Internalising and Externalising in Parents and Offspring: The Impact of Sex and Early Life Adversity

By

Emerald O'Neill-Murchison

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Abstract

The aim of this study was to investigate associations between maternal and paternal externalising, experiences of early life adversity (ELA), internalising and externalising in adult offspring, and the role of sex. Using data collected in wave three of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) we utilised structural equation modelling to test a hypothesised moderated mediation model associating maternal and paternal externalising, and internalising and externalising in offspring, mediated by early life adversities and moderated by sex. Our study included a total of *n*=26,728 participants which were randomly split into two groups and analysed separately to test whether it was possible to replicate our own results. No direct association was demonstrated between maternal externalising or paternal externalising and internalising or externalising in offspring; experiences of ELA were associated only with internalising in offspring, but not with externalising; ELA was associated with maternal externalising and paternal externalising in females only. The relationship between paternal externalising and internalising in male offspring was mediated by ELA, as was the relationship between maternal externalising and internalising in male offspring. The relationship between parental externalising and internalising in females however was not mediated by ELA. Results did not provide support for the study's hypotheses.

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Introduction

Human psychopathology is deeply complex, encompassing a breadth of symptoms and disorders which negatively impact the lives of individuals worldwide, indiscriminate of age, sex, or nationality. Many factors influence the development of mental, or behavioural symptoms both directly, and through complex interactions; experiences of childhood trauma, parental antisociality/criminality and parental problematic substance use being just a few. These experiences are often associated with an elevated risk of developing a mental or behavioural disorder in childhood and/or adulthood, and additionally, are often co-occurring or potential causes of one another. Despite this, their interactions are still poorly understood, albeit key for targeted interventions. Thus, investigating the relationship between parental externalising, early life adversity (ELA) and psychopathology symptomatology in adult offspring should provide more insight into how these variables interact, and how they may impact psychopathology.

Defining Internalising and Externalising

To start, it is crucial define the main concepts of this thesis; internalising and externalising. Internalising and externalising are two fundamental dimensions, originally identified in child psychopathology research but since replicated in adult samples, derived from research examining the latent structure of mental disorders (Achenbach, 1996; Kotov et al., 2017). These two dimensions account for commonly observed patterns of comorbidity among mental disorders and suggest that common underlying liabilities accounted for by the two dimensions explain most psychiatric comorbidity (Keyes et al., 2012). The internalising dimension (also referred to as internalising symptoms) is considered to account for disorders which are characterised by negative affect, indicate a proclivity towards mood and anxiety disorders, and can be broadly categorised into two clusters; a distress cluster and a fear cluster (Curran et al., 2016; March-Llanes et al., 2017; Keyes et al., 2012; Kotov et al., 2017).

Examples of disorders belonging to the distress cluster include Major Depressive Disorder (MDD), Generalised Anxiety Disorder (GAD), and Post-Traumatic Stress Disorder (PTSD) while examples of those belonging to the fear cluster include Specific Phobias, Panic Disorder (PD), Social Phobia, and Obsessive-Compulsive Disorder (OCD) (Achenbach, 1996; Kotov et al., 2017).

Conversely, the externalising dimension (or externalising symptoms) accounts for disorders which are characterised by disinhibition and antagonistic features. This dimension encompasses symptoms which have a dimension of antisocial behaviour and/or substance-related problems (for which the feature disinhibition is prominent), some examples being Antisocial Personality Disorder (ASPD), Conduct Disorder (CD), and Substance Use Disorders/Dependencies (Kotov et al., 2017).

The Impact of Parental Externalising on Offspring

The impact of parental externalising, on offspring, such as parental anti-sociality, parental substance use disorders, and parental criminal offending, is the focus of numerous studies. Empirical evidence consistently indicates offspring of parents exhibiting externalising symptoms or engaging in antisocial and criminal behaviours, are at a higher risk of developing internalising and externalising symptoms and engaging in similarly antisocial behaviours themselves (Besemer, 2014; Foley et al., 2001; Furtado et al., 2006; Herndon & Iacono, 2005; Kim et al., 2009; Long et al., 2018; Whitten et al., 2019).

While the externalising dimension of psychopathology accounts for a number of disorders, research has focused heavily on the influence of parental substance use disorders (SUD), predominantly alcohol use disorder (AUD). Additionally, many studies investigating externalising behaviour typically focus on paternal influence (Kim et al., 2009). This is probably due to the common assumption that males are more likely to develop externalising symptoms than females.

Two hypotheses have been suggested in regards to the severity of the impact that parental criminality/offending behaviour has on offspring (Whitten et al., 2019). The first, coined as the Sensitive Period Hypothesis, considers the age of the child in relation to the time of the parental offending to play an important role in the severity of the impact, with early childhood being argued as the most sensitive period (Besemer, 2014; Putnam, 1997). Younger children are proposed to be more vulnerable to the externalising behaviour of their parents than older children are, as they would be expected to have more direct exposure to such behaviours consequently increasing the likelihood of them modelling such behaviours (Whitten et al., 2019). The second hypothesis regarding intergenerational transmission of offending behaviours suggests that frequency of parental offending, rather than age of the child at the time of the offending, has the greatest influence on offspring behaviour.

Whitten et al., (2019), as well as a number of other studies on the intergeneration transmission of offending behaviour, support this hypothesis. They found parental conviction rate to be a significant predictor of conviction rates for both sons and daughters, and that the continuity and severity of parental offending may be more important than the timing of parental offending.

Maternal Versus Paternal Externalising

There is also some evidence for a differential role of parental gender on the impact of their externalising on their offspring (Long et al., 2018). A number of studies indicate that maternal externalising presents a greater risk to the development of externalising behaviours in both male and female offspring (Long et al., 2018). An investigation into the role of parental and offspring sex on externalising symptoms among offspring of parents with AUD found that maternal AUD consistently conferred greater risk for both sons and daughters than paternal AUD did (Long et al., 2018). Similarly, Herndon and Iacono (2005) reported that while diagnoses of adult antisocial behaviour (AAD) (according to DSM-III-R criteria) in

both mothers and fathers increased the odds of children developing a range of disorders (in both the externalising and internalising dimensions), maternal diagnoses of antisocial behaviour significantly increased the odds of developing ADHD, or exhibiting MDD or any other internalising disorder above that of paternal diagnoses.

However, other researchers investigating the impact of parental offending on offspring have reported maternal offending does not have a significantly greater effect on offspring than paternal offending (Whitten et al., 2019).

Long et al. (2018) note that the maternal externalising may confer a higher risk as offspring may be more likely to live with their mothers. In such situations it could be expected that maternal externalising would exert a stronger influence on offspring than paternal externalising. To test this hypothesis they ran an additional analysis on household composition and evidence of a socialisation effect was demonstrated. Wherein, offspring were more likely to develop externalising symptoms when residing with the affected (AUD) parent, and in the event offspring resided with two affected parents, risk conferred by maternal AUD and paternal AUD was more equal. Additionally, having two affected parents substantially increased the risk of offspring developing externalising symptoms compared to offspring of one affected parent.

Another question integral to forming a comprehensive understanding of the impact of sex is whether maternal and paternal externalising differentially effect offspring as a product of offspring sex. In their study on parental AUD, Long et al., (2018) reported that, within the broad spectrum of externalising symptoms (rather than specifically AUD), their findings supported sex-specific transmission of psychopathology, where maternal AUD increased risk for a number of disorders in female offspring more so than in male offspring, and paternal AUD increased risk for sons more so than daughters (though risk was increased for both females and male offspring regardless of parent sex compared to offspring of unaffected

parents). Similarly, while having a father with alcohol use disorder was found to elevate the risk of offspring developing externalising symptoms regardless of offspring sex, this risk is even higher for sons (Furtado et al., 2006).

Thus, conclusions regarding the differential effects of maternal versus paternal externalising behaviours are inconsistent so far, and need to consider the interaction with sex of the child. As emphasised by Long et al., (2018) understanding whether the transmission of externalising symptoms from parent to child differs by parental or offspring sex is critical for informing etiology of disorders in the internalising and externalising dimensions and improving intervention efforts.

Early Life Adversity

Outside of parental externalising there are innumerable other negative experiences which can occur in early life. These experiences, be it one time events, or ongoing circumstances, are referred to as early life adversity (ELA) and have been the subject of a multitude of studies. The vast majority of which have presented strong evidence that such experiences have a strong and cumulative impact on adult health; with links being made to negative physical and psychological health outcomes (Felitti et al., 1998; Kessler et al 1997; Moreira et al 2020). An association between ELA and all classes of disorders, including both internalising and externalising, throughout the life course, in which ELAs trigger, maintain, aggravate and increase the prevalence and recurrence of psychiatric disorders has been demonstrated in a multitude of studies (Kessler et al., 2010).

Defining Early Life Adversity

Early life adversity (ELA) as defined by Goff and Tottenham (2015) is the exposure to adverse events during childhood which threatens or harms the emotional and/or physical well-being of the child to an extent that exceeds their ability to cope. Also commonly referred to as adverse childhood experiences (ACE), a term that became prevalent in literature post

the seminary ACE studies in the 1990s, or childhood trauma, these experiences and the consequential impacts on individual's psychological and physical wellbeing have been well studied (see Mersky et al., 2013 for an overview).

The term childhood maltreatment (CM) is also frequently used in the literature to refer to a collection of ELAs. These are; neglect (physical and emotional), in which there is a failure to provide the basic care needs (physical), emotional needs, and psychological needs of a child (emotional neglect); physical abuse; which is defined as physical assault which poses risk, or results in injury of a child; sexual abuse; referring to any kind of sexual conduct involving a child; and emotional abuse which refers to any conduct (such as verbal assaults or demeaning behaviour) assaulting a child's well-being or sense of worth (Berstein et al., 2003). Through estimates of worldwide prevalence, these are suggested to be common experiences for young people worldwide (Vachon et al., 2015a**).**

In the interest of clarity, the terms CM and ACE will be used when referring to literature and research which specifically uses these terms. CM will also be used when referring to research focussing on only the combination of physical, emotional, and sexual abuse, and physical and emotion neglect. ELA will be used to refer to research and literature referring to childhood trauma as early life adversities, or any other term not including CM or ACE which refers specifically to traumatic experiences which occurred at 18 years or younger.

Co-occurrence of Early Life Adversities

It is a well-established finding that co-occurrence of ELAs is common, with the majority of individuals who experience ELA reporting they have experienced more than one form of maltreatment (Arata et al., 2005; Chartier et al., 2010; Curran et al., 2018; Felitti et al., 1998; Kessler et al 1997; Ney et al., 1994). Arata et al., (2007) report that of the different forms of CM, neglect (unspecified) is the form that is most often experienced in isolation,

with physical and sexual abuse being the two forms that were most infrequently experienced in isolation (Vachon et al., 2015a). Some forms of maltreatment have been found to be highly correlated with one another; physical and psychological abuse are an example of two forms of maltreatment which are highly likely to be experienced by the same individual (Arata et al., 2007; Ney et al., 1994). As such a longstanding challenge in the investigation of the impact of ELAs on later psychopathology has been capturing the direct impact of each form of ELA versus combined effects of ELAs (Kessler et al 1997; Vachon et al 2015a; Zhang et al., 2019).

Vachon and colleagues (2015a, 2015b) discuss the typical assumption that is held by society, and within research, that some forms of maltreatment are more harmful than others; an assumption which can be seen reflected in the legal system where certain forms of abuse are illegal and/or more harshly punished than others. Consequently, scientific literature predominantly focuses on physical and sexual abuse, two forms of maltreatment that are seen to be particularly harmful (Ney et al., 1994; Vachon et al., 2015a). Vachon and colleagues (2015a, 2015b) go on to report that findings from their studies suggest that different forms of maltreatment, with the exception of child sexual abuse, in fact have equivalent effects on psychopathology. The implication of this finding is effective treatment for any specific form of maltreatment is likely to have benefits for psychological health (Vachon et al., 2015b).

Though all non-sexual forms of maltreatment were found to be associated with a range of mental health problems, the evidence regarding sexual abuse is less consistent (Vachon et al., 2015a, 2015b). While experiences of sexual abuse were not found to be significantly related to psychopathology in their own studies, other researchers have reported their findings do indicate that child sexual abuse elevates the risk of psychopathology. Consequently, continued research is needed in order to draw a firm conclusion regarding the

relationship between experiences of child sexual abuse and the development of psychopathology.

Kirisci et al., (2001) suggest that child neglect (unspecified) is the most prevalent form of maltreatment and has a more severe impact on an individual's development than other forms of maltreatment, such as sexual or physical abuse. Arata et al., (2007) later report findings in line with this opinion, as did an earlier study by Ney et al., (1994) which indicated that the most severe psychological outcomes were associated with neglect, as opposed to abuse.

Parental Externalising and Early Life Adversity

As well as being associated with internalising and externalising in offspring, past research additionally demonstrates a relationship between parental externalising and ELA in offspring. Recent research investigating adverse childhood experiences (ACEs) among children of incarcerated parents found that children exposed to parental incarceration were far more likely to have other ACEs compared to children who were not (Turney, 2018). While 72.2% of children not exposed to parental incarceration had another ACE, only 14.3% of children who did have an incarcerated parent had no other ACEs. Additionally children exposed to parental incarceration were nine time more likely than their counterparts to experience abuse (unspecified) in their home, or witness violence.

Parental substance abuse has also been reported to have detrimental effects on the parenting role, endangering the welfare of children (Wolock et al., 1996). Numerous studies have reported significantly higher rates of child maltreatment in offspring of substance abusers, with some findings indicating that rates of physical and sexual abuse were increased by two-fold amongst individuals who reported parental substance abuse (Anda et al., 2002; Chaffin et al., 1996; Famularo et al., 1992; Walsh et al., 2003). Interestingly, findings from a study by Anda and colleagues (2002) indicate that depressive disorders among individuals

who were exposed to parental AUD are largely, perhaps even solely, due to the increased likelihood of experiencing ACEs in a household with parental substance abuse.

While there is the direct risk of maltreatment by parents with antisocial behaviours or substance abuse disorders, parental externalising additionally increases a general risk of exposure to abuse in offspring by making children vulnerable due to a potential lack of supervision (Walsh et al., 2002).

Internalising and ELA

Setting aside parental externalising for a moment; numerous studies have provided evidence of a strong positive relationship between experiences of early life adversity and the subsequent development of internalising disorders and symptoms, such as anxiety disorder or mood disorders, during adolescence and in later life (Kim et al., 2003). Curran et al., (2018) found that higher scores on the internalising dimension were predictive of increased likelihood of experiencing higher levels of ELAs, exhibiting the positive correlation between experiences of childhood trauma and internalising. It has also been established that a strong graded relationship exists between the prevalence and risk of affective disturbances and the number of ELAs experienced by an individual; a phenomenon referred to as a dose-response relationship (Anda et al., 2006).

Anxiety

In a systematic review statistically significant associations were observed between physical abuse, emotional abuse, and neglect (unspecified) and a significant increased risk of developing an anxiety disorder (Norman et al., 2012). Similarly, a review by Carr et al., (2013) also reported that early life stressors appeared to have a powerful relationship with the development of mood and anxiety disorders. Reviewing literature regarding traumatic experiences of physical abuse, emotional abuse, sexual abuse and neglect (emotional,

physical and unspecified) observed that all subtypes of early life stress were associated with anxiety disorders, with only one study reporting contrary results (Wonderlich et al., 2007).

While anxiety appears to have a link to ELA overall, links have also been reported between ELA and specific disorders; post-traumatic stress disorder (PTSD) in particular was associated with early life adversity (Jonas et al., 2011). Carr et al., (2013) also noted that sexual abuse was reported to be particular associated with PTSD, panic disorder, OCD and agoraphobia; while emotional abuse has a strong correlation with social phobia and PTSD (especially combined with a substance abuse disorder).

Mood Disorders

The study of mood disorders and their association to childhood trauma has predominantly focussed on Major Depressive Disorder. In 1999, Kender and colleagues reported evidence that stressful life events truly and substantially increased risk of subsequently developing major depression. In line with this, current and past literature alike have established the existence of a strong positive relationship with ELA and MDD (Kim et al., 2003). Additionally, it has been found that ELA contributes to the persistence and severity of mood disorders (Carr et al., 2013).

Wiersma et al., (2009) established that multiple stresses in early life can be independent determinants of chronic depression, a statement which is congruent with the vast majority of research. Childhood physical abuse, sexual abuse, as well as unspecified neglect, each as independent experiences of trauma, have been reported to be predictive of the subsequent development of mood disorders, particularly MDD and Bipolar disorder (see review by Carr et al., 2013). It was noted that in this review only one study did not find an association between physical abuse and mood disorders (Wonderlich et al., 2007). A strong link was also observed between emotional abuse and emotional neglect and depressive symptoms and major depressive disorder, with emotional neglect also being associated with

earlier onset of first depressive episodes (see review by Carr et al., 2013). However, one of the studies reviewed reported that physical neglect was not a significant determinant of subsequent mood disorders (Wonderlich et al., 2007).

The findings reported by those studies reviewed by Carr et al., (2013) are consistent with findings from other studies investigating the relationship between ELA and the development of mood disorders across the life course. In 2007, Arata and colleagues reported that participants with maltreatment histories in childhood were more depressed than control participants. Furthermore a systematic review reported that individuals who had been physically abused, emotionally abused and neglected (unspecified), were at a higher risk of developing depressive disorder than non-abused counterparts (Norman et al., 2012). It has also been reported that both the risk and prevalence of depressed mood increases as a function of the number of childhood traumatic exposures reported (Felitti et al., 1998).

In reviewing the literature it is clear that a well-established association between ELA and both mood disorders and anxiety disorders exist, in which experiences of ELA contribute to increased risk, and severity of disorders in the internalising dimension.

Externalising and ELA

As with internalising disorders, evidence of a strong positive association between ELA and externalising disorders has been exemplified across numerous studies. This significant positive relationship is evident in the findings of Curran et al., (2018), who also report a higher degree of interpersonal maltreatment in childhood is indicative of increases in externalising across the life course. They also noted that externalising was associated with experiences of family violence, physical abuse, sexual abuse and physical neglect.

Behavioural Problems/Offending Behaviours

Findings most often support a positive correlation between experiences of ELA and behavioural or conduct problems. This is evident in the findings of both Dodge et al., (1995),

and Boden et al., (2010) who found various ELAs to be positively associated with conduct problems in young people (under 18) (Zhang et al., 2019). A review aimed at demonstrating psychopathic subtypes differ in terms of ACEs for example, reported that adult women with violent behaviours were found to have experienced more ACEs than non-offending women (Moreira et al., 2020). Findings from a twin study reviewed, conducted by Schwartz et al., (2019), demonstrate that a higher number of ACEs was significantly associated with a higher prevalence of antisocial behaviour (Moreira et al., 2020). Additionally, in comparison to their co-twins, twins exposed to a higher level of ACEs had a higher likelihood of engaging in antisocial behaviours. Similarly, childhood experiences of physical abuse and neglect (unspecified) have been associated with the odds of developing childhood behavioural or conduct problems doubling (Norman et al., 2012). It has been found also that the predictive effects of the positive relationship between ELAs and disruptive behavioural disorders persisted throughout the life course, not only throughout adolescence (see review by Carr et al., 2013).

Personality Disorders

A number of Personality Disorders are categorised as externalising disorders, and are reported to be positively associated with experiences of ELA. One study found that participants who had documented histories of physical abuse, sexual abuse and neglect (unspecified) during childhood were more than four times as likely to develop a personality disorder than non-neglected, non-abused participants (Johnson et al., 1999). The finding of a positive association between ELA and personality disorders was also supported by Carr et al., (2013) in their literature review. While the current research does support a positive association between ELA and subsequent PDs, it should be noted that there are still a number of studies which have found no significant association between some ELAs and PDs (Laporte et al., 2011; Wonderlich et al., 2007)

Antisocial Personality Disorder (ASPD)/Antisocial traits. Research has consistently found a relationship between physical abuse and the development of ASPD as well as criminal behaviour (Ball et al., 2009; Lobbestael et al., 2010; Moreira et al., 2020; Schoor et al., 2020). In fact, the link between physical abuse in childhood and ASPD was the most consistent finding of Schoor et al.'s., (2020) systematic review.

Despite numerous studies reporting a significant association between physical neglect and ASPD, several studies report finding no such significant association (Kim et al., 2016; see Schorr et al., 2020 for review). Interestingly Lobbestael et al., (2010) also report that sexual abuse is not linked to ASPD, though it is linked to a number of other personality disorders including paranoid, schizoid, borderline and avoidant traits.

Substance Abuse and Dependence

Childhood trauma is widely considered to be an important risk factor for substance use, abuse, and dependence (Dube et al., 2003). The prevalence of ELA has been consistently reported to be elevated in individuals with substance use disorders (Anda et al., 2006; Anderson and Teicher, 2009; Dube et al., 2003; Keyes et al., 2011; Lijffijt et al., 2014).

Childhood trauma and risk of substance use disorders share a dose-response relationship. This claim is supported by findings from studies such Dube et al., (2003), in which the likelihood of ever having drug problems or a substance addiction increased as ACE scores increased; and Anda et al., (2006) in which substance use and abuse also increased as ACE scores increased, with the risk of alcoholism for individuals with four or more ACEs increasing 7.2-fold compared to individuals with less than four ACEs. ELA is associated with increased opportunities to try substances, as well as to an earlier onset of use for a number of substances (stimulates, opiates, sedatives and alcohol) than individuals who experienced no, or less severe trauma in childhood (Lijffijt et al., 2014). Additionally, it is apparent that

experiencing ELA increases the risk of transition from experimental to regular use of marijuana, cocaine and other stimulants, nicotine and sedatives (Lijffijt et al., 2014).

Varying findings regarding the relationship between specific experiences of trauma and substance use have been reported. In ten studies reviewed by Carr et al., (2013), physical abuse was associated with substance abuse disorders, compared to only one study which did not find a significant association. Child sexual abuse (CSA) has also been associated with subsequent substance use and dependence, with youth who have experienced a combination of physical and sexual abuse exhibiting especially high risk of substance use (Carr et al., 2013; Moran et al., 2004; Nelson et al., 2006). Additionally, neglect (unspecified) was found to be a particularly salient risk factor for the development of substance use disorders (Kirisci et al., 2001).

However, while the link between ELA and substance use/abuse and dependency is well established, there are still many studies with contrary findings. One study for example, reports higher rates of substance use were only identified in youth who reported having experiences of both physical abuse and neglect (with or without sexual abuse), while other maltreatment groups had the same rate of substance abuse as non-maltreatment youth (Arata et al., 2007).

Research has furthermore predominantly focussed on alcohol use and abuse. As with other substance use disorders overall, ELA is a consistent risk factor for early onset of drinking, as well as alcohol use disorders (Keyes et al., 2011). Though the strength of this relationship is attenuated in studies where family history of alcoholism is controlled for, results continue to indicate a persistent relationship between ELA and adult risk of alcohol use disorders (Keyes et al., 2011; Nelson et al., 2002; Pilowsky et al., 2009). One twin study found that increased exposure to ACEs was positively associated with a higher prevalence of alcohol use problems (Schwartz et al., 2019).

As with substance use disorders more generally, physical abuse has been found to be significantly associated with an alcohol abuse/dependence diagnosis, as has emotional abuse (Norman et al., 2012). Interestingly however, though identified in other studies as a salient risk factor in the development of substance use disorders, Norman and colleagues (2012) did not find that child neglect (unspecified) was significantly associated with problem drinking. They also report no evidence of a dose-response relationship between the frequency of abuse and/or neglect and alcohol use issues, contrary to the findings of many other studies.

Gender Differences in Internalising and Externalising

To begin, it should be clarified that current and past research use both the terms gender and sex, with the two terms appearing interchangeably throughout the literature. For example, many studies refer to their participants as male or female, however their discussions' use the term gender as opposed to sex. In reviewing the literature, we were therefore interested in both sex differences and gender differences, and refer to either sex or gender in accordance to the terms used by specific studies.

The predominant gender difference that is discussed throughout the literature is that following experiences of childhood trauma, internalising appears to be more strongly associated in females and externalising more strongly associated in males (Current et al., 2018; Eaton et al., 2012; Grant et al., 2006; Steeger et al., 2017). In line with this, of the 69 studies reviewed by Grant et al., (2006) which reported a moderating effect for sex, 39 reported males were more likely to respond to stressors with externalising symptoms and females with internalising symptoms. This finding however has been inconsistent, and a number of other studies report finding no systemic sex difference in the associations between ELA and psychopathology; instead reporting that both female and males show similar responses to stressful life events (Kessler et al., 1997*;* Kim et al., 2003). One such study by Arata and colleagues (2007) found that while there were overall gender differences in their

results, there was no evidence for a significant interaction between maltreatment group and gender. From this we can infer that negative outcomes were not related to how these gender differences interacted with child maltreatment.

It is also important to note that independent of experiences of ELA, many studies have reported results indicating that women show markedly higher prevalence rates of disorders in the internalising dimension while men demonstrate a higher prevalence of disorders in the externalising dimension such as ASPD, and substance dependence (Grant & Weissman, 2007; Kessler et al.,1993, 1994; Eaton et al., 2012). The reasons for the apparent difference in rates of internalising and externalising disorders in males and females may be explained by a number of artefactual determinants or genuine explanatory factors (Grant & Weissman, 2007). These could include a range of factors such as response bias, differential service utilisation rates, sex-biased diagnostic criteria, and biological, sociocultural and psychosocial factors just to name a few (Eaton et al., 2012; Grant & Weissman, 2007). As such, it is difficult to judge whether gender/sex differences are in fact differential responses to trauma or are mediated by another independent variable not yet explored.

To add to the complicated picture of gender differences, there are those studies which have found evidence to support sides of the debate across different ELA types. This is demonstrated by Keyes et al., (2012) who found sexual abuse was related to both internalising and externalising dimensions in both males and females, while emotional abuse was related to both dimensions in females but only internalising in male, and physical abuse was associated only with internalising in females and externalising in males. These findings are in accordance with earlier findings by McGee et al., (1997) who found boys demonstrated higher rates of externalising and aggressive behaviours in association to their physical abuse, while physical abuse was a better predictor of internalising symptoms for girls. Regarding substance use in individuals, a small difference in gender was observed when it came to risk

of alcohol problem drinking; the effect of physical abuse presented stronger risk among males and neglect (unspecified) presented increased risk for females (Norman et al., 2012). It is apparent that a clear consensus regarding the gender differences across the internalising and externalising dimensions has not yet been met.

Dose-Response Relationship

As noted previously, ELAs are often co-occurring, and researchers widely acknowledge the existence of a dose-response relationship between experiences of trauma and the prevalence and severity of negative outcomes (Afifi et al., 2008; Carr et al., 2013; Felitti et al 1998). The more cumulative adverse events, or types of ELAs that an individual has been exposed to, the higher the chance of subsequent mental health problems, and the more severe the impact upon their mental health will be (Afifi et al., 2008; Carr et al., 2013; Curran et al., 2013; March-Llanes et al., 2017). Simply stated, "the more stress, the more maladjustment" (Frojd et al., 2009, p. 79). While this finding has been replicated in many studies, it should be noted that some researchers investigating the relationship between CM and internalising and externalising, reported finding evidence of a dose-response relationship for only some forms of CM (Norman et al., 2012). Ney et al., (1994) suggest that specific effects may result from the specific combination of ELAs also, providing the example from their findings that the negative impact of abuse appears to be more extensive when abuse is preceded by neglect.

ELA Focus

While the scope of traumatic or adverse events encompasses a variety of intense events, ranging from interpersonal interactions such as interpersonal violence, to natural disasters and accidents such as car-crashes, our research is primarily concerned with a subset of adverse experiences as outlined in the ACE studies (Kan, 2019). This subset is dominantly related to the interpersonal experiences of an individual in their home environment during

the first 18 years of their life and will be referred to throughout this study as ELAs (Felitti et al., 1998). These being; physical, psychological and sexual abuse, physical and emotional neglect, witnessing domestic violence, and witnessing any of the following in a primary caregiver or significant adult figure; mental illness, and attempted or successful suicide. The reason for primarily focusing on this subset of experiences is that, based on the literature reviewed, these are the experiences which previous research has predominantly focused on.

A Case for Internalising and Externalising Dimensions

Research and discussion have been conducted on the unique impact of specific experiences of childhood trauma, or the impact of ELAs on specific internalising and externalising disorders. While some researchers have found evidence that demonstrated differential effects, there are a number of researchers who suggest that the associations between ELA and psychopathology is better understood through the latent internalising and externalising dimensions as opposed to specific disorders (Curran et al., 2018; Keyes et al., 2012). Inconsistencies within the literature do exist regarding the effect of specific experiences of ELAs and specific outcomes. Despite this, overall evidence from numerous studies demonstrates the strong positive association between ELA and subsequent internalising and externalising symptoms and disorders. Accordingly, this study takes focus on the overall dimensions of internalising and externalising as opposed to specific disorders.

Aims and Justification

Young people's exposure and experiences of parental externalising, as well as ELA, can result in serious negative and lasting consequences for individuals and society. It is important to gain a clearer understanding of these impacts. As such, the aim of this project is to gain further understanding of the relationship between parental externalising and internalising and externalising as mediated by ELA; that is investigating whether ELA

statistically accounts for the relationship between our predictor variable, parental externalising, and our dependent variables, internalising and externalising in offspring.

Vachon et al (2015a) note that few studies in this area have directly tested sex as a moderator, and those that have, report inconsistent results. Thus, our second aim is to add to the literature by including a formal comparison of sex in order to gain a clearer understanding of differential effects of parent and offspring sex. This can be thought of as a similar method to including sex as a moderator. Achieving a better understanding of whether parent to child transmission of psychopathology differs by parent or offspring sex is needed to better understand factors which may contribute to the development of disorders, as well as improvement of intervention methods. To do this we used data collected as part of the National Epidemiological Survey on Alcohol and Related Conditions (NESARC) and tested a mediation model using Structural Equation Modelling. The large sample size allowed maternal and paternal effects to be calculated separately. Additionally, as we were interested in the sex-of-participant effect; whether males and females were impacted by maternal and paternal externalising, and ELAs in similar or different ways, analysis was run by group. This allowed us to compare the results of males and females.

Continued effort to expand our understanding of these associations will provide much needed information and evidence to promote the need for trauma-informed health and welfare approaches. Additionally, these findings can be expected to have treatment implications which will provide utility in informing the development of early intervention and prevention programs targeting identifiable risk factors for the development of internalising and externalising disorders. Understanding which associations are the strongest between parental externalising, ELA, and internalising and externalising, provides information on where to allocate time and resources, in order to maximise the impact of intervention, prevention and treatment. Better understanding the impact of sex also provides

much needed information on whether sex informed approaches are vital to successful outcomes, or whether intervention, prevention and treatment can be valid and reliable for males and females alike without accounting for sex differences.

We hypothesised ELA would positively account for the relationship between parental externalising and internalising and externalising; that is, we expect to find a significant indirect effect. Secondly, we hypothesised that there would be no significant difference overall in the impact of parental externalising between mothers and fathers, wherein both mothers and fathers alike significantly affected both male and female offspring with their externalising behaviour. In regards to this hypothesis we expect our results will demonstrate a significant positive direct effect between both Maternal and Paternal Externalising and Internalising and Externalising in offspring. Lastly, we expected to see sex-specific effects wherein maternal externalising is related to a higher risk of internalising and externalising disorders for female offspring than male offspring, and paternal externalising results in a higher risk of internalising and externalising disorders for male offspring than female offspring.

Methods

Study Design/Sample

We used structural equation modelling to test a hypothesised mediation model associating maternal and paternal externalising, and internalising and externalising in offspring, mediated by early life adversities while also conducting a sex comparison. Data for this project was collected in wave three of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC); a longitudinal survey conducted by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) which collected information from respondents regarding alcohol and drug use and disorder, related risk factors and associated mental and physical disabilities (NIAAA, n.da).

The complete NESARC-III dataset includes 36,309 participants from a nationally representative sample of civilian, non-institutionalised adults, aged 18 years or older, living in the United States of America (50 states as well as the District of Columbia) (Grant et al., n.d.) Participants were randomly selected using multistage probability sampling. Adults identifying as Black, Asian or Hispanic were sampled at higher rates than the remaining population to ensure reliable estimates of these groups.

Some data for our variables of interest were not available for unknown reasons. In order to include only participants who had complete data for the variables of interest, *n*=9,581 participants were excluded from this study (Figure 1). A remaining total of *N*=26,728 participants were selected to be included in analysis. Sex and age distribution of participants are as displayed in Table 1. As data was collected regarding participant sex, not gender, our analysis refers exclusively to sex.

In an effort to replicate our findings, as suggested by Pohlmann (2004), we randomly split our sample into two subsamples of 13,364 participants each. These are referred to as Group One and Group Two. We then estimated the model twice, comparing the results.

Group one comprised of 7,597 females and 5,767 males with a mean age of 46 (S.D= 17.4 years), group two comprised of 7,357 females and 6,007 males with a mean age of 45. (S.D= 17.4 years) (Table 1). For a full table of statistical descriptive information for the full sample, and for Group One and Two see Appendix A Tables A2, A3, A4 and A5.

Figure 1:

Process of participant selection

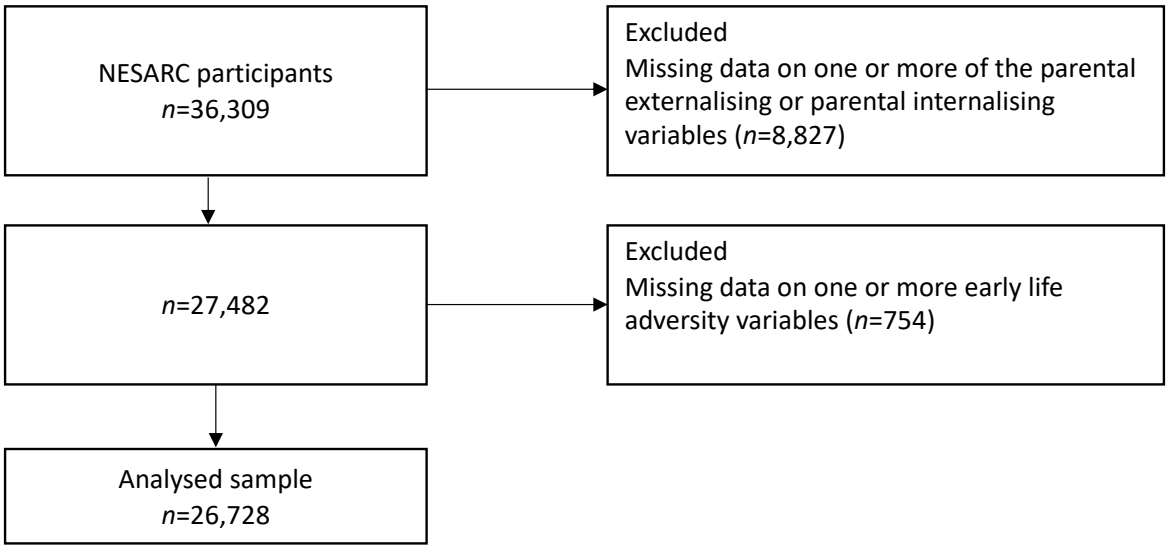


Table 1.

Descriptive statistics for the distribution of sex and age in selected participants (N=26,728)

	Number (*n)*	Percentage (%)	Mean Age (years)	Age SD (years)	Age rage (year)
Male	*n* = 11,774	44.05%	45.0	17.41	18-90
Female	*n* = 14,954	55.95%	45.8	17.41	18-90
Total sample	*n* = 26,728	100%	45.4	17.41	18-90

Measures

Alcohol Use Disorder and Associated Disabilities Interview Schedule- 5 (AUDADIS-5)

The NESARC-III used a computer-assisted diagnostic interview schedule; the AUDADIS-5 (Grant et al., n.d). The following sections included in the AUDADIS-5 used in this project are; Section 1-Background Information; Section 2A- Alcohol Consumption; Section 2B- Alcohol Experiences; Sections 2C and 3D respectively- Alcohol and Medicine/Drug Treatment Utilization; Sections 2D, 3E, 4C, 11B, and 15A- Family History items; Section 3A- Tobacco and Nicotine Use; Section 3B- Medicine and Drug Use; Section 3C- Medicine and Drug Experiences; Sections 4A and 4B respectively- Low Mood I and II; Section 6- Anxiety Panic Disorder; Section 6a- Specific Anxiety Agoraphobia Section 7- Social Situations/Social Anxiety; Section 8- Specific Situations/Specific Phobia; Section 9- General Anxiety GAD; Section 11A- Behaviour; and Section 13- Background Section III.

Mental Health

The AUDADIS-5 asks symptom-level questions, operationalising The Diagnostic and Statistical Manual of Mental Disorders, Fifth edition (DMS-5) in order to make accurate clinical diagnoses for the following internalising disorders; Major Depressive Disorder, Dysthymia, Specific Phobia, Social Phobia, Panic Disorder, Generalised Anxiety Disorder, and Post-traumatic Stress Disorder (NIAAA, 2014). Similarly, the DSM-5 was operationalised to make diagnoses for the externalising disorders; Substance Use Disorders for the following substances; alcohol, sedatives, cannabis, opioids, cocaine, stimulants, hallucinogens, inhalants/solvents, club drugs, heroin, and other drugs, and Antisocial Personality Disorder (NIAAA, 2014).

Two diagnoses appear in the NESARC-III data set for a number of mood and anxiety disorders (NIAAA, 2014). In this project the hierarchical diagnoses were utilised, with

specific mood and anxiety disorders which are the result of either a general medical condition or are substance-induced being excluded.

Parental Externalising

Clinical diagnoses of parental substance use disorders were not possible using the AUDADIS-5. Instead, participants were asked whether, in their judgement, their blood or natural fathers and/or mothers had problems with drugs or alcohol at any time in their life. Problematic use was defined to participants as, "a person who has physical or emotional problems because of drug/alcohol use; problems with a spouse, family or friends because of drug/alcohol use; problems at work or school because of drug or alcohol use; problems because of driving under the influence, or a person who seems to spend a lot of time using drug/alcohol or getting over their bad aftereffects." (NIAAA, n.db, p.1; NIAAA, n.dc, p.1). If participants responded 'yes' for either parent, that parent was considered to have a substance use issue.

Insufficient data was available to diagnose parents with personality disorders. Participants were asked whether, in their judgement, their blood or natural father/mother had behaviour problems at any time. Behavioural problems were defined to participants as 'being cruel to people or animals, fighting or destroying property, trouble keeping a job or paying bills, being impulsive, reckless or not planning ahead, lying or conning people or getting arrested. These people do not seem to care if they hurt others and often have problems at an early age such as truancy, staying out all night or running away" (NIAAA, n.dd, p.1). If participants responded 'yes' to the above question for either parent, that parent was classified as having antisocial behaviour.

Early life adversities

Survey questions relating to early life adversities (physical abuse, physical neglect, emotional abuse, emotional neglect, sexual abuse, witnessing violence and environmental adversity) were based on the occurrence of traumatic events within the respondent's household during the first 18 years of their lives (NIAAA, n.de). The occurrence of each experience was measured in a scale of 'never', 'almost never', 'sometimes', 'fairly often', or 'very often'. Six of the early life adversity questions were measured by yes/no responses (see Appendix B, Table B1).

Procedure

Data collection

Data was gathered through face-to-face interviews carried out by trained staff who visited sampled addresses to select and interview participants (Grant et al., n.d). The first two waves of the NESARC data collection were conducted in 2001-2002 and 2004-2005 respectively. All data included in this analysis was taken from wave three, conducted in April 2012 through to June 2013. Face-to-face interviews were conducted once official informed consent to participate in the NESAR-III study was documented. Consenting participants were then asked questions regarding background, lifestyle, drinking practices, mood, anxiety, behaviour, personality, and medical conditions; saliva samples were also collected from consenting participants. Two incentive payments of $45 were made to each participant who completed the interview. Interview data was electronically transmitted to a home office daily, while saliva samples were sent twice a week via FedEx.

Missing Data

Potential impacts of item nonresponse in the NESARC-III were corrected by the NIAAA using imputation. The process for imputing values for missing, or inconsistent data

varied by survey. Variables were imputed using both assignment; in which relatable information is available on the same person record and analysts are confident about assigning values to items missing data, and allocation; in which the value for missing or inconsistent items cannot be derived from the same person record and is taken from other respondents with similar characteristics. A detailed description of the imputation process and rates can be found in the National Epidemiologic Survey on Alcohol and Related Conditions-III (NESARC-III)- Data Notes (NIAAA, 2014).

Data Access

To access the data collected in the NESARC-III we completed a formal Data Use Agreement (in cooperation with University of Otago) and provided a brief description of our research project and proof of approval by the Human Ethics Committee of Victoria University of Wellington as well as the University of Otago. Upon approval from the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the datum was made available to us to download in a secure and encrypted format.

Ethics

Full ethical approval for the NESARC-III research protocol and informed consent procedure was given by the Westat Institutional Review Boards and the Combined Neuroscience Institutional Review Board of the National Institutes of Health (NIAAA, 2014). A detailed description of the NESARC data collection method and process are outlined in the National Epidemiologic Survey on Alcohol and Related Conditions-III (NESARC-III)- Source and Accuracy Statement (Grant et al., n.d).

Full ethical approval for the use and analysis of the NESARC-III data in this study was given by the Human Ethics Committees of Victoria University of Wellington, and the University of Otago.

Analysis

Selecting and Recoding Variables

Analysis for this project was run using the statistical software RStudio (Version 1.3; RStudio) predominantly using the 'lavaan' package. 66 variables of interest were included from the NESARC dataset. 26 variables regarding experiences of early life adversity were categorised into six subcategories of ELA, these were; emotional abuse (three variables), emotional neglect (five variables), physical abuse (two variables), physical neglect (five variables), sexual abuse (four variables), witnessed violence (four variables), and adverse environmental experiences (three variables).

Variables in the emotional neglect category were reverse coded. Variables in the categories of physical, sexual and emotional abuse, emotional and physical neglect, and witnessed violence were measured on a Likert scale of 1-5, with 1 representing no experience of a specified early life adversity. These 23 variables were recoded into Likert scales of 0-4 with non-experience being represented by 0. Variables in the adverse environmental experiences were coded as 1=yes (experienced) and 2=no (not experienced). These three variables, along with a further ten variables regarding the presence of maternal and paternal alcohol use problems, drug use problems, antisocial behaviours, experiences of anxiety disorders and experiences of mood disorders were recoded to 0=no (not experienced/false) and 1=yes (experienced/true).

Computing ELA Category Variables

New ELA category variables were then created for those categories measured by Likert scales by calculating the mean of variables making up each category, with the new variables representing the intensity of experienced adversity. A new variable was computed for the final ELA category measured by yes/no responses by calculating the sum of variables

making up the category. The sum variable represents the number of different adverse environmental situations a participant had experienced, as opposed to intensity of the experienced adversity.

Testing for Group Differences

To test for group differences in frequency and means of our variables of interest we ran chi-square analyses on our categorical variables and independent samples *t*-tests on our continuous variables. Analyses were run on the full group of participants twice, once comparing male and female participants and a second time comparing Groups One and Two.

Model Description (Figure 2).

The latent variable ELA was indicated by the seven ELA categories outlined above. Latent variables for maternal and paternal externalising were indicated by maternal problem drug use, maternal problem alcohol use, and maternal antisocial behaviour, and paternal problem drug use, paternal problem alcohol use and paternal antisocial behaviour respectively. Maternal and paternal internalising were indicated by maternal anxiety, maternal mood disorder, and paternal anxiety and paternal mood disorder respectively. The maternal and paternal internalising variables were included in analysis as control variables for the model.

Internalising variables; MDD, Dysthymia, Social Phobia, Specific Phobia, GAD, PTSD, and Panic Disorder were used to indicate the latent variable Internalising. Due to low numbers of diagnoses for a number of illicit drug use disorders, nine substance use disorder variables; sedative use disorder, opioid use disorder, club drug use disorder, stimulant use disorder, inhalant/solvent use disorder, hallucinogen use disorder, cocaine use disorder, heroin use disorder and other drug use disorder were aggregated to create one drug use disorder variable where a diagnosis of any of the aforementioned drug use disorders

constituted a yes on the drug use disorder variable. The variables Drug Use Disorder, Alcohol Use Disorder, Cannabis Use Disorder, Tobacco Use Disorder and Antisocial Personality Disorder were then used to indicate the latent variable Externalising.

Statistical Analysis

Testing For Weak Measurement Invariance. For our analysis, we grouped our data by sex into 'males' and 'females' in order to investigate sex-specific effects and compare group results. To be able to make valid group comparisons, we needed to ensure that our latent constructs were being measured with the same factorial structure across groups. That is, factor loadings of the indicators of the latent constructs are equivalent across men and women. Additionally, to be able to run a sex comparison, we needed to show that the latent dimensions are invariant across sex already. Firstly we estimated a multigroup confirmatory factor analysis (CFA) model, specifying our model with the factor group 'sex' in which parameters were freely estimated across both groups. To test meaurement invariance we used the R function 'measurementinvariance' which estimated and compared several versions of constaints. We then compared the model fit, and change in CFI as this is the only test that has consistent validity (Sellbom & Tellegen, 2019), if it has not become worse then that provides evidence of weak variance.

Structural Equation Modelling. We estimated a Structural Equation Model (using lavaan package) to test our hypothesised mediation model with a group factor (see Appendix for full analysis R code). There are a number of fit indices, as recommended by Schreiber et al., (2006), which are commonly used by researchers which we selected to indicate model fitness in assessing our model; these being Comparative Fit Index (CFI), Non-Normed Fit Index (also known as the Tucker-Lewis Index, TLI), and The Root Mean Square Error of Approximation (RMSEA). Criteria for acceptance of a model are TLI $\geq .95$, CFI $\geq .95$, and RMSEA $< .06$ (Schreiber et al., 2006). In order to test whether our model fitness changed

when direct effects were constrained to be the same for males and females we tested a unconstrained and a constrained version of our model on both Group One and Group Two, comparing the resulting CFI, TLI and RMSEA indices. In our unconstrained model factor, loadings for our latent variables were constrained to be equal across groups (sex), while in our constrained model both factor loadings and regressions were constrained.

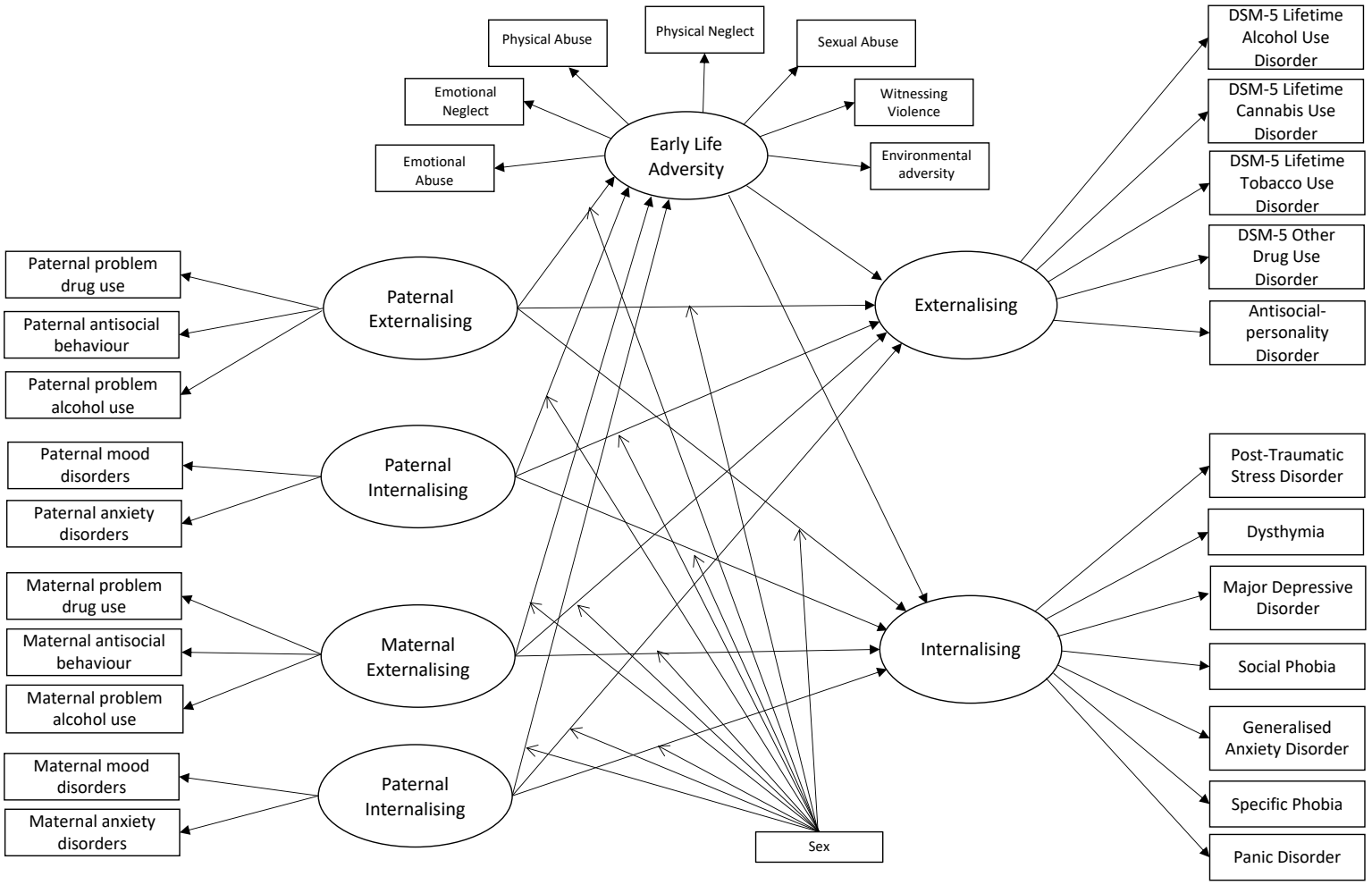
We chose maximum likelihood estimation (MLM) as our estimation method. As we had included eight pathways of interest (see Figure 2) in our model we adjusted our alpha-level for multiple testing by dividing the *p* threshold ($p < .05$) by eight resulting in a significance criterion of $p < .00625$.

Standardised values are reported for the direct, indirect and total effects of our model for both Group One and Two.

Effect of Sex. Firstly we conducted a Chi-square test of differences to determine whether there was a significant change in model fit. Secondly we used Chi-Square tests for releasing single constraints, equivalent to modification indices to determine which specific paths were significantly moderated by sex.

Figure 2.

Hypothesised SEM model



Results

Group Differences

Results from chi-square tests and independent sample t-tests indicate that the only variables that do not differ significantly in frequency or mean between males and females are emotional abuse and emotional neglect (see Appendix A, Tables A1 and A2). Results from chi-square tests and independent sample t-tests comparing frequencies and means of variables between Group One and Group Two indicate that the only variable that the Groups significantly differ on is sex (see Appendix A, Tables A3, and A4).

Zero-order Correlation

Cohen's convention was used when interpreting correlation analysis. This can be characterised as weak correlation (r= .1 to <.3), medium correlation (r >.3 to <.5) and strong correlation (r = >.5) (Rosnow et al., 1992). Correlation analysis of the full sample of participants, indicated that all included variables were significantly correlated with each another at a significance level of p<.01 (see Appendix A, Table A5). Correlation analyses separately for male and female participants of the full sample indicated that all variables were significantly related to each other at a significance of p<.01; the highest correlations were found for physical abuse and emotional abuse (r = .78 and r =.80 respectively) (see Appendix A, Table A6).

Within the Group One subset all variables were found to be significantly correlated at p<.01, and again emotional abuse and physical abuse has the strongest correlation (r =.79), the same was found for the Group Two subset (r =.79) (see Appendix A, Table A7 and Table A8). Within males in Group One, paternal drug use and specific phobia were not significantly correlated, nor was paternal anxiety and maternal alcohol use (Table 2). In males in Group Two, paternal drug use and dysthymia were not significantly correlated, nor was environmental early life adversities and social phobia (see Appendix A, Table A9) All other variables were significantly correlated at p<.05 (Table 2; see Appendix A, Table A9).

Within females in Group One, paternal drug use and dysthymia were not significantly correlated (Table 2), in females in Group Two paternal drug use and social phobia were not significantly correlated (see Appendix A, Table A9) All other variables were significantly correlated at $p<.05$ and the highest correlations were found between physical abuse and emotional abuse in both Group One males (r =.77) and females (r =.81) as well as Group Two males (r =.78) and females (r =.79) (Table 2; see Appendix A, Table A9).

Table 2.

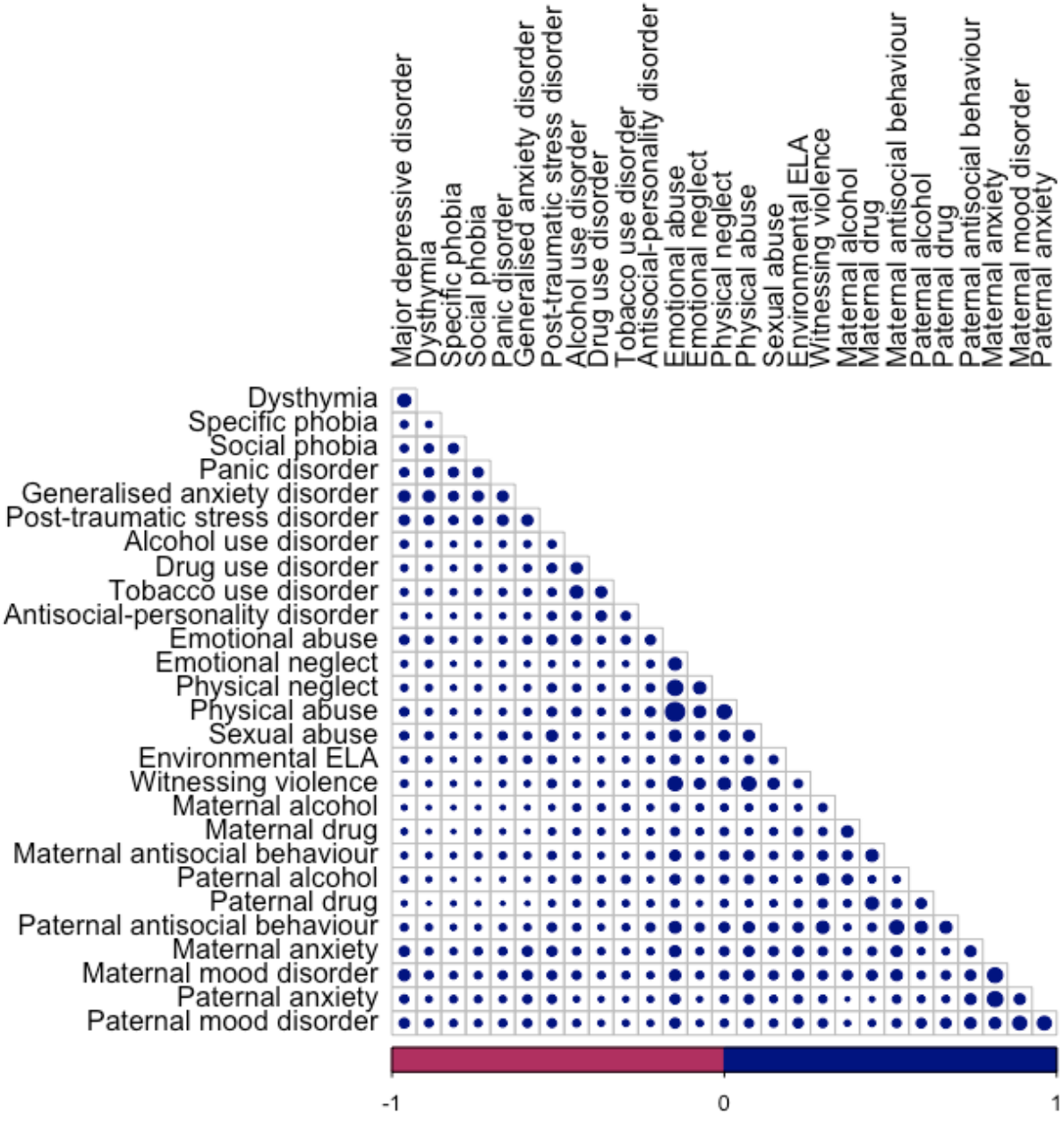
Correlations for Group One male (lower, n=5,767) and female (upper, n=7,597) participants

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1. Major depressive disorder		.32**	.12**	.13**	.17**	.24**	.22**	.17**	.10**	.13**	.08**	.19**	.10**	.11**	.17**	.13**	.10**	.10**	.07**	.07**	.09**	.10**	.06**	.11**	.21**	.27**	.16**	.19**
2. Dysthymia	.35**		.08**	.15**	.18**	.26**	.18**	.10**	.07**	.11**	.05**	.15**	.12**	.13**	.14**	.12**	.07**	.07**	.05**	.04**	.08**	.07**	0.02	.10**	.13**	.16**	.09**	.14**
3. Specific phobia	.10**	.08**		.18**	.20**	.18**	.14**	.09**	.08**	.11**	.09**	.11**	.05**	.08**	.09**	.08**	.06**	.06**	.04**	.04**	.08**	.05**	.07**	.08**	.10**	.10**	.12**	.10**
4. Social phobia	.13**	.18**	.23**		.19**	.22**	.16**	.09**	.12**	.11**	.07**	.12**	.08**	.11**	.10**	.12**	.08**	.07**	.06**	.07**	.12**	.03*	.05**	.07**	.13**	.11**	.11**	.11**
5. Panic disorder	.12**	.16**	.15**	.23**		.23**	.24**	.13**	.14**	.15**	.08**	.18**	.11**	.12**	.14**	.12**	.12**	.09**	.05**	.07**	.13**	.07**	.03**	.12**	.14**	.16**	.11**	.14**
6. Generalised anxiety disorder	.25**	.24**	.19**	.22**	.22**		.25**	.13**	.15**	.14**	.10**	.17**	.09**	.13**	.14**	.11**	.12**	.07**	.05**	.03**	.10**	.06**	.05**	.10**	.22**	.20**	.16**	.15**
7. Post-traumatic stress disorder	.18**	.15**	.18**	.15**	.19**	.21**		.17**	.19**	.18**	.15**	.25**	.11**	.22**	.23**	.28**	.14**	.16**	.09**	.11**	.16**	.10**	.09**	.17**	.21**	.18**	.15**	.15**
8. Alcohol use disorder	.13**	.09**	.09**	.09**	.09**	.10**	.11**		.25**	.32**	.14**	.17**	.07**	.10**	.13**	.11**	.08**	.08**	.15**	.13**	.14**	.12**	.11**	.12**	.16**	.19**	.08**	.13**
9. Drug use disorder	.12**	.12**	.08**	.09**	.09**	.09**	.16**	.22**		.24**	.15**	.13**	.09**	.11**	.12**	.12**	.06**	.06**	.10**	.09**	.08**	.10**	.11**	.07**	.11**	.12**	.06**	.08**
10. Tobacco use disorder	.12**	.09**	.09**	.08**	.09**	.10**	.09**	.30**	.24**		.12**	.16**	.08**	.10**	.15**	.13**	.10**	.11**	.12**	.10**	.11**	.13**	.09**	.10**	.13**	.14**	.08**	.10**
11. Antisocial-personality disorder	.12**	.09**	.06**	.10**	.10**	.11**	.17**	.15**	.25**	.18**		.21**	.11**	.17**	.19**	.19**	.10**	.12**	.10**	.11**	.15**	.06**	.09**	.14**	.12**	.10**	.09**	.09**
12. Emotional abuse	.18**	.14**	.11**	.11**	.09**	.14**	.19**	.17**	.15**	.14**	.27**		.39**	.57**	.81**	.32**	.21**	.44**	.17**	.20**	.32**	.21**	.15**	.33**	.27**	.27**	.21**	.21**
13. Emotional neglect	.09**	.11**	.05**	.07**	.03*	.05**	.05**	.05**	.09**	.03*	.12**	.28**		.38**	.36**	.24**	.12**	.21**	.12**	.12**	.17**	.13**	.07**	.16**	.11**	.13**	.08**	.11**
14. Physical neglect	.10**	.09**	.06**	.08**	.06**	.08**	.14**	.09**	.11**	.09**	.19**	.52**	.28**		.53**	.34**	.16**	.37**	.17**	.20**	.25**	.16**	.14**	.23**	.18**	.18**	.13**	.13**
15. Physical abuse	.15**	.11**	.09**	.07**	.08**	.10**	.16**	.14**	.14**	.16**	.27**	.77**	.24**	.47**		.32**	.16**	.42**	.13**	.13**	.27**	.17**	.12**	.26**	.21**	.23**	.17**	.17**
16. Sexual abuse	.09**	.10**	.06**	.04**	.09**	.08**	.17**	.06**	.08**	.06**	.13**	.20**	.09**	.24**	.19**		.15**	.27**	.12**	.13**	.16**	.13**	.12**	.18**	.17**	.15**	.11**	.13**
17. Environmental ELA	.07**	.07**	.05**	.06**	.08**	.09**	.08**	.05**	.09**	.06**	.11**	.14**	.12**	.12**	.13**	.07**		.12**	.08**	.15**	.19**	.07**	.12**	.17**	.19**	.22**	.13**	.17**
18. Witnessing violence	.11**	.05**	.07**	.03*	.08**	.06**	.14**	.07**	.09**	.10**	.17**	.41**	.18**	.35**	.42**	.21**	.14**		.14**	.14**	.19**	.24**	.16**	.31**	.15**	.14**	.11**	.12**
19. Maternal alcohol	.04**	.03*	.04**	.03*	.03*	.05**	.03*	.08**	.11**	.11**	.08**	.14**	.10**	.10**	.11**	.06**	.07**	.11**		.28**	.20**	.20**	.12**	.10**	.14**	.19**	.05**	.09**
20. Maternal drug	.09**	.05**	.04**	.07**	.04**	.08**	.06**	.08**	.11**	.09**	.12**	.14**	.08**	.15**	.13**	.05**	.15**	.11**	.22**		.31**	.12**	.35**	.15**	.15**	.21**	.06**	.11**
21. Maternal antisocial behaviour	.08**	.07**	.06**	.08**	.04**	.09**	.09**	.08**	.09**	.07**	.14**	.22**	.13**	.14**	.17**	.08**	.15**	.14**	.17**	.25**		.10**	.19**	.38**	.25**	.25**	.15**	.15**
22. Paternal alcohol	.10**	.05**	.03*	.05**	.04**	.06**	.08**	.17**	.12**	.16**	.11**	.21**	.12**	.16**	.15**	.08**	.07**	.24**	.21**	.09**	.10**		.21**	.28**	.11**	.15**	.11**	.20**
23. Paternal drug	.07**	.05**	0.02	.05**	.03**	.04**	.09**	.09**	.09**	.10**	.12**	.13**	.05**	.13**	.11**	.05**	.12**	.15**	.11**	.27**	.14**	.21**		.29**	.11**	.16**	.11**	.22**
24. Paternal antisocial behaviour	.13**	.09**	.05**	.09**	.07**	.11**	.14**	.14**	.11**	.12**	.19**	.31**	.14**	.23**	.25**	.12**	.16**	.30**	.09**	.13**	.39**	.29**	.26**		.24**	.23**	.25**	.25**
25. Maternal anxiety	.19**	.10**	.10**	.12**	.11**	.17**	.16**	.11**	.10**	.07**	.11**	.24**	.08**	.15**	.19**	.10**	.15**	.14**	.10**	.15**	.19**	.10**	.10**	.22**		.45**	.45**	.25**
26. Maternal mood disorder	.22**	.12**	.12**	.12**	.11**	.16**	.13**	.14**	.12**	.11**	.11**	.22**	.08**	.14**	.18**	.07**	.18**	.13**	.18**	.18**	.23**	.10**	.15**	.20**	.43**		.23**	.36**
27. Paternal anxiety	.13**	.08**	.10**	.11**	.09**	.15**	.15**	.09**	.07**	.07**	.09**	.17**	.03**	.12**	.15**	.05**	.11**	.10**	0.02	.06**	.11**	.10**	.11**	.22**	.48**	.23**		.38**
28. Paternal mood disorder	.21**	.14**	.12**	.12**	.11**	.15**	.17**	.12**	.09**	.08**	.10**	.20**	.07**	.13**	.14**	.07**	.15**	.13**	.06**	.07**	.16**	.18**	.19**	.24**	.28**	.40**	.37**	

Note. * indicates $p < .05$. ** indicates $p < .01$. Results for male participants are displayed in the lower left half of the table, results for female participants are shown in the upper right half of the table.

Figure 3.

Correlation plot of Group One participants (n=13,364). Blue dots indicate positive correlation, red dots indicate negative correlation. Dot size indicates strength of correlation; the larger the dot the closer to a correlation of 1



Measurement Invariance

Results from our multi-group CFA meaurement invariance tests are displayed in Table 3. The change in CFI from the constrained model to the unconstrained model in Group One was 0.009 (Table 3), this meets the threshold (0.01) indicating the model is weakly invariant; therefore meeting the assumption that factor loadings of the indicators of the latent construct are equivalent across men and women. Additionally, as latent dimensions are invarience across sex already we are able to run a sex comparison. The change in CFI in Group Two however was 0.01, also meeting the threshold for weak invariance (Table 3).

Table 3.

Results of multi-group CFA measurement invariance testing for participants in Group One and Group Two

	Group One	Group Two
Chi-square	3938.800	4071.000
Chi-square Df	504.000	518.000
Comparative Fit Index (CFI)	0.940	0.940
RMSEA	0.032	0.032
Change in CFI	0.009	0.010

Structural Equation Modelling

Overall Model Fitness

Model fit indicies indicated that the model provided a good fit to the data (Table 4). As our hypothesised model appears to be a good fit for the data we did not conduct post-hoc modifications.

Table 4.

Model fit indices comparison of free running models and constrained models and results of a Chi-Squared test of differences for participant in Groups One and Two

	Group One Unconstrained Model	Group One Constrained Model	Group Two Unconstrained Model	Group Two Constrained Model
Robust Compative Fit Index (CFI)	0.922	0.921	0.921	0.918
Robust Tucker-Lewis Index (TLI)	0.91	0.911	0.909	0.908
Robust RMSEA	0.034	0.034	0.035	0.035
Chi-square	6925.1	7025.5	7141.6	7334.8
Chi-square Df	708	722	708	722
Chi-square diff	49.652		99.402	

Weightings of Observed Variables on Latent Dimensions

Emotional abuse had the highest estimate for the latent dimension Early Life Adversity in both Group One and Group Two males and females (Figures 4 and 5, see Appendix A, Figures A3 and A4).

In Group One males and Group Two males and females DSM-5 Drug Use Disorder has the strongest association with Externalising, while DSM-5 Cannabis Use Disorder (CUD) had the strongest association in Group One females (Figures 4 and 5, see Appendix A, Figures A3 and A4). Generalised Anxiety Disorder had the highest estimate with the latent dimension Internalising across males and females in both groups (Figures 4 and 5, see Appendix A, Figures A3 and A4).

Similarly Paternal Antisocial Behaviour had the highest factor loading on Externalising in Group One and Two males and females for Externalising (Figures 4 and 5, see Appendix A, Figures A3 and A4). Lastly, Maternal Antisocial Behaviour had the estimate for the latent dimension Maternal Externalising in Group One males and females, and Group Two females, while Maternal Problematic Alcohol use had the highest estimate for Group Two males (Figures 4 and 5, see Appendix A, Figures A3 and A4).

Hypothesis One, ELA as a Significant Mediator

We hypothesised Early Life Adversity would mediate the relationship between both Paternal and Maternal Externalising and latent Externalising and Internalising dimensions in offspring; that is ELA would account for the relationship between Parental Externalising, and Internalising and Externalising in male and female offspring.

A significant indirect effect (standardised indirect coefficient .084, p=.001) was found between Paternal Externalising and Internalising mediated by ELA in male participants (Table 7). This result was replicated in Group Two males (see Appendix A, Table A12). Results also demonstrated a significant indirect effect (standardised indirect coefficient .103,

p=.001) between Maternal Externalising and Internalising mediated by ELA in male offspring which was again replicated in Group Two males (Table 7, see Appendix A, Table A12).

While a significant direct effect did not exist between either Paternal Externalising and Externalising in females, or ELA and Externalising in females, results did indicate a significant total effect (standardised total coefficient .403, *p*=.004) of Paternal Externalising and ELA on Externalising in female offspring. This indicates that Paternal Externalising and ELA as a combination do have a significant association with Externalising in females.

These results demonstrate mixed support for our hypotheses; that we would find a relationship between parental externalising and internalising and externalising in offspring, and that these relationships would be mediated by experiences of ELA. Results support our prediction that the relationship between Paternal Externalising and Internalising, and Maternal Externalising and Internalising is mediated by ELA, but only in male offspring. We found no evidence to support our hypothesis that ELA mediates the relationship between Paternal or Maternal Externalising and Internalising in female offspring, or Externalising in male or female offspring.

Hypothesis Two, Parental Externalising Impacts Male and Female Offspring

We hypothesised that there would be no significant difference overall in the impact of parental externalising between mothers and fathers, wherein Maternal and Paternal Externalising would have a significant impact on both male and female offspring.

Results indicate a significant direct effect (standardised coefficient .286, *p*<.001) between Paternal Externalising and ELA in Group One females, replicated in Group Two females (Figure 4, see Appendix A, Figure A3). Similarly a significant direct effect (standardised coefficient .349, *p*<.001) between Maternal Externalising and ELA was also

demonstrated in Group One females, replicated in Group Two females (Figure 4, see Appendix A, Figure A3).

ELA was found to have a significant direct association with Internalising in Group One females (standardised coefficient .295, $p<.001$) and males (standardised coefficient .271, $p<.001$) (Figures 4 and 5), a finding which was replicated in both females and males in Group Two (see Appendix A, Figures A3 and A4). However a significant direct effect between ELA and Externalising was not demonstrated (see Appendix A, Table A11).

No significant association between either Maternal or Paternal Externalising and Internalising or Externalising in offspring was found in sons or daughters. As such, results do not provide support for our prediction.

Figure 4.

Sructural equation model of Group One female participants indicating the association between latent dimensions of parental externalising and internalising, early life adversity and latent dimensions of internalising and externalising (n=7,597). Circles represent latent variables and rectangles represent observed variables

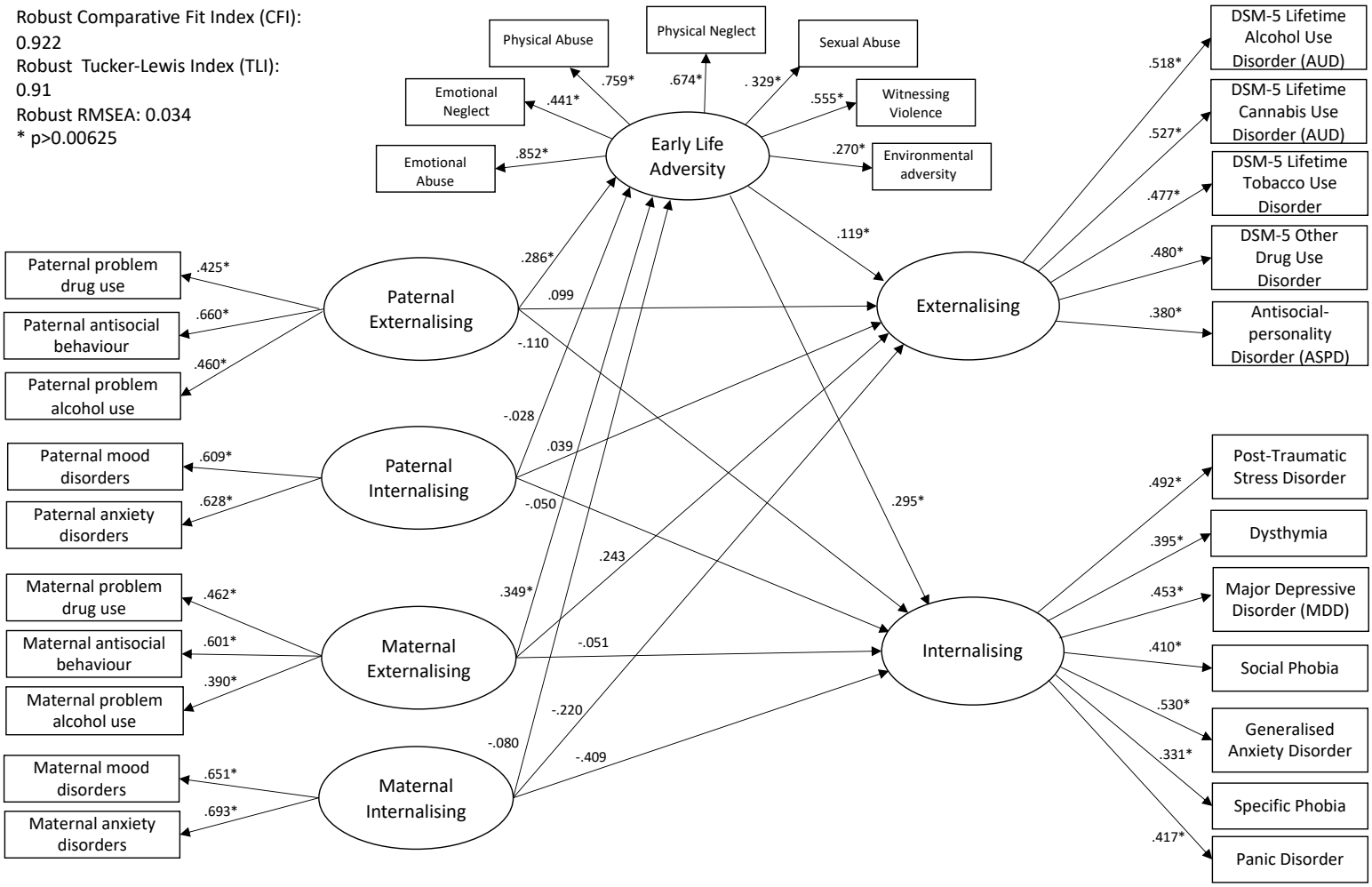


Table 5.

Indirect effects (via ELA) in the structural equation model for Group One female participants (n=7,597)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	-0.010	0.083	0.874
Maternal Internalising	-0.132	0.128	0.270
Paternal Externalising	0.170	0.072	0.015
Paternal Internalising	0.109	0.116	0.339
Externalising			
Maternal Externalising	-0.004	0.047	0.871
Maternal Internalising	-0.051	0.067	0.219
Paternal Externalising	0.065	0.045	0.022
Paternal Internalising	0.042	0.060	0.281

Table 6.

Total effects in the structural equation model for Group One female participants (n=7,597)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.150	0.254	0.451
Maternal Internalising	-0.046	0.367	0.894
Paternal Externalising	-0.012	0.162	0.941
Paternal Internalising	-0.423	0.346	0.212
Externalising			
Maternal Externalising	0.085	0.352	0.641
Maternal Internalising	0.322	0.504	0.322
Paternal Externalising	0.403	0.220	0.004
Paternal Internalising	0.228	0.462	0.446

Figure 5.

Structural equation model of Group One male particiants indicating the association between latent dimensions of parental externalising and internalising, early life adversity and latent dimensions of internalising and externalising (n=5,767). Circles represent latent variables and rectangles represent observed variables

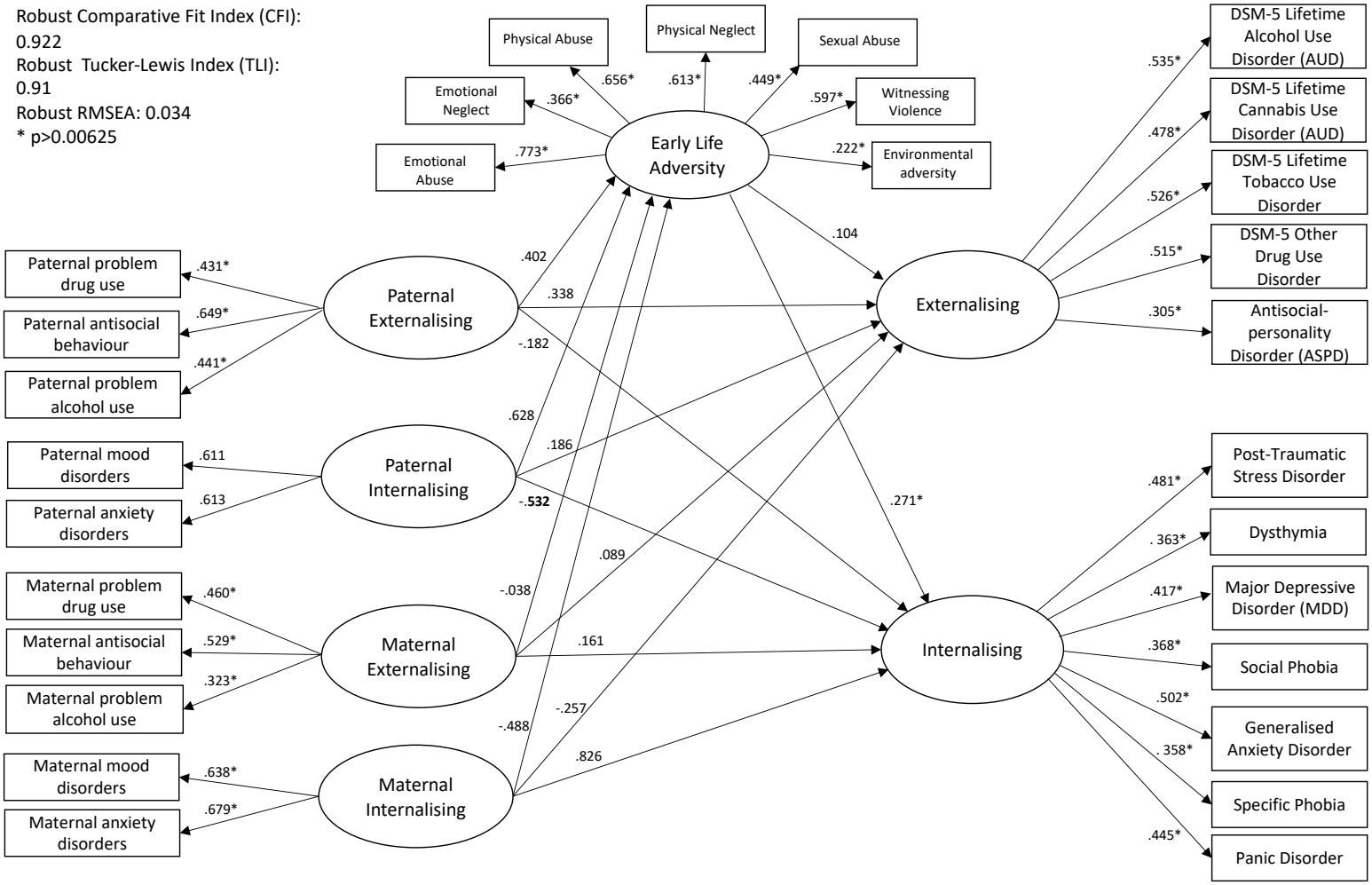


Table 7.

Indirect effects (via ELA) in the structural equation model for Group One male participants (n=5,767)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.103	0.040	0.001
Maternal Internalising	-0.024	0.036	0.405
Paternal Externalising	0.084	0.031	0.001
Paternal Internalising	-0.008	0.764	0.035
Externalising			
Maternal Externalising	0.285	0.124	0.007
Maternal Internalising	-0.200	0.131	0.073
Paternal Externalising	0.133	0.106	0.143
Paternal Internalising	0.035	0.131	0.754

Table 8.

Total effects in the structural equation model for Group One male participants (n=5,767)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.052	0.131	0.614
Maternal Internalising	-0.432	0.151	<.001
Paternal Externalising	0.035	0.113	0.696
Paternal Internalising	0.151	-0.118	0.317
Externalising			
Maternal Externalising	0.285	0.124	0.007
Maternal Internalising	-0.200	0.073	0.131
Paternal Externalising	0.133	0.106	0.143
Paternal Internalising	0.035	0.131	0.754

Hypothesis Three, Sex-Specific Interactions

We expected to see sex-specific interactions where a stronger effect would be evident for Paternal Externalising on male offspring and Maternal Externalising on female offspring.

Based comparing the direct effects in th models for males and females, there seems no significant direct associations between either Maternal or Paternal Externalising and Internalising or Externalising in male or female offspring. Additionally, the only significant mediation effects demonstrated in our results was between Paternal Externalising and Internalising in males mediated by ELA, and Maternal Externalising and Internalising in males mediated by ELA (see Appendix A, Table, A12).

As such, our results provide only partial evidence of a sex-specific interaction; while Paternal Externalising has a more stronger impact on male offspring than female offspring, Maternal Externalising does not have a stronger impact on female offspring than male offspring. It must be noted that this conclusion has been drawn on the basis of comparing the direct and indirect effects of the SEM model rather than a specific test of moderation.

Test for Effect of Sex

Results of a Chi-sqare test for difference indicated that there was a significant difference between our constrained and unconstrained model fitness indicies (Table 4). This indicates that there was an overall effect of sex on the model.

Comparing single releases of constraints with Chi-square tests demonstrated that the direct associations between the following variables were significantly moderated by sex (i.e significantly different for male and female offspring); Early Life Adversity and Paternal Externalising (X^2(1, *n= 13,364*)= 21.102, *p*<.001); Internalising and Paternal Externalising (X^2(1, *n= 13,364*)= 7.173, *p*=.007); Internalising and Paternal Internalising (X^2(1, *n= 13,364*)=12.753, *df*=1, *p*<.001); Internalising and Maternal Internalising (X^2(1, *n= 13,364*)=24.674, *df*=1, *p*<.001); Externalising and Maternal Internalising (X^2 (1, *n= 13,364*)= 4.684, *df*=1, *p*=.03); and Externalising and Early Life Adversity (X^2 (1, *n= 13,364*)=4.054, *df*=1, *p*=.044).

Discussion

This study investigated the role of Early Life Adversity (ELA) and sex of the offspring in the relationship between parental internalising and externalising and offspring symptomatology. Including ELA as a mediator of the association between parental externalising and internalising and externalising in offspring showed that for the most part, experiences of ELA better explain the development of psychopathology in offspring than parental externalising. Additionally, we aimed to replicate past findings regarding the direct effect between parental externalising, ELA, and internalising and externalising in offspring.

We found ELA does mediate the relationship between both paternal and maternal externalising and internalising in male offspring. We did not find any evidence however that ELA mediates the relationship between either maternal or paternal externalising and internalising in females or externalising in male or female offspring.

Additionally we found no direct effect between parental externalising and internalising in male or female offspring. We did however find a direct effect between both paternal externalising and experiences of ELA, and maternal externalising and experiences of ELA in female offspring. Additionally, we found a significant effect of sex on the association between paternal externalising and ELA, supporting the finding that this relationship significantly differs between male and female offspring.

While a relationship exists between ELA and internalising in male and female offspring, results do not demonstrate a direct effect between ELA and externalising in offspring of any sex.

Direct Associations of Parental Externalising and ELA With Externalising

Contrary to past research which has demonstrated many times that parental externalising confers a higher risk of offspring developing externalising behaviour, in this study we did not find a significant relationship between either maternal or paternal

externalising and externalising symptoms in male or female offspring (Besemer, 2014; Foley et al., 2001; Furtado et al., 2006; Herndon & Iacono, 2005; Kim et al., 2009; Long et al., 2018; Whitten et al., 2019). This is in direct opposition to the strong links that have been drawn between parental externalising and the development of substance use problems in offspring, as well as behaviour difficulties and/or the engagement of offending behaviour (Besemer et al., 2017; Long et al., 2018). Furthermore, though there is an abundance of studies which report a strong association between ELA and externalising symptoms across the life course, our results did not replicate this finding in females or in males (see review by Curran et al., 2018).

One explanation for this may be that in past research parental externalising has been operationalised through parental reported symptoms that were fed into diagnoses for DSM-5 disorders, i.e. AUD or ASPD, or by criminal convictions of offences committed by the parent (Besemer, 2014; Long et al., 2018). In this study, parental externalising was operationalised through offspring reporting symptoms: participants were given a list of behaviours/situations that would indicate antisocial behaviour, or problematic substance use and were asked whether their parents engaged in those problematic behaviours. Affirmative responses provided no information regarding the intensity or seriousness of the parental externalising. With this in mind, in this study parental externalising can more accurately be thought of as offspring perceived parental externalising. Consequently, it is possible that in this study we have captured a broader range of participants who report parental externalising than usual studies due to a lower threshold of parental externalising.

Our results may additionally reflect the relationship between offspring-perceived problem behaviour in their parents and their own behaviour. If offspring are engaging in externalising behaviour themselves, their own perception of what is problematic and unproblematic behaviour may be biased. Consequently their perception of parental

externalising may also be biased, leading to under-reporting of parental externalising by offspring who also exhibit externalising symptoms/behaviours.

This does not however explain the unexpected lack of evidence for a relationship between experiences of ELA and externalising. Researchers have discussed a strong link between experience of ELA and the increased risk of subsequent externalising in victims, however no such relationship was demonstrated within our sample of participants (Johnson et al., 1999; Norman et al., 2012; Zhang et al., 2019; Lijffijt et al., 2014). One possible explanation for this is that due to publication biases, other research reporting a lack of evidence for a relationship between ELA and externalising in males and females has not been published, while research which demonstrates a significant relationship has continued to be published, skewing the literature.

Direct Associations of Parental Externalising and ELA With Internalising

Our results do not demonstrate a significant association between externalising in either parent and internalising in offspring. This was again surprising when considering the results reported by past studies, but again may be a result of a lowered threshold of what constitutes parental externalising in this study compared to traditional studies (Herndon and Iacono, 2005; Long et al., 2018). However, though no significant direct effect between paternal externalising and internalising was demonstrated in either male or female offspring, there was still a small significant effect of sex on this pathway. This indicates that this association significantly differs for males and females.

The relationship demonstrated between ELA and internalising in this study are in line with previous findings which have consistently found a significant association between various forms of ELA and internalising disorders (Curran et al., 2018; Kim et al., 2003; see review by Norman et al., 2012).

The role of Maternal Externalising

Contrary to our expectations, our results indicate that maternal externalising does not have a significant direct effect with subsequent internalising in offspring; whether it be in sons or daughters. Additionally, we found that ELA did not mediate the relationship between maternal externalising and internalising in daughters; whether or not daughters have experienced ELA has no impact on the association between maternal externalising and internalising in female offspring.

There was no significant total effect of maternal externalising and ELA on internalising in females either, meaning that the impact of maternal externalising and ELA together as a combination also do not significantly increase the risk of females exhibiting internalising symptoms in adulthood compared to maternal externalising or ELA in isolation.

Conversely, though no direct effect existed between maternal externalising and internalising in sons, an association between these variables did exist when mediated by ELA. From this finding we can infer that experiences of ELA partially account for the relationship between maternal externalising and internalising in males. As with female participants, we found no significant total effect of maternal externalising and ELA in combination on internalising in males, indicating that internalising in males is not better explained by maternal externalising and ELA together than by ELA alone.

These findings only partially support our first hypothesis; that ELA would positively account for the relationship between maternal externalising and internalising. While the current evidence supports this prediction in male offspring, our results provide no evidence to support this claim for female offspring. Our results also demonstrate a lack of association between maternal externalising and internalising in offspring. In short, despite previous claims that maternal externalising presents an increased risk of externalising in offspring, our results indicate that in actuality maternal externalising appears to have no effect on the risk of

internalising in female or male offspring (Foley et al., 2001; Furtado et al., 2006; Herndon & Iacono, 2005; Kim et al., 2009). Additionally, similarly to findings by Anda et al., (2002), any apparent links between maternal externalising and internalising in offspring are better explained by offspring's experiences of ELA.

The Role of Paternal Externalising

Similarly, we found no direct effect between paternal externalising and internalising in either male or female offspring. Again these results are unexpected, and contrary to findings reported by previous studies (Foley et al., 2001; Furtado et al., 2006; Herndon & Iacono, 2005; Kim et al., 2009).

We did however find that experiences of ELA positively account for the relationship between paternal externalising and internalising in male offspring. This is, experiences of ELA positively account for the relationship between paternal externalising and internalising in male offspring. This finding is partially in line with the prediction we made in our first hypothesis; that we would find evidence of a significant mediation effect by ELA on paternal externalising and internalising in offspring. Again, this supports the claim by Anda et al., (2002) that depressive disorders among the children of parents with AUD are due to a greater likelihood of having experienced ACEs in a home with substance abusing parents.

Once more, our results indicated no evidence of a total effect, which implies that the combination of paternal externalising and ELA together does not explain internalising in male offspring better than ELA does alone. These findings demonstrate that when accounting for parental externalising in isolation from ELA, male offspring who perceive their parent to exhibit externalising symptoms may not be at a higher risk of developing internalising symptoms than offspring who do not. Instead, when an association is found between paternal externalising and internalising in either male or female offspring, it may be that experiences

of ELA better explain the association. This is also the case for male offspring of mothers who exhibit externalising behaviours.

Though no direct effect between paternal externalising and externalising in offspring was demonstrated by our results, we did find that the combination of paternal externalising and ELA together did significantly increase the risk of externalising in female offspring only. This findings was particularly unexpected for two reasons. Firstly, literature often claims that males have higher rates of externalising than females, and other research has stated that paternal externalising has a greater impact on sons than daughters (Furtado et al., 2006; Long et al., 2018). Secondly, no direct effect was found between either Paternal Externalising and Externalising in female offspring, or between ELA and Externalising in female offspring meaning that alone they do not appear to impact externalising in females. Being exposed to the combination of the two however, does have a significant effect on the risk of female offspring developing externalising symptoms. Again, these unexpected findings could be due to a bias in the perception of externalising behaviour in offspring who themselves engage in externalising behaviours, leading to under-reporting of perceived parental externalising in our participants. Though why these biases would have a greater impact on results regarding male participants then female participants remains unclear and further investigation should be taken.

Strengths

Our large sample size allowed us to randomly split our participant sample in two and run our analyses twice in order to investigate whether our results could be replicated as suggested by Pohlmann (2004). Additionally, by including both paternal and maternal externalising as well as parental internalising in one model, we were able to control for covariance between these factors. This allowed us to investigate the relationship between both maternal and paternal externalising with ELA and internalising and externalising in

offspring while controlling for internalising in either parent, and externalising in the co-parent. Consequently any associations demonstrated in our results account for the impact of externalising and internalising by the co-parent.

Another strength of our estimated model is that ELAs regarding parental externalising behaviours, such as parental substance abuse or imprisonment (antisocial behaviour) were not included in our operationalised measure of ELA. By excluding parental externalising factors from our ELA latent dimension we avoided the confounding impact these factors would have had on the hypothesised mediation model.

Limitations and Future Directions

Primary Parent/Caregiver

Particularly salient for this discussion is the idea suggested by Long et al., (2018) that a socialisation effect may exist, wherein rather than either maternal externalising or paternal externalising having a strong impact on their offspring, it is the primary parent who exerts the strongest influence. Unfortunately as part of our analysis we were unable to account for household composition, this does somewhat constrain our study in that we cannot rule this out as an influence. Ideally, in future studies, as exemplified by Long et al., (2018), data on household composition should be included in analysis in order to account for socialisation effects. This way, results will better reflect the true influence of sex. This would also allow us to investigate differences in the influence that absent versus permanent parental figures have on offspring. Providing useful information for better understanding the mechanisms by which parents influence health outcomes in their offspring.

Further to this, we have solely investigated parental externalising by biological parents. This does not necessarily reflect the realities of many families, and it is important to understand what influence other parental figures such as adoptive parents, or step-parents have on offspring. Gathering in-depth data regarding the externalising of all potential parental

figures would enrich future research by providing an even clearer, and broader picture of the relationship between parental externalising and internalising and externalising in offspring. It would also ensure that family members who may be critical to positive health outcomes in offspring are not prematurely excluded from intervention, or treatment approaches.

As we did not analyse any data on the quantity and quality of parents involvement we do not know how often either parent exerted direct influences on their children either, making it difficult to disentangle biological versus environmental risk factors associated with parental externalising. This is a common issue faced in this area of research as genetic vulnerabilities and family adversity often overlap, particularly in early life, to increase risk; a phenomenon which has been termed the passive gene-environment correlation (Yan et al., 2020). By including such information in future analysis we would be able to infer not just the strength of the relationship between variables, but also more regarding the mechanisms by which they interact.

Yan and colleagues (2020) discuss parental closeness and parent-child conflict as factors which may contribute to externalising and internalising in offspring. In one study Yan et al., (2019) report that father-offspring relationships predicted depressive symptoms in both sons and daughters even when taking into account mother-child relationships; higher rates of father-child conflict predicted depressive symptoms in both sons and daughters, and decreasing father-daughter closeness also predicted depressive symptoms in daughters. Mother-child relationships however demonstrated less consistent and salient associations with child depressive symptoms when father-child relationships were controlled for, and while mother-child conflict predicted depressive symptoms in sons, mother-child closeness was not associated with depressive symptoms in either sons or daughters (Yan et al., 2019). Though these studies focus on youth as opposed to adult offspring, the results of our study reflect similar results; in which accounting for externalising in the other parent, maternal

externalising appears to have a greater impact on male offspring than female offspring. With the findings of Yan et al., (2019) in mind, it again becomes clear that future research may find utility in collecting information regarding household composition as well as a measurement of parenting quality to include in analysis. Further allowing us to draw information regarding the potential mechanisms behind the relationship between parental externalising and internalising and externalising in offspring.

Measuring Parental Externalising

A primary set of limitations for this study is the limited data used to measure parental externalising. As noted previously, participants were asked whether their mother or father had engaged in problematic behaviour and were given a list of behaviours that this refers to. A simple yes or no to this question constituted whether we coded their mothers and fathers as exhibiting antisocial behaviour. Similarly participants were asked whether either of their parents engaged in problem substance use. While these were the only type of variables available in this dataset, we are unable to draw any conclusions about the intensity, frequency, timing or extent of parental anti-social behaviour, and/or problem substance use. Were we able to include the intensity or frequency in parental externalising, as we have in our early life adversities categories, we would perhaps see different interactions and influences demonstrated by our results. Logically it follows that in the future, it would be valuable to collect more extensive data regarding parental behaviour.

Without this information we were also unable to test whether it was the timing of externalising behaviour by parents during our participants childhood, or the frequency of externalising behaviour across childhood which had a more severe, and/or lasting impact on offspring. Unfortunately, we were therefore unable to test the findings by Whitten et al., (2019) which indicated that frequency of parental offending, rather than the age of the child at the time of the offending had a greater influence on offspring. It would have particularly

been interesting to test this hypothesis using our data as we were focussed on a wider range of anti-social behaviours than solely criminality/offending in both parents and in offspring (our participants). Future research should endeavour to collect data regarding the timing, frequency, and intensity of parental externalising in order to test hypotheses of intergenerational transmission of externalising behaviour. This would provide valuable information when allocating resources to intervention and treatment.

It would be informative to draw on collected data which can provide a confirmed diagnosis of substance use disorders by parent-report, or alternative methods and to compare related models to our current ones. In this study as parental externalising was measured using off-spring report we are unable to assess how accurate and reliable this information is. There is a possibility of both under, and over reporting by our participants. Firstly there is a potential risk that offspring may not have been aware of, or fully able to comprehend their parents externalising behaviour at the time of exposure. Secondly, perceptions of externalising behaviour may be biased in offspring who exhibit externalising symptoms themselves. These factors would lead to under-reporting of parental externalising. On the other hand, our potentially lower threshold for parental externalising may have also resulted in over-reporting by offspring. Additionally, data on parental externalising may also be confounded with the quality of child-parent relationships, adding another potential bias to these responses.

While this is a limitation in terms of reliability, measuring parental externalising from the perspective of offspring is also a strength. While it is important to uncover the relationship between these factors in reality, it is also important to understand how offspring perception of their experiences influences and changes the significance of these relationships. While this may explain why many results from this study were not in accordance with the

general consensus of current literature, it does provide an opportunity to compare results and further explore these differences.

None the less, diagnoses in research such as this may have utility in ensuring that inter-participant definitions of problem substance use are reliable. We should note that while we do not use a formal diagnosis of substance use disorders for parents in this study, the NESARC data-set does attempt to overcome this limitation by including a clear definition of what is considered to be problem substance use.

Furthermore, as the survey questions we used to measure parental externalising offer no temporal information we have no way of knowing if the parental externalising reported by our participants preceded adulthood and/or the development of any internalising or externalising symptoms in our participants. Without the temporal ordering of events, we are unable to specify the order of parental externalising, and internalising and/or externalising in offspring (DeLisi et al., 2020). Therefore, while we are able to test the strength of the relationship between our independent variables and dependent variables, our results cannot be used to support any claims regarding causation. In this way, longitudinal design methods would provide the ability to specify the ordering of events and subsequent reactions (DeLisi et al., 2020).

Retrospective Recollection

Like many other studies interested in the impact of ELA, another set of limitations of this study relates to the assessment of ELAs retrospectively. While self-report is a common assessment method when it comes to measuring ELAs it does inherently have a number of shortcomings. Firstly, there is the potential for bias such as recall bias as well as under, or over-reporting (Zhang et al., 2019). It has been demonstrated that reports of physical and sexual abuse for example can be highly unstable over time (Zhang et al., 2019).

Additionally, concurrent mental health issues may potentially impact the recall of childhood trauma or maltreatment (Latham et al., 2020). It has been suggested for example that individuals experiencing MDD may be more likely to remember and report negative events than positive ones. Considering that our results indicated a significant relationship between ELA and internalising, but not externalising, it is possible that biases such as this could influence recollection and therefore research outcomes. This possibility is one which should be further investigated in future research.

Some researchers have attempted to overcome the limitations of recollective recall by using prospective reports, that is official records of childhood trauma made at the time of the ELA (Latham et al., 2020). Prospective reports can come from a range of sources including official records, and caregiver reports. While avoiding time-related limitations they do come with their own limitations and biases, and results demonstrate only low-moderate between-method agreement when both prospective and retrospective recollection has been used, suggesting the two methods capture largely separate groups of individuals (Lathan et al., 2020). Additionally, recent findings suggest that these two types of reporting are differentially associated to adult outcomes with a stronger relationship demonstrated between ELA and later psychopathology when retrospective self-reporting was used (Newbury et al., 2018).

It appears that this relationship may be contingent on whether ELA experiences are recalled in adulthood. With this in mind, though the time-related limitations of retrospective recollection should be acknowledged, this method of data collection is still considered valid and reliable, and is often considered the gold-standard in ELA research (Zhang et al., 2019).

Sex and Gender

Throughout the literature there was no clear consensus on whether research should, or was, focusing its hypotheses on sex, or on gender. Where differences between sex or genders

were talked about, the terms used to refer to different sexes, or different genders appeared to be interchangeable; or in the least, no clarification was given around how data regarding participants sex or gender was collected. This is unsurprising considering that until perhaps more recent times the spectrum of gender was not so openly acknowledged or researched, with traditional research focusing on only two genders, male and female. Due to the limitation of the data used in our own research we too fall under this category. However it must be acknowledged that in this study, as well as in the wider literature, there is a gap regarding our knowledge and understanding of experiences of ELA, and parental externalising and the impact that these experiences have on gender diverse individuals. Furthermore, as participants were asked only for their sex, it is possible that in our study any participants identifying as a gender minority was included as either a male or a female. As there is no way of telling this from the data, it is possible that this has an influence on our results. It is our hope that future research takes the steps required to include a more diverse range of participants and gender identities rather than excluding or potentially mis-gendering individuals.

Investigating Sibling Relationships

An interesting direction for future studies to take when replicating and building on these findings would be to gather sibling data. There are a number of interesting lines of research that this could result in; firstly it would be interesting to see the moderation impact of sibling relationships on the association between parental externalising, experiences of ELA and internalising and externalising in offspring. Katz et al., (2018) report that siblings use their brother and sisters as sources of comfort and reassurance in the face of challenges and environmental threats. This raises the question; does having siblings lessen the impact? It has also been found that warmth in sibling relationships, less conflict, and less differential

treatment by parents is associated with reduced risk of externalising and internalising (Katz et al., 2018). Thus it appears sibling relationships may play a key role.

Additionally, collecting sibling data would allow researchers to investigate whether different relationships exist between our variables of interest depending on whether you are an older or younger sibling. Differential treatment by parents; maltreatment of older siblings leading to the development of avoidance strategies by younger siblings; higher likelihood of older siblings to report experiences of abuse, and the tendency for older siblings to experience domestic violence as more extreme are all factors that may impact experiences of parental externalising and/or ELA and the subsequent development of internalising and externalising symptoms in offspring depending on their order of birth (Katz et al., 2018; Witte et al., 2018).

It would be interesting to compare outcomes of siblings who share the same environment and thus potentially similar experiences of parental externalising and/or experiences of ELA to further investigate how similar experiences may impact females and males differently. Furthermore, though of course siblings in the same household may have vastly different experiences of ELA, it may provide some indication of the extent of under, or over reporting amongst participants, particularly if siblings are questioned about their own experiences as well as that of their siblings.

Conclusions

In this study we were interested in investigating the relationship between maternal and paternal externalising and internalising and externalising in offspring, and whether these relationships were mediated by experiences of ELA. We were further interested in the effect of sex on these relationships; specifically we were interested to see if mothers and fathers differentially impacted daughters and sons. We predicted firstly that ELA would be a significant mediator of the relationship between paternal externalising and internalising and

externalising in offspring, and secondly that there would be no significant difference overall in the impact that parental externalising has on offspring between mothers and fathers; but that externalising in mothers would have a stronger relationship with externalising and internalising in females, while externalising in fathers would have a stronger relationship with externalising and internalising in sons.

We did not find any evidence for a mediation of ELA on parental externalising and externalising in offspring, or a direct association between ELA and externalising. While we did find evidence of a mediation effect of ELA on the relationships between both Maternal and Paternal Externalising and Internalising in males, we did not find evidence for a mediation effect on Parental externalising and its relationship with Internalising in females. As such our results only partially support our hypothesis regarding ELA as a mediator.

Furthermore, our results did not demonstrate a significant association between either maternal or paternal externalising and externalising or internalising in male or female offspring. Though maternal externalising and paternal externalising had a significant relationship with internalising in males mediated by ELA, they did not have a significant association with internalising in females mediated by ELA. Additionally, while paternal externalising in isolation was not associated with externalising in either sex, when combined with experiences of ELA it was significantly associated with externalising in female offspring. In light of these results, we cannot conclude that there was no significant difference between the impact of maternal and paternal externalising; or that maternal externalising has a greater effect on internalising and externalising in female offspring and paternal externalising a greater impact on externalising in male offspring. It does however appear that paternal externalising does have a greater impact on internalising in male offspring than female offspring.

It is interesting to note that our results demonstrated that the worst outcomes can be predicted for male offspring who have experienced maternal and paternal externalising and ELA; wherein they have a highest likelihood of developing internalising symptoms.

Finally, though previous research has indicated that parental externalising has lasting adverse effects on offspring across their life-course, we found that these effects can be accounted for by experiences of ELA. In short, experiences of ELA, rather than parental externalising, appear to be the driving force behind the development of internalising symptoms in offspring.

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Appendix A

Supplementary Tables and Figures

Table A1.

Results of a Chi-Square tests for categorical variables of interest for the full sample of participants (n=26,728) comparing males (n=11,774) and females (n=14,954)

					Frequency	
	X^2	df	p= (<.05)	V	Male	Female
Major Depressive Disorder	377.570	1	0.001	<.01	1562	3373
Dysthymia	49.514	1	0.001	<.01	434	826
Specific Phobia	185.960	1	0.001	<.01	427	1131
Social Phobia	14.858	1	0.001	<.01	304	508
Panic Disorder	136.330	1	0.001	<.01	336	874
Generalised Anxiety Disorder	100.490	1	0.001	<.01	596	1222
Post-Traumatic Stress Disorder	119.080	1	0.001	<.01	437	1010
Alcohol Use Disorder	665.970	1	0.001	<.01	3990	2980
Drug Use Disorder	55.329	1	0.001	<.01	683	577
Tobacco Dependency	268.600	1	0.001	<.01	3481	3119
Anti-social Personality Disorder	254.910	1	0.001	<.01	637	275
Maternal Alcohol Use	15.227	1	0.001	<.01	780	1178
Maternal Drug Use	45.976	1	0.001	<.01	331	656
Maternal Antisocial Behaviour	24.386	1	0.001	<.01	475	797
Paternal Alcohol Use	8.304	1	0.001	<.01	2736	3702
Paternal Drug Use	17.514	1	0.001	<.01	588	925
Paternal Antisocial Bahaviour	7.777	1	0.001	<.01	1101	1552
Maternal Mood Disorder	120.120	1	0.001	<.01	2430	3947
Maternal Anxierty	50.663	1	0.001	<.01	1965	3006
Paternal Mood Disorder	29.352	1	0.001	<.01	1609	12554
Paternal Anxiety	3.643	1	0.001	<.01	1342	1818

Note: V denotes Cramer's V

Table A2.

Results of independent t-tests on continuous variables of interest in the full sample of participants (n=26,728) comparing male (n=11,774) and females (n=14,954)

					Males		Females	
	t	df	p= (<.05)	d_{Cohen}	Mean	SD	Mean	SD
Age	-3.519	25615	0.001	0.043	45.004	17.1	45.755	17.65
Emotional Abuse	0.560	26037	0.576	-0.007	0.493	0.79	0.487	0.85
Emotional Neglect	-0.690	25887	0.490	0.008	0.574	0.84	0.581	0.89
Physical Abuse	2.988	25686	0.003	-0.037	0.417	0.76	0.389	0.76
Physical Neglect	2.989	26110	0.003	-0.036	0.228	0.45	0.211	0.49
Sexual Abuse	-20.646	23819	0.001	0.241	0.047	0.26	0.145	0.49
Witnessing Violence	-9.134	26725	0.001	0.110	0.139	0.46	0.198	0.59

Note: dCohen denotes Cohen's D

Table A3.

Results of an independent t-tests on continuous variables of interest in the full sample of participants (n=26,728) comparing Group One (n=13,364) and Group Two (n=13,364)

					Group One		Group Two	
	t	df	p= (<.05)	d_{Cohen}	Mean	SD	Mean	SD
Age	1.208	26722	0.227	-0.015	45.553	17.52	45.296	17.3
Emotional Abuse	2.025	26726	0.043	-0.024	0.500	0.83	0.480	0.83
Emotional Neglect	1.549	26717	0.122	-0.018	0.586	0.88	0.570	0.86
Physical Abuse	1.664	26722	0.096	-0.019	0.409	0.78	0.394	0.77
Physical Neglect	1.988	26718	0.047	-0.025	0.225	0.48	0.213	0.47
Sexual Abuse	1.084	26706	0.278	-0.012	0.104	0.42	0.099	0.41
Witnessing Violence	0.648	26720	0.517	-0.007	0.174	0.54	0.170	0.53

Note: dCohen denotes Cohen's D

Table A4.

Results of Chi-Square tests for categorical variables of interest for the full sample of participants (n=26,728) comparing random subsets Group One (13,364) and Group Two (13,364)

					Frequency	
	X^2	df	p= (<.05)	V	Group One	Group Two
Sex (Male)	8.744	1	0.003	<.01	5,767	6,007
Major Depressive Disorder	0.090	1	0.765	<.01	2458	2477
Dysthymia	1.203	1	0.273	<.01	649	611
Specific Phobia	0.011	1	0.917	<.01	777	781
Social Phobia	2.240	1	0.134	<.01	385	427
Panic Disorder	0.419	1	0.518	<.01	594	616
Generalised Anxiety Disorder	4.780	1	0.029	<.01	954	864
Post-Traumatic Stress Disorder	1.000	1	0.317	<.01	742	705
Alcohol Use Disorder	0.609	1	0.435	<.01	3457	3513
Drug Use Disorder	0.163	1	0.686	<.01	637	623
Tobacco Dependency	3.094	1	0.079	<.01	3362	3238
Antisocial Personality Disorder	1.471	1	0.225	<.01	474	438
Maternal Alcohol Use	0.432	1	0.511	<.01	993	965
Maternal Drug Use	0.127	1	0.721	<.01	488	499
Maternal Antisocial Behaviour	1.747	1	0.186	<.01	659	613
Paternal Alcohol Use	0.082	1	0.775	<.01	3229	3209
Paternal Drug Use	0.511	1	0.475	<.01	770	743
Paternal Antisocial Bahaviour	1.360	1	0.244	<.01	1355	1298
Maternal Mood Disorder	3.322	1	0.068	<.01	3252	3125
Maternal Anxierty	0.803	1	0.370	<.01	2514	2457
Paternal Mood Disorder	0.648	1	0.421	<.01	1981	2028
Paternal Anxiety	0.116	1	0.733	<.01	1589	1571

Note: V denotes Cramer's V

Table A5.

Correlations for the full sample (n=26,728)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1. Major depressive disorder																											
2. Dysthymia	.33**																										
3. Specific phobia	.12**	.09**																									
4. Social phobia	.13**	.14**	.22**																								
5. Panic disorder	.17**	.17**	.17**	.21**																							
6. Generalised anxiety disorder	.26**	.26**	.17**	.22**	.24**																						
7. Post-traumatic stress disorder	.21**	.18**	.15**	.16**	.23**	.24**																					
8. Alcohol use disorder	.14**	.10**	.08**	.08**	.11**	.11**	.13**																				
9. Drug use disorder	.11**	.10**	.07**	.10**	.11**	.12**	.15**	.25**																			
10. Tobacco use disorder	.12**	.10**	.09**	.09**	.12**	.12**	.13**	.32**	.24**																		
11. Antisocial-personality disorder	.07**	.07**	.06**	.07**	.08**	.10**	.13**	.17**	.19**	.15**																	
12. Emotional abuse	.19**	.15**	.10**	.11**	.14**	.16**	.23**	.17**	.15**	.16**	.22**																
13. Emotional neglect	.09**	.11**	.04**	.07**	.07**	.09**	.10**	.05**	.07**	.06**	.10**	.36**															
14. Physical neglect	.11**	.12**	.07**	.09**	.10**	.12**	.19**	.11**	.09**	.10**	.16**	.56**	.35**														
15. Physical abuse	.16**	.12**	.09**	.09**	.12**	.13**	.20**	.14**	.13**	.16**	.22**	.79**	.32**	.51**													
16. Sexual abuse	.13**	.12**	.08**	.09**	.12**	.11**	.25**	.07**	.09**	.09**	.11**	.29**	.18**	.29**	.28**												
17. Environmental ELA	.09**	.07**	.06**	.06**	.10**	.10**	.11**	.07**	.07**	.08**	.09**	.19**	.12**	.15**	.17**	.14**											
18. Witnessing violence	.10**	.08**	.06**	.07**	.08**	.08**	.16**	.08**	.07**	.10**	.13**	.44**	.22**	.36**	.43**	.26**	.15**										
19. Maternal alcohol	.06**	.05**	.04**	.04**	.06**	.07**	.08**	.13**	.11**	.12**	.08**	.17**	.11**	.15**	.14**	.10**	.10**	.15**									
20. Maternal drug	.07**	.05**	.04**	.06**	.06**	.05**	.09**	.10**	.10**	.09**	.09**	.18**	.10**	.17**	.14**	.11**	.16**	.15**	.26**								
21. Maternal antisocial behaviour	.09**	.08**	.07**	.08**	.10**	.09**	.14**	.11**	.09**	.09**	.13**	.28**	.16**	.23**	.24**	.14**	.19**	.19**	.21**	.30**							
22. Paternal alcohol	.10**	.07**	.04**	.05**	.06**	.07**	.09**	.15**	.11**	.14**	.09**	.22**	.12**	.17**	.18**	.11**	.07**	.25**	.20**	.09**	.11**						
23. Paternal drug	.07**	.04**	.05**	.04**	.05**	.05**	.08**	.10**	.10**	.09**	.10**	.16**	.07**	.14**	.12**	.09**	.12**	.17**	.12**	.32**	.18**	.21**					
24. Paternal antisocial behaviour	.12**	.09**	.07**	.08**	.10**	.11**	.15**	.13**	.10**	.11**	.16**	.32**	.16**	.23**	.27**	.15**	.16**	.32**	.11**	.16**	.38**	.29**	.29**				
25. Maternal anxiety	.20**	.12**	.11**	.11**	.13**	.19**	.17**	.13**	.10**	.09**	.11**	.25**	.10**	.16**	.20**	.13**	.18**	.16**	.13**	.16**	.23**	.12**	.12**	.23**			
26. Maternal mood disorder	.27**	.16**	.11**	.11**	.14**	.18**	.16**	.15**	.10**	.11**	.10**	.25**	.11**	.17**	.21**	.13**	.20**	.16**	.19**	.21**	.24**	.14**	.16**	.22**	.44**		
27. Paternal anxiety	.15**	.09**	.10**	.10**	.11**	.15**	.14**	.09**	.07**	.07**	.09**	.19**	.06**	.13**	.15**	.08**	.13**	.11**	.05**	.07**	.14**	.12**	.12**	.22**	.45**	.22**	
28. Paternal mood disorder	.21**	.13**	.09**	.11**	.12**	.15**	.14**	.13**	.08**	.08**	.09**	.21**	.09**	.14**	.16**	.10**	.17**	.13**	.08**	.09**	.15**	.20**	.20**	.26**	.25**	.37**	.38**

Note. * indicates $p < .05$. ** indicates $p < .01$.

Figure A1.

Correlation plot for full sample (n=26,728). Blue dots indicate positive correlation, red dots indicate negative correlation. Dot size indicates strength of correlation; the larger the dot the closer to a correlation of 1

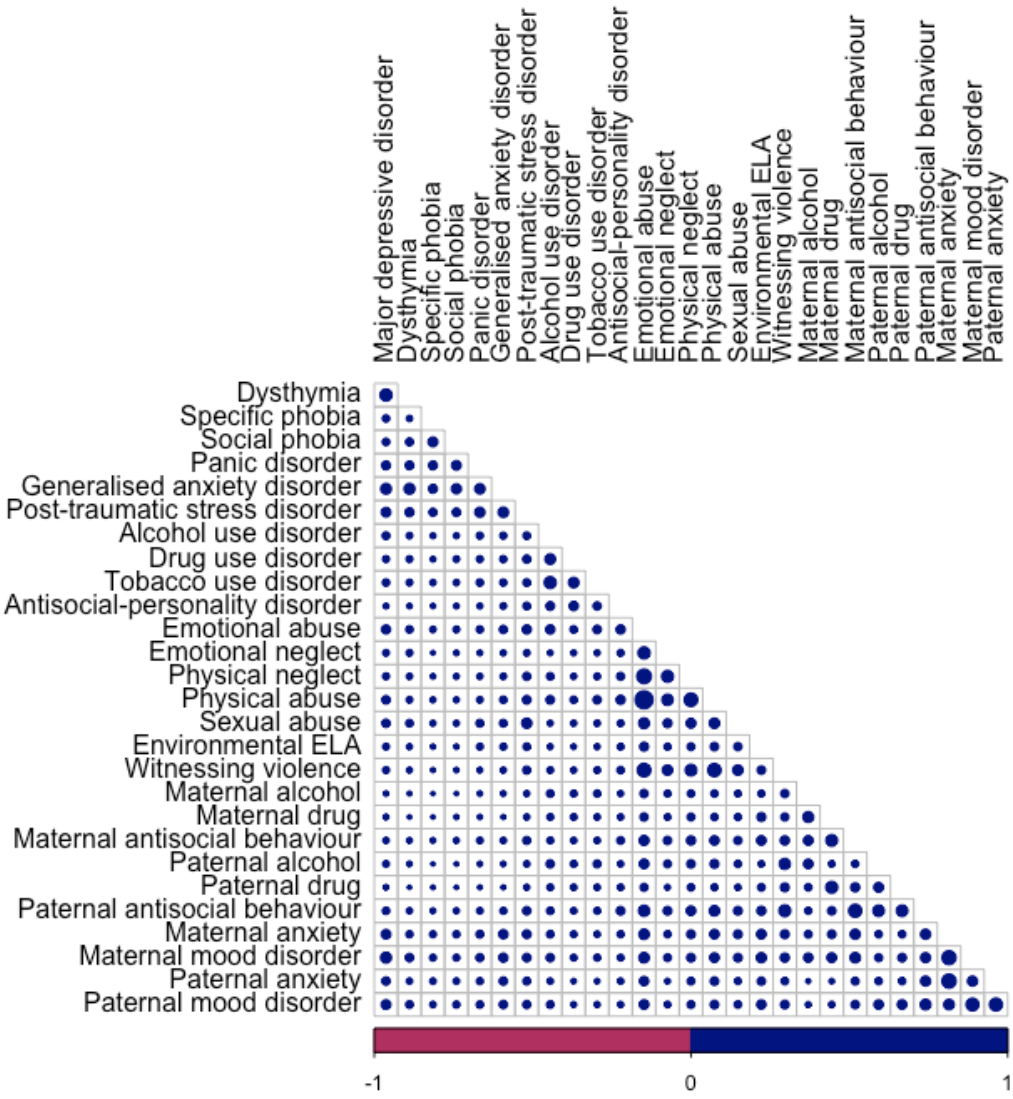

Table A6.

Correlations for the full sample for male (lower n=11,774) and female (upper, n=14,954) participants

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1. Major depressive disorder		.32**	.13**	.14**	.18**	.26**	.22**	.18**	.10**	.15**	.07**	.20**	.11**	.13**	.18**	.13**	.10**	.10**	.07**	.07**	.09**	.11**	.07**	.12**	.22**	.28**	.17**	.21**
2. Dysthymia	.35**		.08**	.14**	.17**	.26**	.18**	.11**	.09**	.12**	.06**	.16**	.11**	.13**	.13**	.13**	.07**	.09**	.06**	.05**	.08**	.07**	.04**	.09**	.12**	.17**	.09**	.14**
3. Specific phobia	.09**	.09**		.20**	.18**	.17**	.14**	.10**	.08**	.11**	.08**	.10**	.04**	.08**	.09**	.08**	.06**	.05**	.03**	.04**	.07**	.04**	.05**	.07**	.11**	.11**	.10**	.09**
4. Social phobia	.11**	.15**	.24**		.21**	.23**	.16**	.09**	.12**	.11**	.07**	.12**	.07**	.10**	.09**	.11**	.08**	.07**	.05**	.06**	.10**	.04**	.03**	.08**	.12**	.11**	.10**	.11**
5. Panic disorder	.13**	.15**	.14**	.21**		.24**	.24**	.15**	.13**	.15**	.10**	.17**	.09**	.12**	.14**	.12**	.10**	.08**	.07**	.06**	.12**	.07**	.05**	.11**	.15**	.16**	.12**	.13**
6. Generalised anxiety disorder	.24**	.25**	.17**	.22**	.22**		.25**	.14**	.13**	.14**	.10**	.17**	.10**	.13**	.14**	.12**	.10**	.08**	.07**	.04**	.10**	.07**	.06**	.11**	.21**	.20**	.16**	.15**
7. Post-traumatic stress disorder	.16**	.15**	.15**	.15**	.18**	.21**		.17**	.18**	.17**	.15**	.25**	.13**	.21**	.23**	.27**	.13**	.16**	.10**	.09**	.16**	.10**	.08**	.16**	.19**	.18**	.15**	.14**
8. Alcohol use disorder	.14**	.10**	.09**	.08**	.10**	.11**	.11**		.25**	.31**	.14**	.17**	.05**	.10**	.13**	.11**	.09**	.09**	.15**	.13**	.13**	.14**	.12**	.13**	.15**	.19**	.10**	.15**
9. Drug use disorder	.13**	.14**	.09**	.08**	.09**	.11**	.14**	.24**		.24**	.15**	.14**	.08**	.09**	.12**	.12**	.06**	.07**	.11**	.11**	.10**	.10**	.10**	.08**	.11**	.11**	.06**	.07**
10. Tobacco use disorder	.12**	.09**	.09**	.07**	.10**	.10**	.10**	.31**	.25**		.12**	.17**	.08**	.09**	.16**	.13**	.09**	.11**	.13**	.11**	.11**	.14**	.09**	.10**	.11**	.14**	.07**	.09**
11. Antisocial-personality disorder	.11**	.09**	.07**	.09**	.09**	.12**	.16**	.17**	.21**	.17**		.20**	.11**	.16**	.19**	.17**	.09**	.13**	.09**	.09**	.14**	.07**	.09**	.14**	.11**	.10**	.08**	.08**
12. Emotional abuse	.18**	.13**	.11**	.11**	.10**	.15**	.19**	.17**	.15**	.15**	.25**		.39**	.58**	.80**	.33**	.21**	.45**	.19**	.20**	.31**	.22**	.16**	.32**	.26**	.26**	.20**	.21**
13. Emotional neglect	.08**	.10**	.04**	.07**	.04**	.06**	.06**	.05**	.07**	.05**	.11**	.31**		.39**	.36**	.23**	.12**	.22**	.12**	.11**	.17**	.12**	.07**	.16**	.11**	.13**	.08**	.10**
14. Physical neglect	.10**	.10**	.08**	.08**	.07**	.10**	.15**	.11**	.09**	.10**	.18**	.52**	.31**		.54**	.33**	.17**	.37**	.17**	.20**	.26**	.17**	.14**	.23**	.18**	.19**	.14**	.14**
15. Physical abuse	.15**	.11**	.09**	.09**	.09**	.11**	.17**	.15**	.15**	.16**	.25**	.78**	.27**	.47**		.34**	.18**	.43**	.15**	.15**	.26**	.18**	.12**	.26**	.21**	.22**	.16**	.16**
16. Sexual abuse	.08**	.09**	.05**	.04**	.08**	.08**	.19**	.06**	.06**	.06**	.12**	.20**	.10**	.23**	.19**		.16**	.28**	.12**	.13**	.17**	.13**	.11**	.17**	.16**	.15**	.10**	.11**
17. Environmental ELA	.07**	.06**	.05**	.04**	.09**	.09**	.08**	.06**	.08**	.06**	.10**	.16**	.12**	.13**	.15**	.08**		.15**	.11**	.16**	.20**	.08**	.12**	.17**	.19**	.21**	.15**	.18**
18. Witnessing violence	.09**	.06**	.06**	.06**	.08**	.07**	.14**	.08**	.09**	.11**	.15**	.44**	.21**	.35**	.45**	.19**	.15**		.16**	.15**	.21**	.25**	.18**	.33**	.17**	.16**	.12**	.13**
19. Maternal alcohol	.05**	.03**	.04**	.03**	.05**	.08**	.05**	.11**	.10**	.11**	.09**	.15**	.09**	.12**	.12**	.06**	.08**	.13**		.27**	.21**	.20**	.13**	.11**	.14**	.19**	.06**	.08**
20. Maternal drug	.07**	.04**	.03**	.05**	.04**	.07**	.06**	.10**	.11**	.09**	.11**	.15**	.08**	.14**	.14**	.04**	.16**	.12**	.25**		.32**	.10**	.34**	.16**	.16**	.22**	.07**	.10**
21. Maternal antisocial behaviour	.08**	.07**	.06**	.05**	.05**	.08**	.10**	.09**	.09**	.09**	.14**	.23**	.14**	.19**	.20**	.08**	.18**	.16**	.20**	.28**		.12**	.19**	.38**	.24**	.25**	.15**	.15**
22. Paternal alcohol	.09**	.06**	.03**	.06**	.06**	.06**	.08**	.17**	.12**	.15**	.11**	.22**	.12**	.17**	.18**	.08**	.07**	.25**	.21**	.09**	.11**		.21**	.28**	.13**	.16**	.12**	.21**
23. Paternal drug	.06**	.04**	.04**	.06**	.05**	.04**	.08**	.10**	.10**	.10**	.12**	.16**	.07**	.14**	.13**	.04**	.13**	.17**	.11**	.29**	.16**	.21**		.30**	.13**	.17**	.13**	.21**
24. Paternal antisocial behaviour	.12**	.08**	.07**	.10**	.07**	.10**	.14**	.13**	.12**	.12**	.19**	.33**	.16**	.23**	.28**	.11**	.15**	.32**	.12**	.15**	.39**	.30**	.28**		.24**	.22**	.24**	.25**
25. Maternal anxiety	.16**	.09**	.09**	.10**	.10**	.15**	.13**	.12**	.10**	.08**	.13**	.24**	.08**	.14**	.19**	.07**	.16**	.15**	.13**	.15**	.21**	.11**	.11**	.21**		.45**	.43**	.25**
26. Maternal mood disorder	.23**	.13**	.10**	.10**	.11**	.15**	.12**	.14**	.10**	.10**	.12**	.23**	.09**	.15**	.20**	.08**	.18**	.14**	.18**	.19**	.23**	.11**	.15**	.21**	.42**		.23**	.35**
27. Paternal anxiety	.13**	.09**	.09**	.09**	.09**	.14**	.12**	.09**	.07**	.07**	.11**	.18**	.04**	.12**	.15**	.04**	.11**	.11**	.05**	.06**	.12**	.12**	.10**	.21**	.48**	.22**		.39**
28. Paternal mood disorder	.20**	.13**	.10**	.11**	.11**	.15**	.14**	.12**	.09**	.08**	.11**	.21**	.07**	.13**	.16**	.08**	.15**	.13**	.07**	.08**	.16**	.18**	.19**	.26**	.25**	.40**	.37**	

Note. * indicates $p < .05$. ** indicates $p < .01$. Results for male participants are displayed in the lower left half of the table, results for female participants are shown in the upper right half of the table.

Table A7.

Correlations for Group One participants (n=13,364)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1. Major depressive disorder																											
2. Dysthymia	.33**																										
3. Specific phobia	.12**	.09**																									
4. Social phobia	.14**	.16**	.20**																								
5. Panic disorder	.16**	.18**	.19**	.20**																							
6. Generalised anxiety disorder	.25**	.26**	.19**	.22**	.23**																						
7. Post-traumatic stress disorder	.21**	.17**	.15**	.16**	.23**	.24**																					
8. Alcohol use disorder	.13**	.08**	.07**	.08**	.09**	.10**	.13**																				
9. Drug use disorder	.10**	.09**	.08**	.10**	.11**	.12**	.17**	.24**																			
10. Tobacco use disorder	.12**	.10**	.09**	.09**	.12**	.12**	.14**	.32**	.24**																		
11. Antisocial-personality disorder	.08**	.06**	.06**	.08**	.07**	.09**	.14**	.16**	.20**	.16**																	
12. Emotional abuse	.18**	.15**	.11**	.12**	.15**	.16**	.23**	.16**	.14**	.15**	.23**																
13. Emotional neglect	.09**	.12**	.05**	.08**	.08**	.08**	.09**	.06**	.09**	.06**	.11**	.35**															
14. Physical neglect	.11**	.11**	.08**	.10**	.10**	.11**	.19**	.09**	.11**	.10**	.17**	.55**	.34**														
15. Physical abuse	.16**	.13**	.09**	.09**	.12**	.12**	.20**	.13**	.13**	.15**	.22**	.79**	.31**	.51**													
16. Sexual abuse	.13**	.11**	.09**	.10**	.12**	.10**	.26**	.07**	.10**	.09**	.13**	.28**	.19**	.30**	.27**												
17. Environmental ELA	.09**	.07**	.06**	.07**	.11**	.11**	.12**	.06**	.07**	.08**	.10**	.18**	.12**	.14**	.15**	.13**											
18. Witnessing violence	.11**	.07**	.07**	.06**	.09**	.07**	.15**	.06**	.07**	.10**	.12**	.42**	.20**	.36**	.42**	.26**	.12**										
19. Maternal alcohol	.06**	.04**	.04**	.05**	.05**	.05**	.07**	.11**	.10**	.11**	.08**	.16**	.11**	.14**	.12**	.10**	.08**	.13**									
20. Maternal drug	.08**	.05**	.04**	.07**	.06**	.05**	.10**	.10**	.10**	.09**	.10**	.18**	.11**	.18**	.13**	.12**	.15**	.13**	.26**								
21. Maternal antisocial behaviour	.09**	.08**	.07**	.10**	.10**	.10**	.13**	.11**	.08**	.09**	.13**	.28**	.15**	.21**	.23**	.14**	.18**	.18**	.19**	.29**							
22. Paternal alcohol	.10**	.06**	.04**	.04**	.06**	.06**	.09**	.14**	.11**	.14**	.08**	.21**	.12**	.16**	.16**	.11**	.07**	.24**	.21**	.11**	.10**						
23. Paternal drug	.06**	.03**	.05**	.05**	.04**	.04**	.09**	.10**	.10**	.09**	.10**	.15**	.06**	.14**	.12**	.10**	.12**	.16**	.12**	.32**	.18**	.21**					
24. Paternal antisocial behaviour	.12**	.10**	.07**	.08**	.11**	.10**	.16**	.12**	.08**	.11**	.16**	.32**	.15**	.23**	.26**	.16**	.17**	.31**	.10**	.14**	.38**	.29**	.28**				
25. Maternal anxiety	.21**	.12**	.10**	.13**	.13**	.20**	.19**	.13**	.10**	.10**	.11**	.25**	.10**	.16**	.20**	.15**	.17**	.15**	.13**	.15**	.23**	.10**	.11**	.23**			
26. Maternal mood disorder	.26**	.14**	.11**	.12**	.15**	.19**	.17**	.15**	.11**	.12**	.10**	.25**	.11**	.16**	.21**	.13**	.20**	.14**	.19**	.21**	.24**	.13**	.16**	.22**	.45**		
27. Paternal anxiety	.15**	.08**	.11**	.11**	.10**	.15**	.15**	.08**	.07**	.07**	.09**	.19**	.06**	.13**	.16**	.09**	.12**	.11**	.04**	.06**	.14**	.11**	.11**	.24**	.46**	.23**	
28. Paternal mood disorder	.20**	.14**	.11**	.11**	.13**	.15**	.16**	.12**	.08**	.09**	.09**	.21**	.09**	.13**	.16**	.11**	.16**	.12**	.07**	.10**	.15**	.19**	.21**	.25**	.26**	.38**	.38**

Note. * indicates $p < .05$. ** indicates $p < .01$.

Table A8.

Correlations for Group Two participants (n=13,364)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
1. Major depressive disorder																											
2. Dysthymia	.33**																										
3. Specific phobia	.13**	.09**																									
4. Social phobia	.12**	.13**	.23**																								
5. Panic disorder	.18**	.16**	.15**	.21**																							
6. Generalised anxiety disorder	.26**	.27**	.16**	.23**	.25**																						
7. Post-traumatic stress disorder	.20**	.18**	.14**	.15**	.23**	.24**																					
8. Alcohol use disorder	.14**	.11**	.08**	.08**	.13**	.12**	.13**																				
9. Drug use disorder	.11**	.12**	.07**	.09**	.11**	.11**	.13**	.26**																			
10. Tobacco use disorder	.13**	.10**	.09**	.09**	.13**	.11**	.13**	.33**	.25**																		
11. Antisocial-personality disorder	.07**	.08**	.06**	.07**	.09**	.11**	.12**	.18**	.17**	.14**																	
12. Emotional abuse	.20**	.15**	.10**	.11**	.14**	.16**	.23**	.17**	.15**	.16**	.21**																
13. Emotional neglect	.10**	.09**	.03**	.06**	.07**	.09**	.12**	.05**	.06**	.07**	.10**	.37**															
14. Physical neglect	.12**	.12**	.07**	.08**	.10**	.13**	.19**	.12**	.08**	.10**	.16**	.57**	.37**														
15. Physical abuse	.16**	.12**	.08**	.09**	.11**	.13**	.20**	.15**	.14**	.16**	.22**	.79**	.33**	.52**													
16. Sexual abuse	.13**	.12**	.08**	.08**	.12**	.12**	.25**	.07**	.08**	.09**	.10**	.29**	.18**	.28**	.29**												
17. Environmental ELA	.09**	.07**	.05**	.05**	.09**	.09**	.10**	.08**	.06**	.07**	.08**	.20**	.12**	.16**	.18**	.15**											
18. Witnessing violence	.10**	.09**	.05**	.08**	.08**	.09**	.16**	.09**	.08**	.10**	.13**	.46**	.24**	.37**	.45**	.25**	.18**										
19. Maternal alcohol	.06**	.06**	.03**	.04**	.08**	.09**	.10**	.14**	.11**	.12**	.08**	.19**	.10**	.16**	.16**	.11**	.13**	.17**									
20. Maternal drug	.07**	.04**	.05**	.05**	.06**	.06**	.07**	.11**	.11**	.10**	.08**	.18**	.09**	.17**	.16**	.10**	.17**	.16**	.26**								
21. Maternal antisocial behaviour	.09**	.08**	.06**	.06**	.10**	.09**	.15**	.11**	.10**	.10**	.13**	.28**	.17**	.26**	.24**	.14**	.21**	.21**	.23**	.32**							
22. Paternal alcohol	.10**	.07**	.03**	.05**	.07**	.07**	.09**	.16**	.11**	.14**	.09**	.23**	.12**	.18**	.19**	.11**	.08**	.25**	.20**	.08**	.13**						
23. Paternal drug	.07**	.05**	.04**	.04**	.07**	.06**	.08**	.11**	.09**	.10**	.10**	.17**	.08**	.14**	.13**	.08**	.13**	.19**	.13**	.32**	.18**	.22**					
24. Paternal antisocial behaviour	.12**	.08**	.07**	.09**	.09**	.12**	.15**	.13**	.11**	.11**	.17**	.33**	.16**	.23**	.28**	.14**	.16**	.34**	.13**	.17**	.38**	.29**	.31**				
25. Maternal anxiety	.20**	.11**	.11**	.10**	.13**	.18**	.14**	.13**	.10**	.09**	.12**	.25**	.09**	.16**	.20**	.11**	.19**	.18**	.14**	.16**	.23**	.14**	.14**	.22**			
26. Maternal mood disorder	.28**	.17**	.10**	.10**	.14**	.18**	.15**	.15**	.09**	.10**	.10**	.26**	.12**	.18**	.20**	.13**	.20**	.18**	.19**	.22**	.24**	.15**	.17**	.22**	.44**		
27. Paternal anxiety	.16**	.10**	.08**	.08**	.12**	.15**	.12**	.10**	.06**	.07**	.10**	.19**	.06**	.13**	.15**	.08**	.14**	.12**	.07**	.08**	.13**	.13**	.13**	.21**	.44**	.22**	
28. Paternal mood disorder	.21**	.13**	.08**	.11**	.12**	.15**	.13**	.14**	.07**	.08**	.09**	.21**	.08**	.14**	.16**	.09**	.17**	.14**	.08**	.09**	.15**	.20**	.20**	.27**	.24**	.37**	.38**

Note. * indicates $p < .05$. ** indicates $p < .01$.

Figure A2.

Correlation plot for Group Two participants (n=13,364). Blue dots indicate positive correlation, red dots indicate negative correlation. Dot size indicates strength of correlation; the larger the dot the closer to a correlation of 1

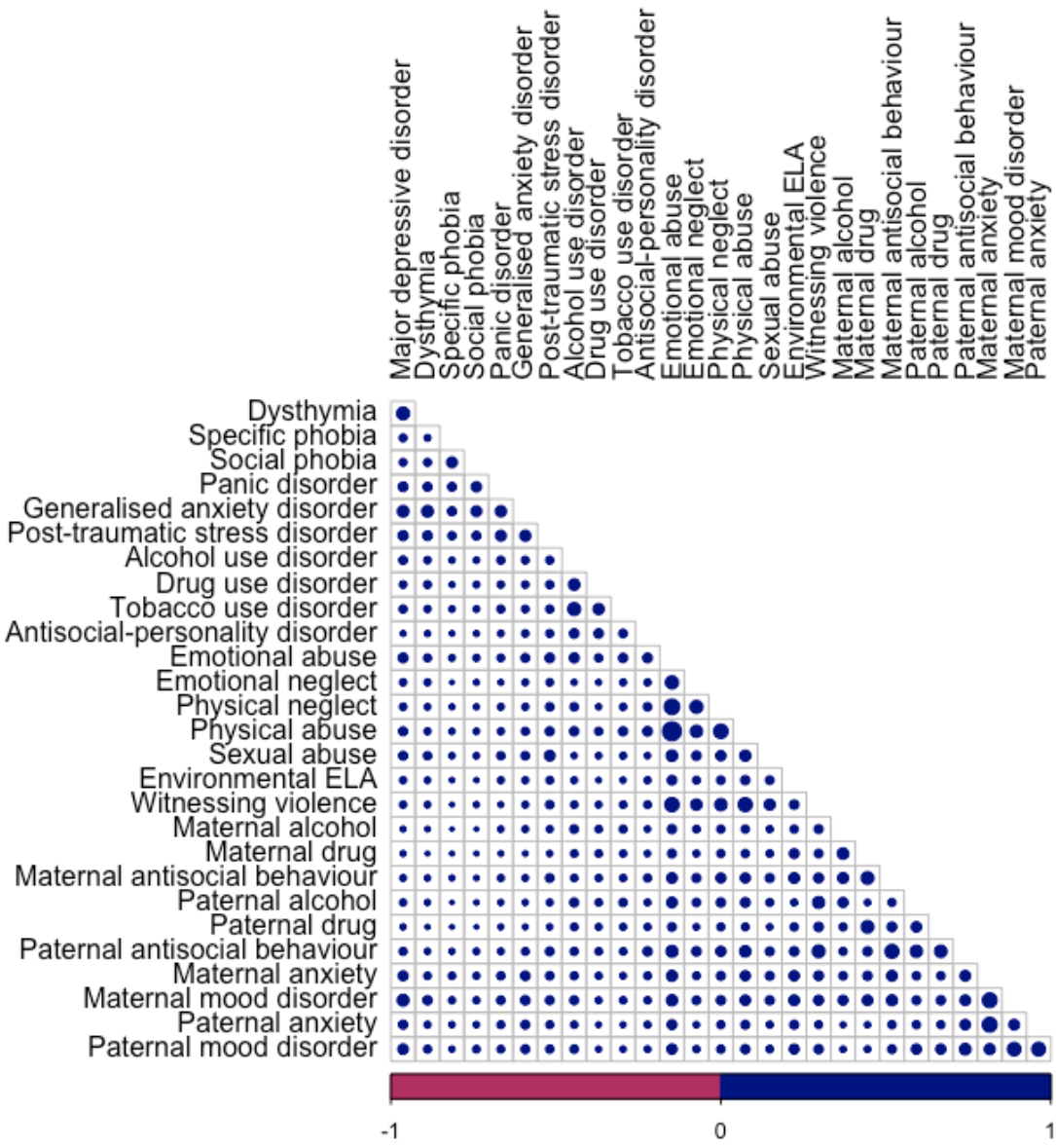

Table A9.

Correlations for Group Two male (lower, n=6,007) and female (upper, n=7,357) participants

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
1. Major depressive disorder		.32**	.13**	.14**	.20**	.27**	.22**	.19**	.11**	.17**	.06**	.22**	.12**	.14**	.18**	.14**	.09**	.10**	.07**	.07**	.10**	.11**	.08**	.13**	.22**	.29**	.18**	.22**
2. Dysthymia	.35**		.08**	.13**	.16**	.27**	.19**	.13**	.11**	.13**	.08**	.17**	.10**	.13**	.13**	.14**	.08**	.11**	.07**	.05**	.08**	.08**	.07**	.09**	.12**	.19**	.10**	.13**
3. Specific phobia	.09**	.09**		.22**	.15**	.15**	.13**	.11**	.07**	.10**	.07**	.10**	.03**	.07**	.09**	.07**	.05**	.04**	.03*	.05**	.07**	.03**	.03**	.06**	.12**	.11**	.08**	.07**
4. Social phobia	.08**	.12**	.25**		.22**	.23**	.16**	.10**	.12**	.11**	.07**	.11**	.06**	.08**	.08**	.09**	.07**	.07**	.05**	.06**	.07**	.05**	0.02	.08**	.11**	.11**	.08**	.12**
5. Panic disorder	.13**	.13**	.13**	.18**		.26**	.25**	.18**	.13**	.16**	.12**	.16**	.08**	.13**	.13**	.13**	.09**	.07**	.09**	.06**	.12**	.07**	.07**	.10**	.16**	.15**	.13**	.11**
6. Generalised anxiety disorder	.24**	.26**	.16**	.22**	.22**		.25**	.15**	.11**	.14**	.10**	.16**	.11**	.14**	.14**	.13**	.09**	.10**	.08**	.05**	.10**	.08**	.07**	.13**	.21**	.19**	.17**	.16**
7. Post-traumatic stress disorder	.14**	.15**	.13**	.14**	.16**	.22**		.18**	.16**	.16**	.14**	.25**	.15**	.21**	.23**	.26**	.11**	.15**	.11**	.07**	.16**	.10**	.08**	.16**	.17**	.17**	.14**	.14**
8. Alcohol use disorder	.15**	.10**	.08**	.08**	.11**	.12**	.10**		.25**	.31**	.14**	.17**	.04**	.10**	.13**	.11**	.11**	.11**	.16**	.12**	.12**	.16**	.13**	.15**	.15**	.19**	.12**	.17**
9. Drug use disorder	.14**	.15**	.09**	.07**	.10**	.12**	.12**	.26**		.23**	.15**	.15**	.06**	.08**	.13**	.12**	.06**	.08**	.12**	.12**	.12**	.10**	.08**	.09**	.10**	.11**	.06**	.07**
10. Tobacco use disorder	.12**	.09**	.09**	.07**	.10**	.10**	.11**	.33**	.25**		.11**	.17**	.08**	.08**	.16**	.14**	.08**	.11**	.15**	.11**	.11**	.15**	.10**	.11**	.10**	.13**	.07**	.09**
11. Antisocial-personality disorder	.11**	.09**	.08**	.08**	.09**	.14**	.14**	.18**	.18**	.15**		.19**	.10**	.15**	.20**	.15**	.08**	.15**	.08**	.07**	.13**	.09**	.10**	.15**	.09**	.10**	.07**	.07**
12. Emotional abuse	.18**	.13**	.10**	.11**	.11**	.16**	.20**	.17**	.15**	.15**	.23**		.40**	.60**	.79**	.35**	.22**	.46**	.21**	.20**	.31**	.23**	.16**	.32**	.25**	.26**	.19**	.20**
13. Emotional neglect	.06**	.08**	.03*	.06**	.04**	.07**	.08**	.05**	.06**	.06**	.10**	.33**		.39**	.36**	.22**	.12**	.24**	.12**	.10**	.18**	.11**	.07**	.16**	.11**	.13**	.08**	.09**
14. Physical neglect	.09**	.11**	.09**	.09**	.08**	.11**	.17**	.13**	.08**	.11**	.17**	.52**	.34**		.55**	.32**	.18**	.38**	.18**	.19**	.27**	.18**	.14**	.23**	.18**	.19**	.14**	.14**
15. Physical abuse	.15**	.11**	.09**	.10**	.10**	.13**	.17**	.16**	.16**	.16**	.24**	.78**	.29**	.47**		.35**	.19**	.44**	.18**	.17**	.25**	.18**	.12**	.26**	.21**	.21**	.15**	.15**
16. Sexual abuse	.07**	.08**	.04**	.05**	.06**	.07**	.21**	.06**	.04**	.05**	.10**	.21**	.10**	.21**	.19**		.17**	.29**	.13**	.13**	.17**	.12**	.10**	.16**	.15**	.14**	.10**	.09**
17. Environmental ELA	.07**	.05**	.05**	0.02	.09**	.10**	.09**	.07**	.07**	.07**	.09**	.18**	.12**	.15**	.17**	.09**		.18**	.14**	.17**	.21**	.08**	.12**	.16**	.20**	.21**	.17**	.18**
18. Witnessing violence	.08**	.07**	.04**	.09**	.09**	.08**	.15**	.09**	.09**	.11**	.14**	.47**	.24**	.35**	.48**	.16**	.17**		.18**	.18**	.22**	.25**	.19**	.35**	.19**	.19**	.12**	.15**
19. Maternal alcohol	.05**	.03*	.04**	.03*	.06**	.11**	.07**	.14**	.10**	.11**	.09**	.16**	.09**	.14**	.13**	.05**	.10**	.14**		.26**	.23**	.20**	.14**	.12**	.13**	.20**	.06**	.08**
20. Maternal drug	.05**	.03*	.03*	.03*	.04**	.06**	.07**	.11**	.10**	.09**	.10**	.16**	.07**	.13**	.15**	.04**	.18**	.13**	.27**		.33**	.08**	.33**	.16**	.17**	.23**	.09**	.09**
21. Maternal antisocial behaviour	.08**	.06**	.05**	.03*	.05**	.07**	.11**	.10**	.09**	.10**	.14**	.25**	.15**	.23**	.22**	.07**	.21**	.19**	.24**	.31**		.13**	.19**	.37**	.23**	.24**	.14**	.14**
22. Paternal alcohol	.08**	.06**	.03*	.06**	.07**	.06**	.08**	.17**	.12**	.13**	.11**	.24**	.12**	.18**	.20**	.08**	.07**	.25**	.20**	.08**	.13**		.22**	.28**	.15**	.17**	.13**	.22**
23. Paternal drug	.04**	0.02	.05**	.07**	.06**	.05**	.06**	.11**	.11**	.10**	.11**	.18**	.08**	.14**	.15**	.03*	.14**	.18**	.11**	.30**	.17**	.21**		.31**	.14**	.17**	.15**	.21**
24. Paternal antisocial behaviour	.12**	.07**	.09**	.11**	.07**	.09**	.14**	.12**	.14**	.12**	.20**	.34**	.17**	.23**	.30**	.10**	.15**	.33**	.14**	.17**	.40**	.31**	.30**		.23**	.22**	.22**	.26**
25. Maternal anxiety	.14**	.09**	.09**	.08**	.09**	.13**	.10**	.12**	.11**	.09**	.16**	.25**	.07**	.14**	.20**	.03**	.17**	.16**	.16**	.15**	.22**	.12**	.13**	.21**		.45**	.41**	.25**
26. Maternal mood disorder	.24**	.14**	.07**	.08**	.11**	.14**	.10**	.14**	.09**	.09**	.13**	.25**	.10**	.16**	.21**	.08**	.18**	.16**	.18**	.20**	.23**	.12**	.15**	.22**	.42**		.22**	.34**
27. Paternal anxiety	.13**	.10**	.08**	.08**	.10**	.13**	.09**	.09**	.07**	.07**	.13**	.18**	.05**	.11**	.15**	.03**	.10**	.11**	.08**	.06**	.13**	.13**	.10**	.20**	.48**	.22**		.40**
28. Paternal mood disorder	.19**	.12**	.08**	.11**	.11**	.14**	.12**	.12**	.09**	.08**	.12**	.22**	.07**	.14**	.17**	.08**	.15**	.13**	.08**	.09**	.16**	.18**	.19**	.28**	.22**	.40**	.36**	

Note. * indicates $p < .05$. ** indicates $p < .01$. Results for male participants are displayed in the lower left half of the table, results for female participants are shown in the upper right half of the table.

Table A10.

Standardised factor loadings onto the latent variables for participant Groups One and Two, females and males

	Group One						Group Two					
	Females			Males			Females			Males		
	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)
Internalising												
Major depressive	0.453	0.006	<.001	0.417	0.006	<.001	0.480	0.006	<.001	0.425	0.006	<.001
Dysthymia	0.395	0.004	<.001	0.363	0.004	<.001	0.423	0.004	<.001	0.373	0.004	<.001
Specific Phobia	0.331	0.004	<.001	0.358	0.004	<.001	0.310	0.004	<.001	0.311	0.004	<.001
Social Phobia	0.410	0.004	<.001	0.368	0.004	<.001	0.402	0.004	<.001	0.331	0.004	<.001
Panic Disorder	0.417	0.004	<.001	0.445	0.004	<.001	0.440	0.005	<.001	0.426	0.005	<.001
Generalised Anxiety Disorder	0.530	0.005	<.001	0.502	0.005	<.001	0.553	0.005	<.001	0.503	0.005	<.001
Post-traumatic Stress Disorder	0.492	0.005	<.001	0.481	0.005	<.001	0.469	0.005	<.001	0.444	0.005	<.001
Externalising												
Alcohol use Disorder	0.518	0.006	<.001	0.535	0.006	<.001	0.537	0.006	<.001	0.588	0.006	<.001
Cannabis use Disorder	0.527	0.004	<.001	0.478	0.004	<.001	0.472	0.004	<.001	0.446	0.004	<.001
Drug use Disorder	0.480	0.004	<.001	0.515	0.004	<.001	0.480	0.004	<.001	0.498	0.004	<.001
Tobacco use Disorder	0.477	0.006	<.001	0.526	0.006	<.001	0.468	0.006	<.001	0.539	0.006	<.001
Antisocial Personality Disorder	0.380	0.003	<.001	0.305	0.003	<.001	0.363	0.003	<.001	0.283	0.003	<.001
Early Life Adversity												
Emotional Abuse	0.852	0.017	<.001	0.773	0.017	<.001	0.869	0.017	<.001	0.798	0.017	<.001
Emotional Neglect	0.441	0.012	<.001	0.366	0.012	<.001	0.449	0.011	<.001	0.404	0.011	<.001
Physical Abuse	0.759	0.016	<.001	0.656	0.016	<.001	0.777	0.016	<.001	0.697	0.016	<.001
Physical Neglect	0.674	0.010	<.001	0.613	0.010	<.001	0.685	0.009	<.001	0.613	0.009	<.001
Sexual Abuse	0.329	0.008	<.001	0.449	0.008	<.001	0.291	0.007	<.001	0.419	0.007	<.001
Witnessing Violence	0.555	0.012	<.001	0.597	0.012	<.001	0.596	0.011	<.001	0.619	0.011	<.001
Enviornmental ELA	0.270	0.005	<.001	0.222	0.005	<.001	0.277	0.005	<.001	0.275	0.005	<.001
Paternal Externalising												
Paternal Problematic Alcohol Use	0.460	0.006	<.001	0.441	0.006	<.001	0.470	0.006	<.001	0.470	0.006	<.001
Paternal Problematic Drug Use	0.425	0.005	<.001	0.431	0.005	<.001	0.443	0.005	<.001	0.467	0.005	<.001
Papternal Antisocial Behaviour	0.660	0.006	<.001	0.649	0.006	<.001	0.651	0.006	<.001	0.657	0.006	<.001
Maternal Externalising												
Maternal Problematic Alcohol Use	0.390	0.006	<.001	0.323	0.006	<.001	0.430	0.006	<.001	0.598	0.006	<.001
Maternal Problematic Drug Use	0.462	0.006	<.001	0.460	0.006	<.001	0.487	0.006	<.001	0.495	0.006	<.001
Maternal Antisocial behaviour	0.601	0.007	<.001	0.529	0.007	<.001	0.608	0.006	<.001	0.412	0.006	<.001
Paternal Internalising												
Paternal Anxiety	0.628	0.006	<.001	0.613	0.006	<.001	0.624	0.006	<.001	0.583	0.006	<.001
Paternal Mood disorder	0.609	0.006	<.001	0.611	0.006	<.001	0.634	0.006	<.001	0.616	0.006	<.001
Maternal Internalising												
Maternal Anxiety	0.693	0.005	<.001	0.679	0.005	<.001	0.674	0.006	<.001	0.645	0.006	<.001
Maternal Mood disorder	0.651	0.005	<.001	0.638	0.005	<.001	0.666	0.006	<.001	0.644	0.006	<.001
Model fitness indicies												
Robust Compative Fit Index (CFI)				0.922						0.921		
Robust Tucker-Lewis Index (TLI)				0.910						0.909		
Robust RMSEA				0.034						0.035		

Table A11.

Direct effects in the structural equation model for Group One and Two, females and males

	Group One						Group Two					
	Females			Males			Females			Males		
	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)
Early Life Adversity												
Maternal Externalising	0.349	0.128	<.001	-0.038	0.327	0.873	0.349	0.082	<.001	0.084	0.140	0.441
Maternal Internalising	-0.080	0.127	0.406	-0.488	0.458	0.217	-0.073	0.078	0.215	-0.367	0.237	0.051
Paternal Externalising	0.286	0.123	<.001	0.402	0.421	0.292	0.352	0.068	<.001	0.581	0.102	<.001
Paternal Internalising	-0.028	0.103	0.763	0.628	0.205	0.001	0.021	0.075	0.714	0.297	0.229	0.107
Internalising												
Early Life Adversity	0.295	0.032	<.001	0.271	0.050	<.001	0.275	0.032	<.001	0.334	0.044	0.000
Maternal Externalising	-0.051	0.131	0.618	0.161	0.464	0.280	-0.063	0.076	0.294	0.047	0.108	0.654
Maternal Internalising	-0.409	0.147	<.001	0.826	0.826	0.421	-0.399	0.088	<.001	0.031	0.217	0.884
Paternal Externalising	-0.110	0.148	0.342	-0.182	0.346	0.346	-0.055	0.067	0.300	-0.122	0.096	0.232
Paternal Internalising	-0.050	0.112	0.571	-0.532	0.169	0.395	-0.149	0.086	0.028	-0.414	0.215	0.054
Externalising												
Early Life Adversity	0.119	0.031	0.001	0.104	0.062	0.019	0.066	0.034	0.085	0.073	0.061	0.095
Maternal Externalising	0.243	0.123	0.021	0.089	0.331	0.604	0.281	0.081	<.001	0.181	0.156	0.040
Maternal Internalising	-0.220	0.128	0.081	-0.257	0.490	0.396	-0.066	0.079	0.329	-0.085	0.288	0.608
Paternal Externalising	0.099	0.105	0.267	0.338	0.232	0.023	0.187	0.072	0.002	0.279	0.136	0.001
Paternal Internalising	0.039	0.129	0.726	0.186	0.449	0.521	-0.032	0.081	0.645	0.011	0.276	0.945

Table A12.

Indirect effects in the structural equation model for Group One and Two, females and males

	Group One						Group Two					
	Females			Males			Females			Males		
	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)
Internalising												
Maternal Externalising	-0.010	0.083	0.874	0.103	0.040	0.001	0.028	0.037	0.433	0.096	0.027	<.001
Maternal Internalising	-0.132	0.128	0.270	-0.024	0.036	0.405	-0.123	0.071	0.079	-0.020	0.020	0.215
Paternal Externalising	0.170	0.072	0.015	0.084	0.031	0.001	0.194	0.044	<.001	0.097	0.024	<.001
Paternal Internalising	0.109	0.116	0.339	-0.008	0.764	0.035	0.099	0.067	0.140	0.006	0.714	0.020
Externalising												
Maternal Externalising	-0.004	0.047	0.871	0.285	0.124	0.007	0.043	0.015	0.474	0.023	0.015	0.070
Maternal Internalising	-0.051	0.067	0.219	-0.200	0.131	0.073	-0.027	0.036	0.193	-0.005	0.006	0.358
Paternal Externalising	0.065	0.045	0.022	0.133	0.106	0.143	0.006	0.041	0.092	0.023	0.016	0.090
Paternal Internalising	0.042	0.060	0.281	0.035	0.131	0.754	0.022	0.031	0.222	0.001	0.004	0.722

Table A13.

Total effects in the structural equation model for Group One and Two, females and males

	Group One						Group Two					
	Females			Males			Females			Males		
	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)	Estimate	Std.Err	*p* (<.006)
Internalising												
Maternal Externalising	0.150	0.254	0.451	0.052	0.131	0.614	0.075	0.106	0.464	0.033	0.073	0.564
Maternal Internalising	-0.046	0.367	0.894	-0.432	0.151	<.001	-0.092	0.200	0.642	-0.419	0.089	<.001
Paternal Externalising	-0.012	0.162	0.941	0.035	0.113	0.696	0.072	0.077	0.380	0.042	0.064	0.403
Paternal Internalising	-0.423	0.346	0.212	0.151	-0.118	0.317	-0.314	0.198	0.112	-0.144	0.087	0.036
Externalising												
Maternal Externalising	0.085	0.352	0.641	0.285	0.124	0.007	0.187	0.162	0.040	0.304	0.076	<.001
Maternal Internalising	0.322	0.504	0.322	-0.200	0.073	0.131	-0.112	0.285	0.495	-0.070	0.080	0.304
Paternal Externalising	0.403	0.220	0.004	0.133	0.106	0.143	0.321	0.118	<.001	0.210	0.069	<.001
Paternal Internalising	0.228	0.462	0.446	0.035	0.131	0.754	0.033	0.274	0.836	-0.031	0.082	0.662

Figure A3.

*Structural equation model for Group Two female particiants indicating the association between latent dimensions of parental externalising and internalising, early life adversity and latent dimensions of internalising and (n=*7,357*). Circles represent latent variables and rectangles represent observed variables*

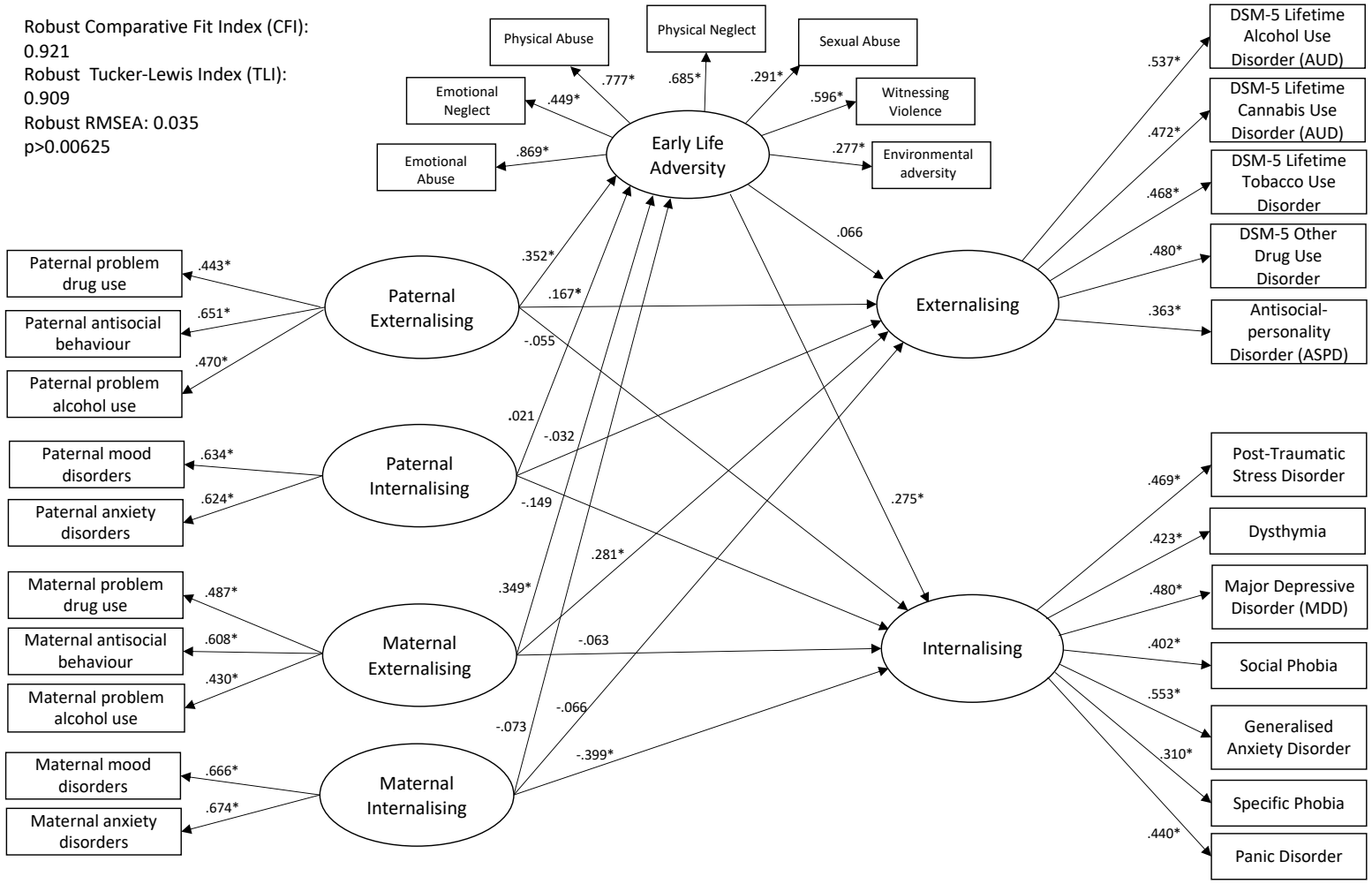

Table A14.

Indirect effects in the structural equation model for Group Two female participants (n=7,357)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.028	0.037	0.433
Maternal Internalising	-0.123	0.071	0.079
Paternal Externalising	0.194	0.044	<.001
Paternal Internalising	0.099	0.067	0.140
Externalising			
Maternal Externalising	0.043	0.015	0.474
Maternal Internalising	-0.027	0.036	0.193
Paternal Externalising	0.006	0.041	0.092
Paternal Internalising	0.022	0.031	0.222

Table A15.

Total effects in the structural equation model for Group Two female participants (n=7,357)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.075	0.106	0.464
Maternal Internalising	-0.092	0.200	0.642
Paternal Externalising	0.072	0.077	0.380
Paternal Internalising	-0.314	0.198	0.112
Externalising			
Maternal Externalising	0.187	0.162	0.040
Maternal Internalising	-0.112	0.285	0.495
Paternal Externalising	0.321	0.118	<.001
Paternal Internalising	0.033	0.274	0.836

Figure A4.

Structural equation model for Group Two male particiants indicating the association between latent dimensions of parental externalising and internalising, early life adversity and latent dimensions of internalising and externalising (n=6,007). Circles represent latent variables and rectangles represent observed variables

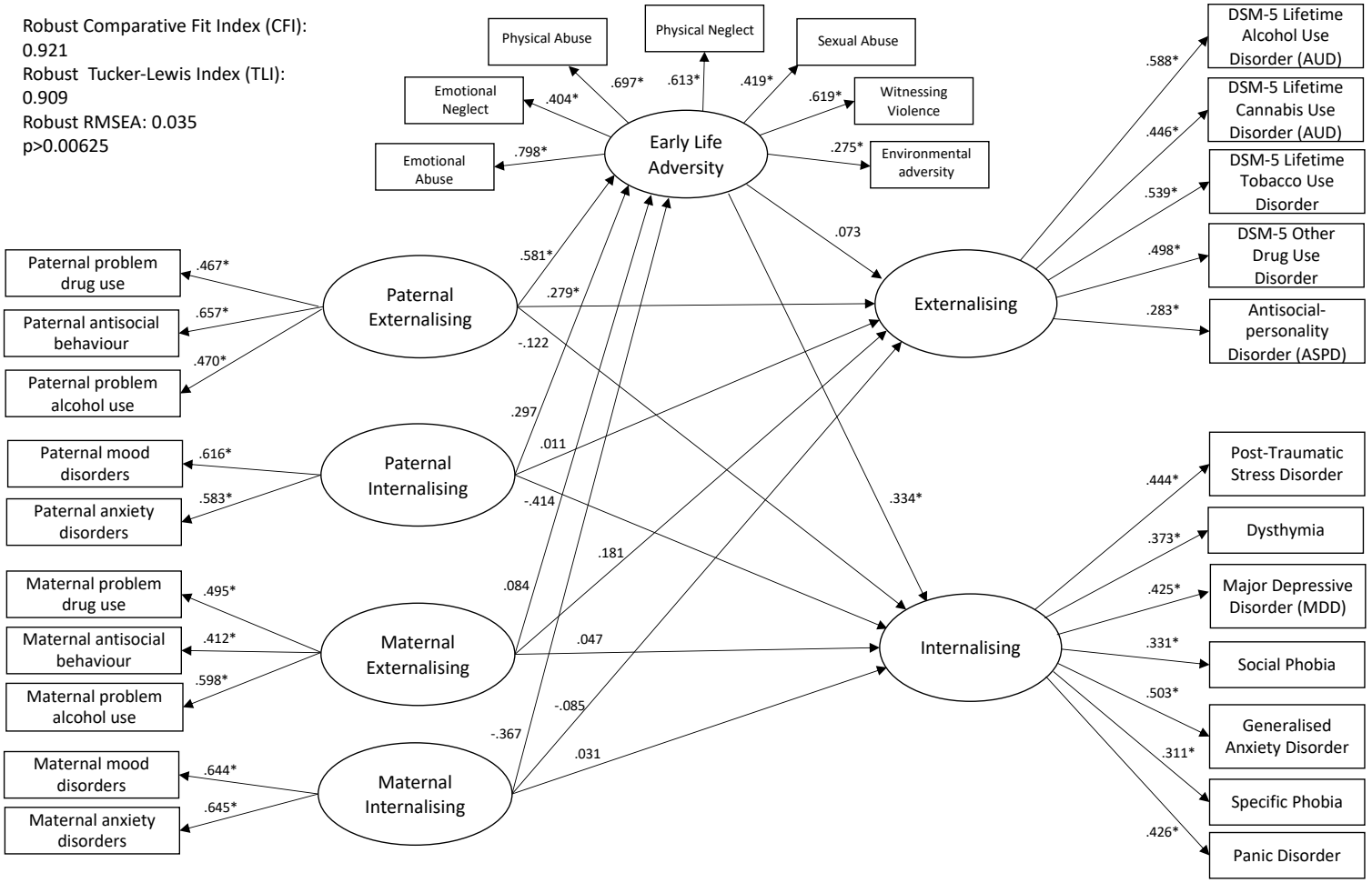

Table A16.

Indirect effects in the structural equation model for Group Two male participants (n=6,007)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.096	0.027	<.001
Maternal Internalising	-0.020	0.020	0.215
Paternal Externalising	0.097	0.024	<.001
Paternal Internalising	0.006	0.714	0.020
Externalising			
Maternal Externalising	0.023	0.015	0.070
Maternal Internalising	-0.005	0.006	0.358
Paternal Externalising	0.023	0.016	0.090
Paternal Internalising	0.001	0.004	0.722

Table A17.

Total effects in the structural equation model for Group Two male participants (n=6,007)

	Estimate	Std.Err	*p* (<.006)
Internalising			
Maternal Externalising	0.033	0.073	0.564
Maternal Internalising	-0.419	0.089	<.001
Paternal Externalising	0.042	0.064	0.403
Paternal Internalising	-0.144	0.087	0.036
Externalising			
Maternal Externalising	0.304	0.076	<.001
Maternal Internalising	-0.070	0.080	0.304
Paternal Externalising	0.210	0.069	<.001
Paternal Internalising	-0.031	0.082	0.662

Appendix B

Code Book

Table B1.

Code book of NESARC variable codes and recoding information

Code	NESARC code	Question	NESARC levels	Recoded?	Recoded levels	Recoded code
id	CASEID	ID number	Type: character	No	-	-
age	NAGE	How old are you as of today?	NA	No	-	-
sex	NSEX	What is your sex?	1=Male 2=Female	No	-	-
Now I'd like to know how true each of the following statements was when you were growing up, that is, BEFORE you were 18 years old.						
eainsult	N13Q1F	How often did a parent or other adult living in your home swear at you, insult you or say hurtful things?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often	eainsultR

			9=Unknown (option not given to respondents)		9=Unknown	
eathreat	N13Q1G	How often did a parent or other adult living in your home threaten to hit you or throw something at you, but didn't do it?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	eathreatR
eafear	N13Q1H	How often did a parent or other adult living in your home act in ANY other way that made you afraid that you would be physically hurt or injured?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	eafearR

ensuccess	N13Q3A	I felt there was someone in my family who wanted me to be a success.	1=Never true 2=Rarely true 3=Sometimes true 4=Often true 5=Very often true *9=Unknown (option not given to respondents)*	Yes	0= Very often true 1= Often true 2= Sometimes true 3= Rarely true 4= Never true 9=Unknown	ensuccessRr
enspecial	N13Q3B	There was someone in my family who helped me feel that I was important or special.	1=Never true 2=Rarely true 3=Sometimes true 4=Often true 5=Very often true *9=Unknown (option not given to respondents)*	Yes	0= Very often true 1= Often true 2= Sometimes true 3= Rarely true 4= Never true 9=Unknown	enspecialRr

ensupport	N13Q3C	My family was a source of strength and support.	1=Never true 2=Rarely true 3=Sometimes true 4=Often true 5=Very often true *9=Unknown (option not given to respondents)*	Yes	0= Very often true 1= Often true 2= Sometimes true 3= Rarely true 4= Never true 9=Unknown	ensupportRr
enclose	N13Q3	I felt that I was part of a close-knit family.	1=Never true 2=Rarely true 3=Sometimes true 4=Often true 5=Very often true *9=Unknown (option not given to respondents)*	Yes	0= Very often true 1= Often true 2= Sometimes true 3= Rarely true 4= Never true 9=Unknown	encloseRr

enbelieve	N13Q3E	Someone in my family believed in me.	1=Never true 2=Rarely true 3=Sometimes true 4=Often true 5=Very often true *9=Unknown (option not given to respondents)*	Yes	0= Very often true 1= Often true 2= Sometimes true 3= Rarely true 4= Never true 9=Unknown	enbelieveRr
phyab	N13Q1I	How often did a parent or other adult living in your home push, grab, shove, slap, or hit you?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phyabR

phyabmark	N13Q1J	How often did a parent or other adult living in your home hit you so hard that you had marks or bruises or were injured?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phyabmarkR
phynsupervision	N13Q1B	How often were you left alone or unsupervised when you were too young to be alone, that is, before you were 10 years old?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phynsupervisionR
phynsupplies	N13Q1C	How often did you go without things you needed like cloths, shoes, or school supplies because a parent or	1=Never 2=Almost never 3=Sometimes	Yes	0=Never 1=Almost Never 2=Sometimes	phynsuppliesR

		other adult living in your home spent the money on themselves?	4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*		3=Fairly Often 4=Very often 9=Unknown	
phynhungry	N13Q1D	How often did a parent or other adult living in your home make you go hungry or not prepare regular meals?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phynhungryR
phynchore	N13Q1A	Before age 18, how often were you made to do chores that were too difficult or dangerous for someone your age?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phynchoreR

			9=Unknown (option not given to respondents)			
phynmedical	N13Q1E	How often did a parent or other adult living in your home ignore or fail to get you medical treatment when you were sick or hurt?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	phynmedicalR
Now I'd like to know if you had any of the following sexual experiences with an adult or any other person BEFORE you were 18 years old. By adult or other person, I mean a parent, stepparent, foster parent, adoptive parent, a relative, friend, family friend, teacher or stranger.						
satouch1	N13Q2A	Before you were 18 years old.... How often did an adult or other person touch or fondle you in a sexual way when you didn't want them to or when you were too young to know what was happening?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	satouch1R

			9=Unknown (option not given to respondents)			
satouch2	N13Q2B	Before you were 18 years old.... How often did an adult or other person have you touch their body in a sexual way when you didn't want to, or you were to young to know what was happening?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Often 4=Very often 9=Unknown	satouch2R
saintercattempt	N13Q2C	Before you were 18 years old.... How often did an adult or other person attempt to have sexual intercourse with you when you didn't want them to or when you were too young to know what was happening?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	saintercattemptR

sainterc	N13Q2D	Before you were 18 years old.... How often did an adult or other person actually have sexual intercourse with you when you didn't want them to or you were too young to know what was happening?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	saintercR
How often did your father, stepfather, foster or adoptive father or mother's boyfriend do ANY of these things to your mother, stepmother, father's girlfriend or your foster or adoptive mother?						
witviolence1	N13Q1K	Push, grab, slap, or throw something at her?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	witviolence1R

witviolence2	N13Q1L	Kick, bite, hit her with a fist, or hit her with something hard?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	witviolence2R
witviolence3	N13Q1M	Repeatedly hit her for a least a few minutes?	1=Never 2=Almost never 3=Sometimes 4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*	Yes	0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very often 9=Unknown	witviolence3R
witviolence4	N13Q1N	Threaten her with a knife or gun or use a knife or gun to hurt her?	1=Never 2=Almost never 3=Sometimes	Yes	0=Never 1=Almost Never 2=Sometimes	witviolence4R

			4=Fairly often 5=Very often *9=Unknown (option not given to respondents)*		3=Fairly Often 4=Very often 9=Unknown	
witsubalc	N13Q4A	Before you were 18 years old, was a parent or other adult living in your home a problem drinker or alcoholic? *(By alcoholic or problem drinking, I mean a person who had physical or emotional problems because of drinking; problems with a spouse, family, or friends because of drinking; problems at work or school because of drinking; problems with police because of drinking- like drunk driving; or a person who seemed to*	1=Yes 2=No *9=Unknown (option not given to respondents)*	Yes	0=No 1=Yes 9=Unknown	witsubalcR

		spend a lot of time drinking or being hung over.)				
witsubdrug	N13Q4B	Before you were 18 years old, did a parent or other adult living in your home have some similar problems with drugs?	1=Yes 2=No *9=Unknown (option not given to respondents)*	Yes	0=No 1=Yes 9=Unknown	witsubdrugR
elaprison	N13Q5	Before you were 18 years old, did a parent or other adult living in your home go to jail or prison?	1=Yes 2=No *9=Unknown (option not given to respondents)*	Yes	0=No 1=Yes 9=Unknown	elaprisonR
elamentalill	N13Q6	Before you were 18 years old, was a parent or other adult living in your home treated or hospitalised for a mental illness?	1=Yes 2=No *9=Unknown (option not given to respondents)*	Yes	0=No 1=Yes 9=Unknown	elamentalillR
elasuicideat	N13Q7	Before you were 18 years old, did a parent or other adult living in your home attempt suicide?	1=Yes 2=No	Yes	0=No 1=Yes 9=Unknown	elasuicideatR

			9=Unknown (option not given to respondents)			
elasuicidesuccess	N13Q8	Before you were 18 years old, did a parent or other adult living in your home actually commit suicide?	1=Yes 2=No *9=Unknown (option not given to respondents)*	Yes	0=No 1=Yes 9=Unknown	elasuicidesuccessR
Now I would like to ask you some questions about whether any of your relatives, regardless or whether they are now living, have EVER been alcoholics or problems drinkers. By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking; problems with a spouse, family or friends because of drinking; problems at work or school because of drinking; problems because of driving after drinking, or a person who seems to spend a lot of time drinking of being hung over.						
dadalc	N2DQ1	Has your blood or natural father been an alcoholic or problem drinking at ANY time in his life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	dadalcR
adadalc	N2DQ14A	Was your adoptive father an alcoholic or problem drinking at ANY time in his life?	1=Yes 2=No	Yes	0=No 1=Yes 9=Unknown	adadalcR

			9=Unknown (option not given to participants)			
mumalc	N2DQ2	has your blood or natural mother been an alcoholic or problem drinker at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	mumalcR
amumalc	N2DQ14B	Was your adoptive mother an alcoholic or problem drinking at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	amumalcR
Now I would like to ask you some further questions about whether your relatives, regardless of whether they are now living, have EVER had problems with drugs. By having problems with drugs I mean a person who has physical or emotional problems because of drug use; problems with a spouse, family or friends because of drug use; problems at work or school because of drug use; problems because of driving under the influence, or a person who seems to spend a lot of time using drugs or getting over their bad aftereffects.						
daddrug	N3EQ1	In your judgement, has your blood or natural father had problems with drugs at ANY time in his life?	1=Yes 2=No	Yes	0=No 1=Yes 9=Unknown	daddrugR

			9=Unknown (option not given to participants)			
mumdrug	N3EQ2	Has your blood or natural mother had problems with drugs at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	mumdrugR
Now I would like to ask you about whether any of your relatives, regardless or whether or no they are now living have ever had behavioural problems (show flashcard 56) By behavioural problems I mean being cruel to people or animals, fighting or destroying property, trouble keeping a job or paying bills, being impulsive, reckless or not planning ahead, lying or conning people or getting arrested. These people also do not seem to care if they hurt others and often have problems at an early age such as truancy, staying out all night or running away.						
mumbeh	N11BQ2	Did your blood or natural mother have some of these behaviour problems like this at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	mumbehR
dadbeh	N11BQ1	In your judgement, did your blood or natural father have some of these	1=Yes 2=No	Yes	0=No 1=Yes	dadbehR

		behaviour problems like this at ANY time in his life?	*9=Unknown (option not given to participants)*		9=Unknown	
Now I would like to ask about whether any of your relatives, regardless of whether or not they are now living, have EVER had a period of feeling anxious or nervous. (FLASHCARD 55). By anxious or nervous I mean times when they were tense, nervous or anxious for at least three months, had panic attacks, were very frightened of objects or situations or avoided them, or had bad reactions to a traumatic or stressful event.						
dadanx	N15AQ1	Was your blood or natural father anxious, nervous or frightened at ANY time in his life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	dadanxR
mumanx	N15AQ2	Was your blood or natural mother anxious, nervous or frightened at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	mumanxR
Now I would like to ask about whether any of your relatives, regardless of whether or not they are now living, have ever been depressed for a period of AT LEAST 2 WEEKS. (FLASHCARD 44) By depressed I mean they felt down, sad, blue or didn't care about things and also ate or slept too little or too much, moved more slowly than usual, were tired or agitated, had trouble concentrating, making decisions or doing things, or felt worthless or thought about suicide.						

dadmood	N4CQ1	Was your blood or natural father depressed at ANY time in his life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	dadmoodR
mummood	N4CQ2	Was your blood or natural mother depressed at ANY time in her life?	1=Yes 2=No *9=Unknown (option not given to participants)*	Yes	0=No 1=Yes 9=Unknown	mummoodR
mdd	lmddisorder	Lifetime DSM-5 major depressive disorder (hierarchical)	0=No 1=Yes	No	-	-
dysthymia	ldysthymia	Lifetime DSM-5 dysthymia (hierarchical)	0=No 1=Yes	No	-	-
specialphobia	lspeind	Lifetime DSM-5 specific phobia	0=No 1=Yes	No	-	-
socialphobia	lsocind	Lifetime DSM-5 social phobia	0=No 1=Yes	No	-	-
panicd	lpanicd	Lifetime DSM-5 panic disorder	0=No	No	-	-

			1=Yes			
agoraphobia	lagoraind	Lifetime DSM-5 agoraphobia	0=No 1=Yes	No	-	-
gad	lgadind	Lifetime DSM-5 generalised anxiety disorder	0=No 1=Yes	No	-	-
ptsd	lptsd	Lifetime DSM-5 posttraumatic stress disorder	0=No 1=Yes	No	-	-
aud	lifeaud5	Lifetime DSM-5 alcohol use disorder	0=No 1=Yes	No	-	-
sedativeud	lsedud5	Lifetime DSM-5 sedative use disorder	0=No 1=Yes	No	-	-
cannabisud	lmaud5	Lifetime DSM-5 cannabis use disorder	0=No 1=Yes	No	-	-
opioidud	lopud5	Lifetime DSM-5 opioid use disorder	0=No 1=Yes	No	-	-
cocaineud	lcocud5	Lifetime DSM-5 cocaine use disorder	0=No 1=Yes	No	-	-

stimulantud	lstiud5	Lifetime DSM-5 stimulant use disorder	0=No 1=Yes	No	-	-
hallucinud	lhalud5	Lifetime DSM-5 hallucinogen use disorder	0=No 1=Yes	No	-	-
inhalantud	lsolud5	Lifetime DSM-5 inhalant/solvent use disorder	0=No 1=Yes	No	-	-
cluddud	lclbud5	Lifetime DSM-5 club drug use disorder	0=No 1=Yes	No	-	-
heroinud	lherud5	Lifetime DSM-5 heroin use disorder	0=No 1=Yes	No	-	-
otherdud	lothud5	Lifetime DSM-5 other drug use disorder	0=No 1=Yes	No	-	-
tobacdep	lnicdep5	Lifetime DSM-5 tobacco use disorder	0=No 1=Yes	No	-	-
aspd	antisoc	DSM-5 antisocial personality disorder	0=No 1=Yes	No	-	-

Appendix C

Analysis Code

```
R1sem<-'
internalising =~ mdd + dysthymia + specificphobia + socialphobia + panicd +
     gad + ptsd
externalising =~ aud + cannabisud + dud + tobacdep + aspd
earlylifeadversity =~ emotionalabuse + emotionalneglect + physicalabuse + physicalneglect +
      sexualabuse + witnessviolence + environmentalela
paternalext =~ dadalcR + daddrugR + dadbehR
maternalext =~ mumalcR + mumdrugR + mumbehR
dadinternal =~ dadanx + dadmood
muminternal =~ mumanx + mummood

#regressions
earlylifeadversity ~  c(a_maternalext_1, a_maternalext_2) * maternalext +
c(a_muminternal_1, a_muminternal_2) * muminternal +
c(a_dadinternal_1, a_dadinternal_2) * dadinternal +
c(a_paternalext_1, a_paternalext_2) * paternalext

internalising ~ c(b_int_1, b_int_2) * earlylifeadversity +
c(c_int_paternalext_1, c_int_paternalext_2) * paternalext +
c(c_int_dadinternal_1, c_int_dadinternal_2) * dadinternal +
c(c_int_maternalext_1, c_int_maternalext_2) * maternalext +
c(c_int_muminternal_1, c_int_muminternal_2) * muminternal

externalising ~ c(b_ext_1, b_ext_2) * earlylifeadversity +
c(c_ext_paternalext_1, c_ext_paternalext_2) * paternalext +
c(c_ext_dadinternal_1, c_ext_dadinternal_2) * dadinternal +
c(c_ext_maternalext_1, c_ext_maternalext_2) * maternalext +
c(c_ext_muminternal_1, c_ext_muminternal_2) * muminternal

#indirect effect
muminternal_int_1 := a_muminternal_1 * b_int_1
muminternal_int_2 := a_muminternal_2 * b_int_2
dadinternal_int_1 := a_dadinternal_1 * b_int_1
dadinternal_int_2 := a_dadinternal_2 * b_int_2
maternalext_int_1 := a_maternalext_1 * b_int_1
maternalext_int_2 := a_maternalext_2 * b_int_2
paternalext_int_1 := a_paternalext_1 * b_int_1
paternalext_int_2 := a_paternalext_2 * b_int_2


muminternal_ext_1 := a_muminternal_1 * b_ext_1
muminternal_ext_2 := a_muminternal_2 * b_ext_2
dadinternal_ext_1 := a_dadinternal_1 * b_ext_1
dadinternal_ext_2 := a_dadinternal_2 * b_ext_2
```

```
maternalext_ext_1 := a_maternalext_1 * b_ext_1
maternalext_ext_2 := a_maternalext_2 * b_ext_2
paternalext_ext_1 := a_paternalext_1 * b_ext_1
paternalext_ext_2 := a_paternalext_2 * b_ext_2


#total effect
total_ext_pat_1 := c_ext_paternalext_1 + paternalext_ext_1
total_ext_pat_2 := c_ext_paternalext_2 + paternalext_ext_2
total_ext_mat_1 := c_ext_maternalext_1 + maternalext_ext_1
total_ext_mat_2 := c_ext_maternalext_2 + maternalext_ext_2
total_ext_dad_1 := c_ext_dadinternal_1 + dadinternal_ext_1
total_ext_dad_2 := c_ext_dadinternal_2 + dadinternal_ext_2
total_ext_mum_1 := c_ext_muminternal_1 + muminternal_ext_1
total_ext_mum_2 := c_ext_muminternal_2 + muminternal_ext_2

total_int_pat_1 := c_int_paternalext_1 + paternalext_int_1
total_int_pat_2 := c_int_paternalext_2 + paternalext_int_2
total_int_mat_1 := c_int_maternalext_1 + maternalext_int_1
total_int_mat_2 := c_int_maternalext_2 + maternalext_int_2
total_int_dad_1 := c_int_dadinternal_1 + dadinternal_int_1
total_int_dad_2 := c_int_dadinternal_2 + dadinternal_int_2
total_int_mum_1 := c_int_muminternal_1 + muminternal_int_1
total_int_mum_2 := c_int_muminternal_2 + muminternal_int_2

#residual correlations
dadinternal ~~ muminternal
paternalext ~~ dadinternal
maternalext ~~ muminternal

#added from the cfa modicationindicies check
emotionalabuse ~~ physicalabuse
daddrugR ~~ mumdrugR
dadbehR ~~ mumbehR
mdd ~~ dysthymia
dadbehR ~~ mumdrugR
dadalcR ~~  mumbehR
emotionalneglect ~~ physicalneglect
dadbehR ~~  mumalcR
emotionalabuse ~~ sexualabuse
dadalcR ~~  mumalcR
physicalneglect ~~ sexualabuse
mumalcR ~~  mumdrugR
emotionalabuse ~~  witnessviolence
'

# constrained model (all regressions constrained)
R1semCON<-'
internalising =~ mdd + dysthymia + specificphobia + socialphobia + panicd +
gad + ptsd
```

```
externalising =~ aud + cannabisud + dud + tobacdep + aspd
earlylifeadversity =~ emotionalabuse + emotionalneglect + physicalabuse + physicalneglect +
sexualabuse + witnessviolence + environmentalela
paternalext =~ dadalcR + daddrugR + dadbehR
maternalext =~ mumalcR + mumdrugR + mumbehR
dadinternal =~ dadanx + dadmood
muminternal =~ mumanx + mummood

#regressions
earlylifeadversity ~  c(a_maternalext_1, a_maternalext_1) * maternalext +
c(a_muminternal_1, a_muminternal_1) * muminternal +
c(a_dadinternal_1, a_dadinternal_1) * dadinternal +
c(a_paternalext_1, a_paternalext_1) * paternalext

internalising ~ c(b_int_1, b_int_1) * earlylifeadversity +
c(c_int_paternalext_1, c_int_paternalext_1) * paternalext +
c(c_int_dadinternal_1, c_int_dadinternal_1) * dadinternal +
c(c_int_maternalext_1, c_int_maternalext_1) * maternalext +
c(c_int_muminternal_1, c_int_muminternal_1) * muminternal

externalising ~ c(b_ext_1, b_ext_1) * earlylifeadversity +
c(c_ext_paternalext_1, c_ext_paternalext_1) * paternalext +
c(c_ext_dadinternal_1, c_ext_dadinternal_1) * dadinternal +
c(c_ext_maternalext_1, c_ext_maternalext_1) * maternalext +
c(c_ext_muminternal_1, c_ext_muminternal_1) * muminternal

#indirect effect
muminternal_int_1 := a_muminternal_1 * b_int_1
dadinternal_int_1 := a_dadinternal_1 * b_int_1
maternalext_int_1 := a_maternalext_1 * b_int_1
paternalext_int_1 := a_paternalext_1 * b_int_1


muminternal_ext_1 := a_muminternal_1 * b_ext_1
dadinternal_ext_1 := a_dadinternal_1 * b_ext_1
maternalext_ext_1 := a_maternalext_1 * b_ext_1
paternalext_ext_1 := a_paternalext_1 * b_ext_1


#total effect
total_ext_pat_1 := c_ext_paternalext_1 + paternalext_ext_1
total_ext_mat_1 := c_ext_maternalext_1 + maternalext_ext_1
total_ext_dad_1 := c_ext_dadinternal_1 + dadinternal_ext_1
total_ext_mum_1 := c_ext_muminternal_1 + muminternal_ext_1

total_int_pat_1 := c_int_paternalext_1 + paternalext_int_1
total_int_mat_1 := c_int_maternalext_1 + maternalext_int_1
total_int_dad_1 := c_int_dadinternal_1 + dadinternal_int_1
total_int_mum_1 := c_int_muminternal_1 + muminternal_int_1
```

```
#residual correlations
dadinternal ~~ muminternal
paternalext ~~ dadinternal
maternalext ~~ muminternal

#added from the cfa modicationindicies check
emotionalabuse ~~ physicalabuse
daddrugR ~~ mumdrugR
dadbehR ~~ mumbehR
mdd ~~ dysthymia
dadbehR ~~ mumdrugR
dadalcR ~~  mumbehR
emotionalneglect ~~ physicalneglect
dadbehR ~~  mumalcR
emotionalabuse ~~ sexualabuse
dadalcR ~~  mumalcR
physicalneglect ~~ sexualabuse
mumalcR ~~  mumdrugR
emotionalabuse ~~  witnessviolence
'

fitR1semCON_l<-lavaan::sem(R1sem, data = R1dt, group = "sex", group.equal=
c("loadings"), estimator = "MLM", std.lv = T)

fitR1semCON_lr<-lavaan::sem(R1semCON, data = R1dt, group = "sex", group.equal=
"loadings", estimator = "MLM", std.lv = T)

summary(fitR1semCON_l, standardized=TRUE, fit.measures=T)

anova(fitR1semCON_l,fitR1semCON_lr)
```