed a meal.....                             | <input type="checkbox"/> |  |
| Not fed in over 24 hours.....                                | <input type="checkbox"/> |  |
| Left without proper baby sitter.....                         | <input type="checkbox"/> |  |
| Poor or unsuitable supervision.....                          | <input type="checkbox"/> |  |
| Not provided routine medical care.....                       | <input type="checkbox"/> |  |
| Obvious medical problem not attended to.....                 | <input type="checkbox"/> |  |
| Unnecessarily left out of school .....                       | <input type="checkbox"/> |  |
| Unresponsive to child's basic emotional needs.....           | <input type="checkbox"/> |  |
| Failure to protect child from physical or emotional harm.... | <input type="checkbox"/> |  |
| Failing to provide adequate food, clothing or shelter.....   | <input type="checkbox"/> |  |

**Frequency**

- Age of onset (months/years):..... Duration of abuse (months/years):.....
- Abuse occurred throughout child's life: **Y / N**
- Abuse isolated incident: **Y / N**
- Abuse inflicted more than 5 years ago: **Y / N**

Is there current experience of abuse? Y / N

**Perpetrator**

Who was the perpetrator? .....

Multiple perpetrators? Y / N

Child has not live with the perpetrator for more than 5 years? Y / N

### SEXUAL ABUSE

**Severity** (please check box)

- No evidence of sexual abuse.....  (0)
- Serious suspicion of sexual abuse, but child denies it and there is no physical evidence  (1)
- Non-genital fondling or Genital fondling in which child's body is not violated.....  (2)
- Genital fondling in which the child's body is violated.....  (3)
- Vaginal or anal intercourse/penetration.....  (4)

Has the child been exposed to sexually explicit material? Y / N

Are there poor sexual boundaries at home? Y / N

**Frequency**

Age of onset (months/years):..... Duration of abuse (months/years):.....

Abuse occurred throughout child's life: Y / N

Abuse isolated incident: Y / N

Abuse inflicted more than 5 years ago: Y / N

Is there current experience of abuse? Y / N

**Perpetrators**

Who was the perpetrator?.....

Was the perpetrator related to the child? Y / N

Multiple perpetrators? Y / N

Perpetrator was a stranger? Y / N

Child has not lived with the perpetrator for more than 5 years? Y / N

### EMOTIONAL MALTREATMENT

**Severity** (please check box)

- No evidence of emotional maltreatment**.....  (0)
- Some experience of emotional maltreatment (*e.g. parental substance/alcohol abuse/mental health problems **without** evidence of emotional unavailability and/or rejection*).....  (1)
- Moderate experience of emotional maltreatment (*e.g. parental substance/alcohol abuse/mental health problems **with** evidence of mild emotional unavailability and/or rejection*).....  (2)
- Extreme parental rejection **without** evidence of parental substance/alcohol abuse/ mental health problems.....  (3)
- Extreme parental rejection **with** evidence of parental substance/alcohol abuse/ mental health problems.....  (4)
- Abandonment.....  (5)

\*Examples of rejection include being ridiculed, called unworthy of love, parent threatens to send away child

**Frequency**

Age of onset (months/years):..... Duration of abuse (months/years):.....

Abuse occurred throughout child's life: **Y / N**

Abuse isolated incident: **Y / N**

Abuse inflicted more than 5 years ago: **Y / N**

Is there current experience of abuse? **Y / N**

---

**EMOTIONAL MALTREATMENT CONTINUED**

---

**Perpetrators**

Who were the perpetrators?.....

Drug/alcohol problems occurred more than 5 years ago? **Y / N**

Drug/alcohol problem occurred throughout child's life? **Y / N**

---

**DOMESTIC VIOLENCE**

---

**Severity of witnessed domestic violence** (please circle)

- |                                                                  |                          |     |
|------------------------------------------------------------------|--------------------------|-----|
| No evidence of domestic violence.....                            | <input type="checkbox"/> | (0) |
| <b>If no evidence</b> , is there suspicion of domestic violence? | <input type="checkbox"/> | (0) |
| Evidence of domestic violence.....                               | <input type="checkbox"/> | (1) |
| Evidence of bruises from domestic violence.....                  | <input type="checkbox"/> | (2) |
| Seeking medical care due to domestic violence.....               | <input type="checkbox"/> | (3) |
| Requiring hospitalisation due to domestic violence               | <input type="checkbox"/> | (4) |

Age of onset (months/years):..... Duration of violence (months/years):.....

Does the parent still live with the abusive partner? **Y / N**

Domestic violence occurred more than 5 years ago? **Y / N**

Parental domestic violence occurred throughout child's life? **Y / N**



*Appendix 5. Childhood Trauma Questionnaire*

(Not included due to copyright restrictions).

Appendix 6. Puberty Development Scale

**Body Development**

For each of the following questions, please tick ( ✓ ) the answer that best describes your daughter's physical development. If you feel you don't know enough to answer a particular question, then just select "I don't know". We will keep all answers Private.

Would you say that your daughter's **growth spurt** has started yet? (A growth spurt is defined as growth in height that is faster than usual.)

- No.
- Yes, barely.
- Yes, definitely.
- Her development seems completed.
- I don't know.

Have you noticed the growth of your daughter's **body hair**? Would you say that growth of her underarm and pubic hair has started yet?

- No.
- Yes, barely.
- Yes, definitely.
- Her development seems completed.
- I don't know.

Have you noticed any changes in your daughter's **skin**, especially **pimples**?

- No.
- Yes, barely.
- Yes, definitely.
- Her development seems completed.
- I don't know.

Have you noticed that her **breasts have begun to grow**?

- No.
- Yes, barely.
- Yes, definitely.
- Her development seems completed.
- I don't know.

Has she begun to **menstruate** (had her first period)?

- No
- Yes      **If YES, how old was she when she had her first period?**  
Years: \_\_\_\_\_ Months: \_\_\_\_\_       Don't know



**Thank you for filling out this questionnaire!**

*Appendix 7. Trauma Symptom Checklist for Children*

(Not included due to copyright restrictions).

Appendix 8. Strengths and Difficulties Questionnaire

**Strengths and Difficulties Questionnaire**

**P 4-16**

For each item, please mark the box for Not True, Somewhat True or Certainly True. It would help us if you answered all items as best you can even if you are not absolutely certain or the item seems daft! Please give your answers on the basis of the child's behaviour over the last six months.

Child's Name .....

Male/Female

Date of Birth.....

	Not True	Somewhat True	Certainly True
Considerate of other people's feelings	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Restless, overactive, cannot stay still for long	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often complains of headaches, stomach-aches or sickness	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Shares readily with other children (treats, toys, pencils etc.)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often has temper tantrums or hot tempers	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Rather solitary, tends to play alone	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Generally obedient, usually does what adults request	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Many worries, often seems worried	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Helpful if someone is hurt, upset or feeling ill	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Constantly fidgeting or squirming	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Has at least one good friend	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often fights with other children or bullies them	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often unhappy, down-hearted or tearful	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Generally liked by other children	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Easily distracted, concentration wanders	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Nervous or clingy in new situations, easily loses confidence	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Kind to younger children	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often lies or cheats	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Picked on or bullied by other children	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Often volunteers to help others (parents, teachers, other children)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Thinks things out before acting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Steals from home, school or elsewhere	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Gets on better with adults than with other children	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Many fears, easily scared	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sees tasks through to the end, good attention span	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Do you have any other comments or concerns?

**Please turn over - there are a few more questions on the other side**

Overall, do you think that your child has difficulties in one or more of the following areas: emotions, concentration, behaviour or being able to get on with other people?

No	Yes- minor difficulties	Yes- definite difficulties	Yes- severe difficulties
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

If you have answered "Yes", please answer the following questions about these difficulties:

- How long have these difficulties been present?

Less than a month	1-5 months	6-12 months	Over a year
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

- Do the difficulties upset or distress your child?

Not at all	Only a little	Quite a lot	A great deal
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

- Do the difficulties interfere with your child's everyday life in the following areas?

	Not at all	Only a little	Quite a lot	A great deal
HOME LIFE	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
FRIENDSHIPS	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
CLASSROOM LEARNING	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
LEISURE ACTIVITIES	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

- Do the difficulties put a burden on you or the family as a whole?

Not at all	Only a little	Quite a lot	A great deal
<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Signature ..... Date .....

Mother/Father/Other (please specify:)

**Thank you very much for your help**

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*Appendix 9. Child and Adolescent Social Support Scale*

(Not included due to copyright restrictions).

Appendix 10. Royal Holloway Ethics self-certification certificate



Ethics Review Details

You have chosen to self certify your project.	
Name:	Kelly, Philip (2016)
Email:	NDJT027@live.rhul.ac.uk
Title of research project or grant:	A longitudinal investigation into the predictive nature of structural brain differences on future psychiatric symptomatology in adolescents exposed to childhood maltreatment, and the moderating influence of social support.
Project type:	Royal Holloway postgraduate research project/grant
Department:	Psychology
Academic supervisor:	Dr Catherine Sebastian
Email address of Academic Supervisor:	Catherine.Sebastian@rhul.ac.uk
Funding Body Category:	No external funder
Funding Body:	
Start date:	11/03/2018
End date:	30/09/2019

Research question summary:

Extant literature has shown that childhood maltreatment is a robust risk factor for a range of psychiatric disorders across the lifespan. The experience of maltreatment has also been shown to be significantly associated with a pattern of localised differences in cortical structure, similar to patterns of atypical structure found in clinical samples. However we know surprisingly little about the functional significance of maltreatment related structural differences and whether they represent true markers of latent vulnerability predictive of future psychiatric disorder.

The aims of the study are to systematically investigate the predictive nature of structural brain differences associated with childhood maltreatment on change in mental health symptomatology in maltreated adolescents, compared to non-maltreated counterparts, and to subsequently examine the moderation of this relationship by social support. We hypothesise that structural differences associated with the experience of maltreatment will be predictive of changes in symptomatology within maltreated individuals compared to non-maltreated individuals. Furthermore, we predict that the structural differences associated with maltreatment experience will be located primarily within cortical areas associated with emotional processing.

Research method summary:

This investigation uses secondary data collected as part of a larger longitudinal study exploring the impact of childhood maltreatment on brain structure, function, and behaviour. The study received full ethical approval from the UCL Research Ethics Committee (0896/002). Data for the longitudinal project was collected between 2011 and 2017, and this current project involves the analysis of historical data only.

84 children and adolescents aged between 10-14 years were recruited from London and the South-East of England. 40 had documented cases of maltreatment, and 43 were control participants matched on a number of factors, such as pubertal status, IQ and age. While the 40 children who experienced maltreatment represent a high-risk group, the secondary data analysis does not require contact with the participants or further data collection.

All participants underwent a structural imaging brain scan within a MRI scanner, and were given questionnaires measuring symptoms of common mental health disorders. Participants were given the questionnaires again after two years. These questionnaires included the Strengths and Difficulties Questionnaire (Goodman, 1997) and The Child and Adolescent Symptom Inventory (Gadow & Sprafkin, 2002); both commonly used within the field of maltreatment research.

In addition, at both time points, and at 6-month intervals between the two time points, the participants completed a questionnaire measuring social support (The Child and Adolescent Social Support Scale; Malecki & Demaray, 2002).

An analysis of the structural imaging brain scans, comparing the two groups (maltreated vs. non-maltreated), will identify differences in brain structure associated with maltreatment. Values taken from areas that show group differences will be used to explore the relationship with change in symptom scores at two year follow-up; and subsequently whether the strength of this relationship is altered by social support.

Risks to participants

Does your research involve any of the below?

Children (under the age of 16),

Yes

Participants with cognitive or physical impairment that may render them unable to give informed consent,

No

Participants who may be vulnerable for personal, emotional, psychological or other reasons,

Yes

Participants who may become vulnerable as a result of the conduct of the study (e.g. because it raises sensitive issues) or as a result of what is revealed in the study (e.g. criminal behaviour, or behaviour which is culturally or socially questionable),

No

Participants in unequal power relations (e.g. groups that you teach or work with, in which participants may feel coerced or unable to withdraw),

No

Participants who are likely to suffer negative consequences if identified (e.g. professional censure, exposure to stigma or abuse, damage to professional or social standing),

Yes

Details,

This project forms part of a larger longitudinal investigation exploring the impact of maltreatment on brain structure, function, and behaviour.

Full ethical approval for the longitudinal project was obtained by UCL Research Ethics Committee (0896/002). The researcher and applicant, Philip Kelly, is identified on a list of researchers engaged with the project as required by the UCL ethics board.

This project involves secondary data analysis only, and therefore will not require the collection of further data. The data collection used within this project began in 2014 and was completed in early 2017. Data is fully anonymised and stored confidentially on password protected drives and password protected central UCL servers. Assent to participate in the study was obtained for all children. Consent was obtained from the legal parent. The following ethical considerations were detailed at the time of data collection and presented below for information.

The importance of ensuring that all children and their families who participated in the study found it a positive experience. All children received a certificate, a book (*Blame My Brain: The Amazing Teenage Brain Revealed* by Nicola Morgan), and a copy of their structural brain scan on a CD. A clear and developmentally appropriate description of the study was provided, which include the reassurance that other children were included in the study and that we would not be looking at their results in isolation.

As the nature of a study on brain differences in children with a history of childhood maltreatment could risk stigmatising those children if a careful and thoughtful approach was not adopted. The study was framed in the context of resilience and a partial aim of this study is to explore the potentially positive role of social support.

The experience of having a brain scan is unfamiliar and can be anxiety provoking for children and parents. Experienced scanner operators who work with children were used throughout the data collection, and appropriate information was provided prior to the scan. Familiarisation to the scanning environment for the child and the family occurred prior to the scan, allowing for the opportunity to ask questions.

In the scenario in which a suspected abnormality was revealed by a scan, standard protocol involving a UCL neuroradiologist contacting the participant's GP was followed. No scanning was undertaken without prior participant (or parent/guardian of participant) consent to contact the GP.

In the case of identifying undetected abuse, the project lead (Prof. McCrory), and experienced clinical psychologist within NSPCC service who had routinely provided assessment and treatment to adolescents who had experienced various forms of adverse experiences,



including maltreatment, facilitated a multiagency response if welfare concerns arose in relation to any of the participants. Confidentiality was explicitly detailed to the parents and children prior to consent to take part in the study.

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## Design and Data

Does your study include any of the following?

Will it be necessary for participants to take part in the study without their knowledge and/or informed consent at the time?.

No

Is there a risk that participants may be or become identifiable?.

No

Is pain or discomfort likely to result from the study?.

No

Could the study induce psychological stress or anxiety, or cause harm or negative consequences beyond the risks encountered in normal life?.

No

Does this research require approval from the NHS?.

No

If so what is the NHS Approval number.

Are drugs, placebos or other substances to be administered to the study participants, or will the study involve invasive, intrusive or potentially harmful procedures of any kind?,

No

Will human tissue including blood, saliva, urine, faeces, sperm or eggs be collected or used in the project?,

No

Will the research involve the use of administrative or secure data that requires permission from the appropriate authorities before use?,

No

Will financial inducements (other than reasonable expenses and compensation for time) be offered to participants?,

No

Is there a risk that any of the material, data, or outcomes to be used in this study has been derived from ethically-unsound procedures?,

No

Details,

### Risks to the Environment / Society

Will the conduct of the research pose risks to the environment, site, society, or artifacts?,

No

Will the research be undertaken on private or government property without permission?,

No

Will geological or sedimentological samples be removed without permission?,

No

Will cultural or archaeological artifacts be removed without permission?,

No

Details,

### Risks to Researchers/Institution

Does your research present any of the following risks to researchers or to the institution?

Is there a possibility that the researcher could be placed in a vulnerable situation either emotionally or physically (e.g. by being alone with vulnerable, or potentially aggressive participants, by entering an unsafe environment, or by working in countries in which there is unrest)?,

No

Is the topic of the research sensitive or controversial such that the researcher could be ethically or legally compromised (e.g. as a result of disclosures made during the research)?,

No

Will the research involve the investigation or observation of illegal practices, or the participation in illegal practices?,

No

Could any aspects of the research mean that the University has failed in its duty to care for researchers, participants, or the environment / society?.

No

Is there any reputational risk concerning the source of your funding?.

No

Is there any other ethical issue that may arise during the conduct of this study that could bring the institution into disrepute?.

No

Details,

#### Declaration

By submitting this form, I declare that the questions above have been answered truthfully and to the best of my knowledge and belief, and that I take full responsibility for these responses. I undertake to observe ethical principles throughout the research project and to report any changes that affect the ethics of the project to the University Research Ethics Committee for review.

Certificate produced for user ID, NDJT027

Date:	01/03/2018 12:03
Signed by:	Kelly, Philip (2016)
Digital Signature:	Philip Kelly
Certificate dated:	3/1/2018 12:53:08 PM
Files uploaded:	

*Appendix 11. Characteristics of the maltreatment histories within the maltreated group*

<b>Kaufman</b> <sup>a</sup>	<b>Count n (%)</b>	<b>Severity</b>			
		<b>Mean</b>	<b>SD</b>	<b>Min</b>	<b>Max</b>
Physical abuse	3 (9.10)	1.00	1.00	1.00	1.00
Neglect	25 (75.76)	4.67	1.15	4.00	6.00
Sexual abuse	3 (9.10)	1.00	1.00	1.00	1.00
Emotional abuse	32 (96.97)	3.67	0.58	3.00	4.00
Domestic violence	18 (54.55)	1.89	1.13	1.00	4.00
<b>Total</b>		<b>7.89</b>	<b>4.23</b>	<b>3.00</b>	<b>14.00</b>
<b>CTQ</b> <sup>b</sup>		<b>Mean</b>	<b>SD</b>	<b>Min</b>	<b>Max</b>
Emotional abuse		8.12	3.64	5.00	21.00
Physical abuse		5.85	3.41	0.00	23.00
Sexual abuse		5.06	0.35	5.00	7.00
Emotional neglect		9.21	4.24	5.00	24.00
Physical neglect		8.00	3.33	5.00	17.00
<b>Combined total</b>		<b>36.09</b>	<b>12.54</b>	<b>24.00</b>	<b>90.00</b>

<sup>a</sup> Social worker rated based on file report

<sup>b</sup> Childhood Trauma questionnaire, child-rated

#### **T1-weighted scan parameters**

- 176 slices; slice thickness = 1 mm; gap between slices = 0.5 mm
- echo time = 2730 msec
- repetition time = 3.57 msec
- field of view = 256 mm x 256mm<sup>2</sup>; matrix size = 256 x 256
- voxel size = 1 x 1 x 1 mm resolution.
- The scanning time was 5.5 min. Foam padding was used against the sides and the back of the head of the participant, to minimize head motion. Ear buds attenuated scanner noise.

#### *Appendix 12. T1-weighted scan parameters*

#### **FreeSurfer pre-processing protocol**

- White matter points were defined from estimates of their location based on their position in Talairach space as well as the voxel and local neighbourhood intensities.
- Each hemisphere underwent automatic computation of skull stripping and classification of white and gray matter.
- A two-dimensional tessellated mesh consisting of over 300,000 vertices is constructed over the white matter surface to distinguish the gray-white matter boundary. This mesh is expanded outwards to meet the gray matter and pial surface boundary.
- The estimated boundaries were manually edited for inconsistencies and errors in the previous steps, and control points and edits to the brain mask were made where necessary.
- The structural measures were calculated in native space, which is then transformed to a spherical atlas and registered to common atlas, preserving the vertex identities.

#### *Appendix 13. FreeSurfer pre-processing protocol*

Appendix 14. Correlations between cortical structure and file-report maltreatment severity (Kaufman measure)

Structural Index	Structural cluster	Kaufman					Total
		EA	PA	SA	NG	DV	
CT	Caudal middle frontal (A)	0.055	-0.02	-0.079	0.302	0.124	0.114
		[-0.21,0.27]	[-0.37,0.34]	[-0.37,0.19]	[-0.29,0.65]	[-0.30,0.49]	[-0.33,0.50]
IGI	Superior frontal (B)	0.193	0.364	0.167	0.212	-0.318	0.227
		[-0.15,0.52]	[-0.01,0.67]	[-0.12,0.49]	[-0.07,0.44]	[-0.63,0.06]	[-0.11,0.51]
	Pars Opercularis (C)	0.098	-0.148	-0.022	-0.001	0.079	-0.134
		[-0.19,0.35]	[-0.46,0.18]	[-0.27,0.28]	[-0.42,0.30]	[-0.30,0.42]	[-0.43,0.15]
	Superior parietal (D)	0.066	0.015	0.139	0.448	-0.057	0.183
	[-0.13,0.26]	[-0.43,0.40]	[-0.13,0.38]	[-0.03,0.71]	[-0.38,0.21]	[-0.32,0.55]	
	Fusiform (E)	0.039	0.311	0.226	0.09	0.068	0.306
		[-0.41,0.45]	[-0.06,0.62]	[-0.26,0.69]	[-0.27,0.41]	[-0.33,0.44]	[-0.12,0.65]

Notes: EA=emotional abuse;PA=physical abuse; SA=sexual abuse; NG=neglect; DV=domestic violence; Bias corrected and accelerated (BCa) bootstrapping applied (95%CI)

Appendix 15. Correlations between cortical structure and self-report maltreatment severity (CTQ measure)

		<b>CTQ</b>					
<b>Structural Index</b>	<b>Structural cluster</b>	<b>EA</b>	<b>PA</b>	<b>SA</b>	<b>EN</b>	<b>PN</b>	<b>Total</b>
<b>CT</b>	<b>Caudal middle frontal (A)</b>	0.95	-0.09	-0.14	0.094	-0.155	0.016
		[-0.25,0.42]	[-0.31,0.08]	[-0.37,0.14]	[-0.28,0.40]	[-0.48,0.17]	[-0.29,0.36]
<b>IGI</b>	<b>Superior frontal (B)</b>	-0.007	0.013	-0.099	-0.054	0.327	0.084
		[-0.42,0.38]	[-0.01,0.14]	[-0.29,0.03]	[-0.52,0.33]	[-0.05,0.63]	[-0.31,0.41]
	<b>Pars Opercularis (C)</b>	-0.065	-0.118	-0.317	-0.313	-0.021	-0.165
		[-0.41,0.34]	[-0.38,0.20]	[0.11,-0.46]	[-0.56,-0.03]	[-0.30,0.26]	[-0.42,0.09]
		<b>Superior parietal (D)</b>	-0.02	0.144	0.205	-0.077	0.231
[-0.40,0.35]	[-0.13,0.38]		[-0.19,0.68]	[-0.44,0.31]	[-0.13,0.49]	[-0.26,0.36]	
<b>Fusiform (E)</b>	0.162	0.07	0.105	0.221	0.185	0.212	
	[-0.23,0.53]	[-0.29,0.57]	[-0.08,0.37]	[-0.20,0.60]	[-0.23,0.57]	[-0.19,0.63]	

Notes: EA=emotional abuse; PA=physical abuse; SA=sexual abuse; EN=emotional neglect; PN=physical neglect; Bias corrected and accelerated (BCa) bootstrapping applied (95%CI)

Appendix 16. Bivariate correlations between social support and file-report measures of maltreatment experience (Kaufman)

<b>Social support</b>	<b>Kaufman</b>					
	<b>Physical abuse</b>	<b>Neglect</b>	<b>Sexual abuse</b>	<b>Emotional abuse</b>	<b>Domestic violence</b>	<b>Total Severity</b>
<b>Parent Frequency</b>	-0.28 [-0.70,0.08]	-0.29 [-0.50,0.01]	-0.41* [-0.75,-0.30]	-0.06 [-0.43,0.28]	-0.18 [-0.54,0.19]	-0.29 [-0.54,0.15]
<b>Parent Importance</b>	0.20 [-0.11,0.46]	-0.19 [-0.52,0.12]	-0.24 [-0.52,0.16]	0.24 [-0.32,0.51]	-0.1 [-0.46,0.25]	-0.09 [-0.47,0.26]
<b>Teacher Frequency</b>	-0.11 [-0.38,0.16]	-0.20 [-0.49,0.13]	-0.25 [-0.54,-0.18]	-0.01 [-0.50,0.33]	-0.06 [-0.41,0.27]	-0.2 [-0.50,0.11]
<b>Teacher Importance</b>	0.22 [-0.05,0.49]	-0.24 [-0.53,0.06]	-0.16 [-0.35,0.10]	-0.16 [-0.39,0.13]	-0.06 [-0.44,0.32]	-0.28 [-0.55,0.01]
<b>Classmate Frequency</b>	-0.11 [-0.37,0.11]	-0.52* [-0.72,-0.21]	-0.17 [-0.39,-0.10]	-0.18 [-0.38,0.07]	0.11 [-0.25,0.41]	-0.43* [-0.68,-0.11]
<b>Classmate Importance</b>	0.08 [-0.14,0.28]	-0.44* [-0.68,-0.18]	-0.16 [-0.38,-0.09]	-0.01 [-0.43,0.26]	0.01 [-0.37,0.35]	-0.38* [-0.66,-0.01]
<b>Close friend Frequency</b>	-0.14 [-0.36,0.02]	-0.40* [-0.67,-0.01]	-0.25 [-0.55,-0.16]	-0.09 [-0.51,0.24]	0.03 [-0.33,0.38]	-0.34 [-0.61,0.05]
<b>Close friend Importance</b>	0.05 [-0.14,0.25]	-0.38* [-0.70,-0.06]	0.17 [-0.40,0.10]	-0.06 [-0.46,0.18]	-0.23 [-0.52,0.10]	-0.43* [-0.74,-0.12]
<b>Total Frequency</b>	-0.21 [-0.54,0.15]	-0.43 [-0.71,-0.07]	-0.32 [-0.42,-0.34]	-0.13 [-0.46,0.14]	-0.06 [-0.43,0.36]	-0.41 [-0.74,-0.03]
<b>Total Importance</b>	0.12 [-0.29,0.48]	-0.43 [-0.68,-0.13]	-0.11 [-0.32,0.2]	-0.07 [-0.33,0.10]	-0.03 [-0.36,0.32]	-0.42 [-0.65,-0.18]

\*p<.05; Bias corrected and accelerated (95% CI) bootstrapping applied and reported



Appendix 17. Bivariate correlations between social support and self-report measures of maltreatment experience (CTQ)

Social support	CTQ					
	Emotional abuse	Physical abuse	Sexual abuse†	Emotional neglect	physical neglect	Total Severity
<b>Parent Frequency</b>	-0.43*	-0.57*	0.23	-0.63*	-0.42*	-0.61*
	[-0.73,-0.02]	[-0.80,0.06]	[-,-]	[-0.83,-0.22]	[-0.73,0.22]	[-0.83,0.03]
<b>Parent Importance</b>	-0.12	-0.06	0.24	-0.3	-0.08	-0.18
	[-0.51,0.24]	[-0.33,0.30]	[-,-]	[-0.63,0.00]	[-0.43,0.36]	[-0.52,0.14]
<b>Teacher Frequency</b>	-0.23	-0.16	0.29	-0.46*	-0.14	-0.22
	[-0.59,0.11]	[-0.43,0.23]	[-,-]	[-0.73,-0.14]	[-0.63,-0.04]	[-0.66,0.08]
<b>Teacher Importance</b>	-0.07	0.07	0.29	-0.46*	-0.14	-0.22
	[-0.33,0.22]	[-0.11,0.42]	[-,-]	[-0.72,-0.18]	[-0.44,0.23]	[-0.48,0.06]
<b>Classmate Frequency</b>	-0.45*	-0.34	0.31	-0.44*	-0.48*	-0.50*
	[-0.74,0.11]	[-0.63,0.03]	[-,-]	[-0.71,0.14]	[-0.74,-0.19]	[-0.78,-0.21]
<b>Classmate Importance</b>	-0.14	-0.02	0.27	-0.39*	-0.19	-0.25
	[-0.41,0.16]	[-0.24,0.31]	[-,-]	[-0.63,-0.14]	[-0.48,0.18]	[-0.49,0.02]
<b>Close friend Frequency</b>	-0.34	-0.35	0.25	-0.46*	-0.33	-0.45*
	[-0.67,0.13]	[-0.79,-0.08]	[-,-]	[-0.71,-0.12]	[-0.58,-0.05]	[-0.74,-0.14]
<b>Close friend Importance</b>	-0.15	-0.05	2.6	-0.27	-0.09	-0.19
	[-0.56,0.21]	[-0.47,0.22]	[-,-]	[-0.61,0.04]	[-0.38,0.24]	[-0.56,0.10]
<b>Total Frequency</b>	-0.48	-0.44	0.33	-0.63	-0.47	-0.61
	[-0.78,-0.03]	[-0.69,-0.07]	[-,-]	[-0.81,-0.31]	[-0.70,-0.16]	[-0.80,-0.35]
<b>Total Importance</b>	-0.17	-0.03	0.37	-0.39	-0.14	-0.24
	[-0.56,0.22]	[-0.44,0.41]	[-,-]	[-0.73,-0.04]	[-0.44,0.15]	[-0.59,0.05]

\*p<.05; Bias corrected and accelerated (95% CI) bootstrapping applied and reported. † CI could not be computed

