Exposure to early childhood maltreatment and its effect over time on social cognition

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Running Head: Child maltreatment and social cognition

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Abstract

Social cognitive deficits can have many negative consequences, spanning social withdrawal to psychopathology. Prior work has shown that child maltreatment may associate with poorer social cognitive skills in later life. However, no studies have examined this association from early childhood into adolescence. Using data from the Avon Longitudinal Study of Parents and Children (n=4438), we examined the association between maltreatment (caregiver physical or emotional abuse; sexual or physical abuse), assessed repeatedly (every 1-3 years) from birth to age 9, and social cognitive skills at ages 7.5, 10.5, and 14 years. We evaluated the role of both the developmental timing (defined by age at exposure) and accumulation of maltreatment (defined as the number of occasions exposed) using a Least Angle Regression variable selection procedure, followed by structural equation modeling. Among females, accumulation of maltreatment explained the most variation in social cognitive skills. For males, no significant associations were found. These findings underscore the importance of early intervention to minimize the accumulation of maltreatment and showcase the importance of prospective studies to understand the development of social cognition over time.

Introduction

Epidemiological studies suggest that approximately one out of every six young people in the United States and other developed countries worldwide has experienced some type of childhood maltreatment, such as physical abuse, sexual abuse, emotional abuse, or neglect (Finkelhor et al., 2013; Gilbert et al., 2009; Stoltenborgh et al., 2015). The ubiquity of maltreatment exposure is concerning due to the wide ranging negative health outcomes linked to childhood maltreatment, including depression, post-traumatic stress disorder, other psychiatric problems, as well as brain-based structural and functional changes (McLaughlin et al., 2010; Teicher et al., 2003).

Recent evidence also suggests that exposure to childhood maltreatment may lead to difficulties in social cognition abilities. Social cognition refers to the information processing mechanisms underlying the perception, interpretation, and response to social information that drive social interactions (Crick & Dodge, 1994; Green et al., 2005; Piskulic & Addington, 2011; Sergi et al., 2006; Vauth et al., 2004). It is often conceptualized as the psychological processes that allow individuals to benefit from being part of social group, such as human society (Frith, 2008). This includes skills such as being able to recognize others' emotions and infer their mental states via body language and other social cues. Social cognitive deficits, or difficulties in interpreting social cues and situations, can have many negative consequences. For example, prior studies have linked deficits in social cognition to social misperceptions and even social withdrawal (Green et al., 2005; Piskulic & Addington, 2011), as well as poor vocational outcomes stemming from deficits in work-related social skills (Vauth et al., 2004). Of consequence, children who have experienced abuse or maltreatment have been found to have a wide range of social cognition deficits as compared to their non-maltreated peers, including

difficulties comprehending complex social situations, understanding emotions, identifying facial emotions, and imitating multiple roles in social interactions (Barahal, Waterman & Martin, 1981; Luke & Banerjee, 2013). For example, children exposed to institutional caregiving environments were found to have worse social cognition in all domains, but particularly reciprocal social interactions (Levin et al., 2015). Many theorize that social cognitive deficits can occur following exposure to maltreatment, due to maltreatment being linked with insecure attachments, lowered threshold for limbic system reactivity, and other neurobiological changes (Dvir et al., 2014).

Social cognition may play an important role in mental illness, as well. To date, deficits in social cognition abilities have been linked to the etiology, course, and treatment of a wide range of psychiatric disorders, including schizophrenia, autism, post-traumatic stress disorder, depression, and early onset conduct problems (Ladegaard et al., 2014; Oliver et al., 2011; Piskulic & Addington, 2011; Sasson, Nowlin & Pinkham, 2013; Sergi et al., 2006). For example, prior research has shown that among people with schizophrenia, social cognition abilities can predict functional outcome (i.e., ability to work independently and social problem solving) and, when considered with broader cognitive skills (i.e., verbal memory and attention), can explain nearly 80% of the variance in social functioning (Addington et al., 2010; Sergi et al., 2006). Additionally, mentalizing impairment – a subset of social cognition defined as a person's capacity to understand one's own and others' behavior in regards to a mental state – has been shown to partially mediate or explain the association between childhood abuse and negative symptoms in nonaffective psychotic disorder (Weijers et al., 2018). As social cognition is widely implicated in psychiatric disorders, research into the features that predict social cognition problems, including childhood maltreatment, are needed.

However, research on the relationship between childhood maltreatment and social cognition has been limited in three important ways. First, studies of maltreated children have often assessed social cognition deficits in adulthood – and not throughout childhood and adolescence, when social cognitive skills are developing (Happé & Frith, 2014). In particular, few studies have examined social cognition during middle childhood, when important gains in social perspective taking are made (Bosacki, 2000; Van Der Graaff et al., 2014). This is a shortcoming as social cognition is not a fixed state during early life, but rather the result of an ongoing neurodevelopmental process that continues throughout childhood and adolescence. The capacity to mentalize – or understand the mental state of others – develops in the first five years of life (Frith & Frith, 2007). By age four, most children have developed the understanding that others may hold beliefs that are different from their own and that other's beliefs can be untrue (Barresi & Moore, 1996; Kilford, Garrett & Blakemore, 2016). During adolescence, brain structures important for social cognition, including grey matter density in the superior temporal lobe, undergo rapid development and change (Burnett et al., 2011; Mills et al., 2014; Vetter et al., 2013). Previous work has indicated that social cognition may be highly plastic and malleable depending on the social network and social experiences of the child across time and development (Cabrera et al., 2011; Ford, Clark & Stansfeld, 2011; Happé & Frith, 2014). Although there are a number of longitudinal studies of social cognition, few of these start with children; of these, none to our knowledge examine repeated measures of social cognition throughout childhood (Magiati, Tay & Howlin, 2014). These shortcomings exist despite evidence suggesting that the foundations of social cognitive skills are gained in very early life (Happé & Frith, 2014; Mills et al., 2014). To address these gaps, prospective and longitudinal studies are needed that include repeated,

standardized measures to capture the development of social cognition beginning in infancy throughout childhood and adolescence.

Second, although childhood maltreatment experiences have been linked to social cognition deficits, including emotional regulation problems (Dvir et al., 2014), there is limited knowledge of how certain features of these maltreatment experiences predict social cognition. For example, little is known whether the developmental timing of occurrence and the number of times a child is exposed to maltreatment shapes their social cognition, or whether being exposed to maltreatment, regardless of these timing aspects, is more important. Thus, efforts are needed to statistically evaluate hypotheses described by life course theory (Ben-Shlomo & Kuh, 2002; Kuh et al., 2003), including those focused on sensitive periods, accumulation, ever-exposed models, respectively. Studies on a host of other psychosocial outcomes, such as depression risk, psychosis risk, lower ego resiliency, and neurocognitive functioning (Barahal et al., 1981; Dale et al., 2010; Dunn et al., 2018; Luke & Banerjee, 2013; Manly et al., 2001) have found that the effects of childhood maltreatment, and of adversity more broadly, can vary considerably depending on the timing and accumulation of abuse exposures. Indeed, there is evidence to suggest that there may be sensitive periods when the developing brain is particularly susceptible to these adverse social experiences (Dunn et al., 2013; Dunn et al., 2017). Further, prior work has also demonstrated the importance of exposure timing in predicting neurophysiology, neurobiological structure, and broader cognitive functioning (Anderson et al., 2016; Bosch et al., 2012; Cicchetti et al., 2015). Given this growing body of evidence to support time-dependent effects of maltreatment, there is a need for research that accounts for not only changes in social cognition over time, but also the temporal features of maltreatment exposure across childhood. Such investigations will allow researchers to evaluate the effects of different theories and

106 determine which theoretical models alone or in combination best explain child development
107 outcomes.

108 Finally, there is a distinct lack of population-based social cognition research, with the
109 majority of prior studies being conducted in clinical cohorts and other highly selected or
110 homogeneous samples (e.g. college undergraduates). While social cognition has been an
111 excellent predictor of functional outcomes (e.g. vocational functioning) in previous studies using
112 clinical samples (e.g. people diagnosed with schizophrenia), these findings have not been
113 generalized to typically-developing populations. Efforts to expand work on the links between
114 maltreatment exposure and social cognition to more representative samples can improve
115 reproducibility and generate results that are more widely generalizable. Moreover, a focus on
116 triangulation, where multiple approaches are used to address the same research question –
117 including studying different population groups – can yield results that are less likely to be driven
118 by methodological artifacts and are closer to the underlying truth (Munafò & Davey Smith,
119 2018).

120 The current study aimed to address these gaps and expand upon prior literature by
121 prospectively examining the relationship between exposure to childhood maltreatment and social
122 cognition deficits. Data came from a large, population-based sample of children followed from
123 infancy to adolescence called the Avon Longitudinal Study of Parents and Children (ALSPAC).
124 Our aims were to: (1) determine how exposure to different types of maltreatment between birth
125 and age 9 was associated with the development of social cognition skill deficits from ages 7.5
126 through 14; and (2) evaluate the effects of the different life course models in relation to these
127 later social cognition skill deficits. Specifically, the three life course models test were: (1) a
128 sensitive period model (Ben-Shlomo & Kuh, 2002), in which the effect of maltreatment depends

on the developmental time period of the exposure; (2) an accumulation model (Evans, Li & Whipple, 2013), in which the effect of maltreatment increases with the number of occasions exposed, regardless of timing; and (3) an ever-exposed model, in which the effect of maltreatment does not depend on the timing or amount of exposure, but rather its presence or absence.

Methods

Sample and Procedures

ALSPAC is a prospective, longitudinal birth-cohort of children born to pregnant mothers living in the county of Avon England, which is 120 miles west of London (Boyd et al., 2013; Fraser et al., 2013). ALSPAC was designed to generate new knowledge on the pathways to health across the lifespan, with a focus on genetic and environmental determinants. Women residing in Avon, UK who gave birth between 1st April 1991 to 31st December 1992 were recruited through media advertisements and visits by research staff at multiple sites within the community. The study was also advertised at routine antenatal and maternity health services appointments. Approximately 85 percent of eligible pregnant women agreed to participate (N=14,541), and 76% of eligible live births (N=14,062) who were alive at 12 months of age (N=13,988 children) were enrolled. An additional 913 children who would have been eligible, but whose mothers did not choose to participate during pregnancy, were enrolled after age 7 years. Response rates to data collection have been good (75% have completed at least one follow-up). Compared to the general population in the UK based on the 1991 Census, mothers enrolled in the ALSPAC were more likely to be White, married, and home-owners. However, despite having generally higher socioeconomic positions than non-ALSPAC enrolled women,

they were more likely to live in overcrowded conditions (Fraser et al., 2013). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committee. The ALSPAC website contains details of all the data that is available through a fully searchable data dictionary and variable search tool: http://www.bristol.ac.uk/alspac/researchers/our-data/.

The current analysis was based on an analytic sample of 4438 children (out of a possible 9677 children with one measure of social cognition) who met three inclusion criteria. First, we restricted our analytic sample to singleton births to prevent confounding associated with the unique social structure and support that multiple-birth children can have in the home (Lang, Cox & Flores, 2013; Prino et al., 2016). Second, the measurement of social cognition came from mailed questionnaires, and we further restricted the sample to only those children whose caregivers had completed these questionnaires for all of the first three timepoints (ages 7.5, 10.5 and 14 years) when social cognition was measured. Finally, to ensure a consistent reporter of child social cognition across time, we restricted the sample to only those children who had mothers and maternal figures as the sole reporters of their social cognition skills over the three timepoints of assessment. As expected, given attrition patterns in the ALSPAC, children in the included sample (n=4438) were slightly more socio-demographically advantaged as compared to children in the excluded sample who had at least one social cognition measure (n=5239) (**Supplemental Table 1**). We additionally compared the distribution of covariates and outcome scores between the analytic sample (n=4438) and the subset of excluded participants with complete social cognition outcome data at all three time points, but who lacked consistent maternal reports (n=688). These two samples had largely similar sociodemographic characteristics and social cognition scores at all three time points; however, the excluded sample

(without consistent maternal reports) were more likely to be born to mothers with slightly higher education (**Supplemental Table 2**).

Measures

Exposure to Child Maltreatment

We examined two types of child maltreatment, measured using mailed questionnaires. Each maltreatment type was measured on seven occasions before age 10; the time frame covered by each assessment varied, with an average duration of 19 months (**Table 1; Supplemental Table 3).**

Caregiver physical or emotional abuse. Children were coded as having been exposed to physical or emotional abuse if the mother, partner, or both responded affirmatively to any of the following items: (1) "Your partner was physically cruel to your children"; (2) "You were physically cruel to your children"; (3) "Your partner was emotionally cruel to your children"; (4) "You were emotionally cruel to your children". Physical abuse and emotional abuse items were examined together, rather than separately, given their strong correlation overall (average correlation=0.55) and to ensure an adequate sample size for these analyses given the rarity of abuse exposure in this population-based sample (**Supplemental Table 4**).

Sexual or physical abuse. Exposure to sexual or physical abuse was determined through an inventory asking the mother to indicate whether or not the child had been either "sexually abused" or "physically hurt by someone". If the mother indicated the child had been exposed to either physical or sexual abuse, the child was coded as having experienced abuse. Due to low prevalence of sexual abuse in the sample, under 10 cases per timepoint, sexual and physical abuse were considered in the same measure; the tetrachoric correlation between any exposure to

physical abuse before age 10 and any exposure to sexual abuse before age 10 was moderate ($r_{tetrachoric} = 0.39$).

Variable encoding. For each type of maltreatment, we generated three sets of encoded variables: (1) a single variable denoting the total number of time periods of exposure to each type of maltreatment, to test the accumulation hypothesis (coded as 0-6); (2) a set of variables indicating presence vs. absence of each type of maltreatment at a specific developmental stage, to test the sensitive period hypothesis; and (3) a single variable denoting whether the child had ever experienced exposure to a specific type of maltreatment regardless of timing or number of exposures, to test the exposure hypothesis.

Social Cognition

Social cognition skills were measured using the Social Communication Disorder Checklist at ages 7.5, 10.5, and 14 years. This 12-item measure was designed to capture the main features of individual social cognition ability, as reported by caregivers, and was originally developed and created for children with Turner's syndrome, which is a disorder characterized by social adjustment problems (Skuse et al., 1997). Sample items included "Child did not realize when others were upset/angry," "Child was very demanding of other people's time," and "Child did not pick up on body language". Response options to each item on this questionnaire were: 1= not true, 2= quite true or sometimes true, and 3= very or often true and were asked in relation to the child's behavior over the past 6 months. These responses were summed to create a total score, with higher scores indicating more social cognition difficulties. The Social Communication Disorder Checklist has demonstrated excellent internal consistency reliability

overall α=0.98 (Skuse et al., 1997), and in our analytic sample (age 7.5 α=0.79; age 10.5 α=0.73; age 14 α=0.80).

Covariates

All multivariable analyses controlled for the following covariates, measured at the time of the child's birth: child race/ethnicity; maternal marital status; highest level of maternal education; maternal age; homeownership status; parent social class; number of previous pregnancies; and levels of maternal depression, as assessed by the Edinburgh Postnatal Depression Scale (EPDS) (Cox, Holden & Sagovsky, 1987) (see **Supplemental Materials**). Covariates were selected for inclusion because they were found to be potential confounders in our sample (see **Supplemental Table 1)**, or because they have been included routinely in longitudinal birth-cohorts when studying child outcomes (Hibbeln et al., 2007; Oliver et al., 2011).

Analyses

We began by running univariate and bivariate analyses to examine the distribution of covariates and exposure to maltreatment in the total analytic sample. We then used a two-stage structured lifecourse modeling approach (SLCMA) (Dunn et al., 2018; Smith et al., 2016; Smith et al., 2015) to evaluate the extent to which the temporal characteristics of maltreatment were associated with deficits in social cognition. For these analyses, we tested – for each type of maltreatment – three lifecourse theoretical models to determine which one explained the most outcome variability (i.e. r^2) (Ben-Shlomo & Kuh, 2002). The major advantage of the structured approach relative to other methods, including standard multiple regression, is that it provides an

unbiased way to compare multiple competing theoretical models simultaneously and identify the most parsimonious explanation for the observed outcome variation (see **Supplemental Materials and Supplemental Figure 1** for more details). These analyses were performed so that each lifecourse theoretical model was tested for both types of maltreatment at each time point when social cognition was measured (age 7.5, 10.5, and 14).

Given that the final maltreatment measurement time point (at age 8/9) occurred after the first social cognition measurement time point (at age 7.5), the theoretical models tested to explain social cognition at age 7.5 did not include maltreatment exposure at age 9 as a predictor. Maltreatment exposure at age 9 was included in the analyses examining social cognition at 10.5 years and 14 years, allowing us to examine the links between maltreatment (between ages 0-9) on social cognition over almost a decade-long period **(Figure 1)**.

We conducted all analyses using a multiply imputed dataset to reduce potential bias and minimize loss of power due to attrition (see **Supplemental Materials**). All analyses were stratified by sex, given previous literature documenting sex differences in exposure to childhood maltreatment (Briere & Elliott, 2003) and social cognition development (Gur et al., 2012).

After selecting the life course theoretical models in the first stage of the SLCMA that explained the most outcome variability, we then performed a linear regression of the theoretical model chosen in the second stage of the SLCMA within each of the 20 multiply imputed datasets and calculated pooled effect estimates (regression coefficients) across datasets using Rubin's rules (Rubin, 1987; van Buuren & Groothuis-Oudshoorn, 2011). We used the p-value from the covariance test to calculate unbiased confidence intervals for the effect estimates (Lockhart et al., 2014; Smith et al., 2015).

Finally, we wanted to evaluate how well the theoretical models selected in the SLCMA analyses fit the social cognition data across multiple timepoints and how these measurements related to one other. To do this, we used structural equation modeling (SEM), which allowed us to further explore within a single analysis how maltreatment exposure was associated with social cognition across timepoints. In SEM, one or more measurement models – describing the relationships between latent factors and observed indicators – are joined together in a structural model, where associations between latent variables, covariates, and observed variables are estimated (Kline, 2010). We modeled the effects of sexual or physical abuse only in females becausethis model showed the strongest effect estimates in the SLCMA results and analyses of males would likely be uninformative, for reasons described later. Our hypothesis based on the SLCMA results was that the accumulation of sexual or physical abuse from 18 months to 6.75 years would predict social cognition at age 7.5, which in turn would predict social cognition at age 10.5, and subsequently social cognition at age 14. We also hypothesized based on the SLCMA results that the accumulation of sexual or physical abuse from 18 months to 8 years would also independently predict social cognition at 14 years (**Figure 2**). To evaluate goodness-of-fit, we used standard SEM fit statistics, including the normed comparative fit index (CFI) (Bentler, 1990), Tucker-Lewis index (TLI) (Tucker & Lewis, 1973), and root mean square error of approximation (RMSEA) (Steiger, 1990). We conducted these SEM analyses using the lavaan package in R 3.3.2 (Yves, 2012).

Results

Sample Characteristics

The analytic sample was gender-balanced (49.71% female) and comprised of predominantly white (97.1%) children from families whose parents were married and owned their home (**Supplemental Table 1**).

Distribution of Exposure to Maltreatment and Social Cognition Skills

Over a quarter of the analytic sample (27%; n=1182) were exposed to at least one type of maltreatment, with 17.44% exposed to physical or emotional abuse, 13.29% exposed to sexual or physical abuse, and 5% exposed to both types.

Exposure to physical or sexual abuse was patterned by child sex (with males being more frequently exposed to either type) and exposure to physical or emotional abuse was patterned by several socio-demographic factors, including maternal material status, home ownership, and number of previous pregnancies ($p<0.01$ for all chi-squared tests comparing children who were exposed to maltreatment to the entire analytic sample; **Supplemental Table 1**). Specifically, children who were exposed to maltreatment were less socioeconomically advantaged and less likely to be firstborn.

Age at exposure to maltreatment varied by type, with males having somewhat of an increase in sexual or physical abuse exposure as they aged. Alternatively, females experienced more physical or emotional abuse at preschool ages with more constant levels of exposure to sexual or physical abuse (**Table 1**).

Within each maltreatment type, exposures were highly correlated over time (**Supplemental Table 5**), with neighboring timepoints being generally more highly correlated than distant timepoints. Physical or emotional abuse by a caregiver had the highest correlation

(r=0.73) between ages 2.75 and 4 years, and sexual or physical by anyone had the highest correlation (r=0.59) between ages 6.75 and 8 years.

Social cognition skills scores were moderately correlated across measurement timepoints (r=0.57-0.67). Notably, mean social cognition skill levels decreased at age 10.5 in both males and females, though significant sex differences were observed across all time points (**Supplemental Table 6**).

Association between Maltreatment and Social Cognition

Results of the SLCMA analysis suggested a different patterning of associations between maltreatment and social cognition based on sex and the type of maltreatment (**Table 2**).

Among females, accumulation was the life course theoretical model consistently selected as the best-fitting one for both types of maltreatment. However, the effects of accumulation were only statistically significant at ages 7.5 and 14 for sexual or physical abuse (ß=0.66, $p<0.01$ and ß=0.65, $p<0.01$, respectively) and age 10.5 (ß=0.27, $p=0.01$) for physical or emotional abuse by a caregiver, with less than 1% of the variance in social cognition explained by the accumulation of each type of maltreatment.

For males, no life course theoretical model achieved statistical significance between exposure to sexual or physical abuse and social cognition. However, an ever-exposed model, was selected as the best fitting model for exposure to physical or emotional abuse on social cognition at age 7.5 (ß=0.86, $p<0.01$). This effect, however, was not observed at the later measurements.

Of note, this general pattern of results was similar after winsorizing the social cognition score values to the 90th percentile, which reduced the effects of extreme scores (**Supplemental Table 7**).

Structural Equation Modeling (SEM)

Building from these results, we used SEM to examine the effect of sexual or physical abuse on females' social cognition across time using the theoretical models identified by the SLCMA. SEM modeling demonstrated the goodness of fit of our hypothesis: the main structural model (**Figure 2**) fit the data adequately (RMSEA=0.09; 95% CI=0.08,0.10; CFI=0.98; TLI=0.95), suggesting that sexual or physical abuse in early life explained lower social cognition scores later in life, via influences on earlier social cognition. Accumulation of sexual or physical abuse from 18 months to 6.75 years predicted social cognition at age 7.5 years and accumulation of sexual or physical abuse from 18 months to 8 years as well as social cognition at age 10.5 predicted social cognition at 14 years. We also saw that social cognition scores at 7.5 years predicted social cognition scores at age 10.5. That is, abuse accumulated between 18 months and 6.75 years of age was associated with social cognition at 7.5 years (ß =0.83, $p<0.01$), which in turn was associated with social cognition at 10.5 years (ß=0.64, $p<0.01$), and subsequently linked to social cognition at 14 years (ß=0.68, $p<0.01$). Social cognition at 14 years was predicted by abuse accumulated between 18 months and 8 years (ß= 0.13, $p<0.01$). Modification indices did not reveal any additional plausible paths between accumulation and the social cognition outcome that would improve model fit. The results of our initial SLCMA testing indicated that for males, the SEM test would unlikely be significant; unlike females, males did not have the same life course theoretical models selected for multiple time points of exposure.

Secondary Analyses

A primary hypothesis tested in this paper is that childhood maltreatment predicts future social cognitive skills. However, children with poor social cognitive skills may also be more

likely than their peers to be exposed to child maltreatment. To explore this possibility, we performed a secondary analysis to examine the association between social cognition and child maltreatment (see **Supplemental Materials**). Results suggested that poorer earlier social cognition skills were generally associated with *lower* levels of exposure to maltreatment (**Supplemental Table 8**).

Discussion

The current study used data from a large, population-based sample of children to examine associations between two types of child maltreatment and the development of subsequent social cognition skills from late childhood through early adolescence. Three main findings emerged from this work. First, childhood maltreatment differentially impacted males and female's social cognition development. Specifically, there were sex differences both in how the characteristics of maltreatment associated with social cognition and the strength of these associations. This sex-dependent pattern of findings was unsurprising in light of prior work showing differences between male and female social cognitive skills between the ages of 8 and 21 (Gur et al., 2012). Our findings are consistent with prior findings in social cognitive development, where females have been shown to outperform males in facial emotion recognition through age 16 (Lawrence, Campbell & Skuse, 2015) and have increased social affect through early development (Messinger et al., 2015). Social cognitive skills in males typically develop later than females and thus we may see a smaller effect size of maltreatment when measuring early time points in males. Thus, we postulate that males and females may be similarly impacted by maltreatment, but that for males, such effects may not appear until later in development. Based on previous work, these effects of maltreatment on social cognition for males may not emerge until late in teenage years or emerging adulthood, as boys begin to catch-up in social cognitive skills with

their female counterparts (Lawrence et al., 2015; Rose & Rudolph, 2006; Van Der Graaff et al., 2014).

Second, our results from the SLCMA suggest that more than any other feature of maltreatment, the accumulation of abuse plays the biggest role in explaining variations in social cognition development specifically for females. In females, the number of time periods exposed to physical or sexual abuse from 0-8 years was positively and linearly associated with social cognition deficits. While prior literature has primarily characterized the effects of the severity of abuse, with more severe abuse leading to greater social cognitive deficits, this is the first study to our knowledge to focus specifically on the number of time periods exposed in relation to social cognition abilities (Barahal et al., 1981; Luke & Banerjee, 2013). Such comparisons are likely to be informative for guiding the development of interventions to combat the negative consequences of exposure to abuse.

Third, our results from the SLCMA and SEM analyses, pertaining to physical or sexual maltreatment, not only emphasize the power of integrating different statistical models, but also expand upon previous work by suggesting that the duration of abuse and its effects on social cognition may persist from childhood to adolescence. Our findings are consistent with prior studies, which have examined the effect of abuse on social cognition during later life, and further suggest that the effects of adversity on social cognition can be observed shortly after abuse and may quickly lead to persistent differences in social cognition (Ford et al., 2011; Germine et al., 2015; Palmier-Claus et al., 2016). These findings emphasize the importance of early identification and intervention efforts to reach children, particularly girls, while these social cognitive deficits are developing, which would be expected to minimize any long-term repercussions of being exposed to adversity. Of note, our results are most likely driven by

400 physical, rather than sexual abuse, due to the former being more common than the latter; this
401 should be considered when interpreting results of this study. Though the effect size was small
402 and there are likely other factors that have not been captured here that shape social cognitive
403 development, our finding strengthen past research and help to inform future work on this topic.
404 Yet, two major questions also emerged from this analysis. The first is: Why were there no
405 significant effects of maltreatment on social cognition in males? Prior studies have shown that
406 males lag substantially behind their female counterparts during the initial development of
407 empathy (Rose & Rudolph, 2006; Van Der Graaff et al., 2014), which is one of the key social
408 cognition skills developed during adolescence. Many items included in our social cognition
409 measure capture experiences that involve empathy skills, including those asking about
410 appropriate responses to other's emotions. Our finding that males had significantly poorer social
411 cognition scores throughout time may suggest that our social cognition measure was capturing a
412 domain that would both be immature in males and unlikely to be affected by maltreatment. We
413 further speculate that the development of social cognition in males may be less stable and more
414 variable in late childhood and adolescence, given previous work noting dramatic changes in
415 social behavior and interaction among males around puberty, such as increased antisocial and
416 aggressive behaviors (Forbes & Dahl, 2010; Rowe et al., 2004). Thus, it might be possible to see
417 more expected adverse effects of maltreatment on social cognitive development reemerge once
418 puberty ends.
419 A second question was: Why do we see a shift in social cognition scores across time and
420 further, why are there differences confined to age 10.5 for the effects of sexual and physical
421 abuse on social cognition? Univariate analyses revealed that for both males and females, social
422 cognition scores worsened considerably at age 10.5. Moreover, among females, a model of

exposure to physical or emotional abuse was selected as explaining a significant amount of variation in social cognition at 10.5, whereas no theoretical models were selected for the other outcome timepoints in the SLCMA modeling. These results are consistent with prior research suggesting that key social cognition skills, such as facial recognition, temporarily plateau or even decline in early adolescence (Carey & Diamond, 1977; Carey, Diamond & Woods, 1980; Germine, Duchaine & Nakayama, 2011). Thus, our measure of social cognition obtained at age 10.5 may fall within an expected aberrant time period, which could account for the unique pattern of results observed during this outcome time point.

There are several strengths of this study. Data came from a large, population-based prospective study. Previous work has tended to only include a single measurement of social cognition, involve retrospective maltreatment reports that do not include repeated measures, or focus on social cognition in clinical samples (e.g. children with autism or Turner's syndrome). To our knowledge, this is the first prospective, population-based study. The use of a prospective study was a particular strength, as it provided a stronger test of our research questions (relative to retrospective or cross-sectional work), and it enabled us to better contextualize these results and identify possibly causal relations. For example, as shown through our secondary analyses, we were able to learn that poorer social cognition skills could also predict lower levels of future exposure to maltreatment. Moreover, our use of a theory-driven analytic method (SCLMA) in combination with SEM was another major innovation. Sample code for implementing the SLCMA is publicly available through a GitHub page (https://github.com/thedunnlab/SLCMA-pipeline).

Some limitations must also be considered. First, the ALSPAC dataset comprised largely children of European-ancestry. Thus, our findings may not be generalizable to more racially and

ethnically diverse populations. Second, the use of maternal self-report questionnaires to capture abuse may be problematic. Mothers may have underreported their child's exposure to abuse, particularly if mothers were implicated in the maltreatment or the maltreatment events occurred outside the home. Reliance on these subjective reports was also likely further complicated because ALSPAC did not provide reporters with clear guidelines or criteria to define these abuse experiences. Thus, some reporters could have downplayed their maltreating behavior and not identified it as abuse, whereas other reporters could have responded to these items considering behaviors that might not fit the traditional definition of maltreatment. Yet, even with these limitations, we found that the prevalence of maltreatment in this sample was comparable to nationally representative samples which use social service or other agency reporting (Gilbert et al., 2009). These similarities in prevalence estimates of maltreatment between our sample and nationally representative samples provides some confidence that our measures may be capturing true childhood maltreatment cases in this sample. Moreover, the use of maternal reporting could also lead to bias in social cognitive scoring, as mother's may expect boys to be less social than girls. Although there were sex differences in social cognition scores here, the magnitude of these differences were similar to those previously reported (Erwin et al., 1992; Gur et al., 2012; Williams et al., 2009). However, previous work on the role of parental bias in social skills suggests that parents identify more social skill problems than children, but are less able to identify subtle symptoms such as withdrawal (Howells Wrobel & Lachar, 1998). Some research even suggests parental reports may be more accurate than child self-reports for social behaviors (Fisher, Mello, & Dykens, 2014). Third, the measures of maltreatment lacked details to characterize the specific nature of the abuse, including its severity. This has been a limitation of other large-scale epidemiological studies when examining abuse. However, by having the

measurement of frequency of maltreatment (defined here as the number of occasions on which it was reported), we can distinguish between children who were exposed only a single time from those who were exposed multiple times, which can help clarify the effect of different maltreatment experiences (Dunn et al., 2018). In future studies, more detailed assessments could prove valuable to document more specific effects of maltreatment features on social cognition and build upon these findings. Fourth, we modeled childhood adversity as an exposure that may impact social cognition; however, our results are only correlational and do not rule out the possibility that preliminary manifestations of poor social cognition in early childhood could also increase risk of maltreatment, which we explored briefly in our secondary analyses. Fifth, as with any longitudinal study, there was attrition over time. Although we attempted to address this attrition by using multiple imputation, the fluctuation in participants reporting outcomes across different outcome timepoints and source of reporting led to smaller sample sizes, since we only analyzed children with maternal reports at all three timepoints. However, we think the conservative exclusion criteria were necessary to minimize potential bias induced by heterogeneity in the mode of reporting. Removing participants who were missing the necessary longitudinal data for our analyses may exclude higher-risk children and/or limit broader generalizability. However, the included sample was comparable to the sample of participants who were excluded. Further work targeting the understanding of reporting patterns in social cognition research could guide the interpretation of our results as well as sample selection of future studies.

In summary, our results suggest that the accumulation of sexual or physical abuse in early childhood can have a lasting impact on female's social cognition skills. These findings underscore the need for further work to measure how maltreatment effects can persist across time

and shape long-term developmental trajectories. These results also emphasize the importance of identifying females who have experienced maltreatment, who may be at risk for having social cognitive challenges.

References

Addington, J., Girard, T. A., Christensen, B. K. & Addington, D. (2010). Social cognition mediates illness-related and cognitive influences on social function in patients with schizophrenia-spectrum disorders. *Journal of psychiatry & neuroscience : JPN 35*(1), 49.

Anderson, C. M., Ohashi, K., Khan, A. & Teicher, M. (2016). Type and Timing of Childhood Maltreatment and Area and Volume of Ventromedial Prefrontal Cortex and Dorsal Anterior Cingulate. *Biological psychiatry 79*(9), 108S-S.

Barahal, R. M., Waterman, J. & Martin, H. P. (1981). The social cognitive development of abused children. *Journal of Consulting and Clinical Psychology 49*(4), 508-16.

Barresi, J. & Moore, C. (1996). Intentional relations and social understanding. *Behavioral and Brain Sciences 19*(1), 107-22.

Ben-Shlomo, Y. & Kuh, D. (2002). A life course approach to chronic disease epidemiology: conceptual models, empirical challenges, and interdisciplinary perspectives. *International Journal of Epidemiology 31*, 285-93.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin 107*, 238-46.

Bosch, N. M., Riese, H., Reijneveld, S. A., Bakker, M. P., Verhulst, F. C., Ormel, J. & Oldehinkel, A. J. (2012). Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. The TRAILS study. *Psychoneuroendocrinology 37*(9), 1439-47.

Boyd, A., Golding, J., Macleod, J., Lawlor, D. A., Fraser, A., Henderson, J., Molloy, L., Ness, A., Ring, S. & Davey Smith, G. (2013). Cohort Profile: the 'children of the 90s'--the index offspring of the Avon Longitudinal Study of Parents and Children. *Int J Epidemiol 42*(1), 111-27.

Briere, J. & Elliott, D. M. (2003). Prevalence and psychological sequelae of self-reported childhood physical and sexual abuse in a general population sample of men and women. *Child abuse & neglect 27*(10), 1205-22.

Burnett, S., Sebastian, C., Cohen Kadosh, K. & Blakemore, S.-J. (2011). The social brain in adolescence: Evidence from functional magnetic resonance imaging and behavioural studies. *Neuroscience and Biobehavioral Reviews 35*(8), 1654-64.

Cabrera, N. J., Fagan, J., Wight, V. & Schadler, C. (2011). Influence of Mother, Father, and Child Risk on Parenting and Children's Cognitive and Social Behaviors. *Child Development 82*(6), 1985-2005.

Carey, S. & Diamond, R. (1977). From Piecemeal to Configurational Representation of Faces. *Science 195*(4275), 312-4.

Carey, S., Diamond, R. & Woods, B. (1980). Development of face recognition: A maturational component? *Developmental Psychology 16*(4), 257-69.

Cicchetti, D. (Ed.) (2016). *Developmental Psychopathology.* John Wiley & Sons, Inc.

Cicchetti, D., Cowell, R. A., Rogosch, F. A. & Toth, S. L. (2015). Childhood maltreatment and its effect on neurocognitive functioning: Timing and chronicity matter. *27*(2), 521-33.

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

Crick, N. R. & Dodge, K. A. (1994). A review and reformulation of social information-processing mechanisms in children's social adjustment. *Psychological Bulletin 115*(1), 74.

Dale, C. L., Findlay, A. M., Adcock, R. A., Vertinski, M., Fisher, M., Genevsky, A., Aldebot, S., Subramaniam, K., Luks, T. L., Simpson, G. V., Nagarajan, S. S. & Vinogradov, S. (2010). Timing is everything: neural response dynamics during syllable processing and its relation to higher-order cognition in schizophrenia and healthy comparison subjects. *Int J Psychophysiol 75*(2), 183-93.

Dunn, E. C., McLaughlin, K. A., Slopen, N., Rosand, J. & Smoller, J. W. (2013). Developmental timing of child maltreatment and symptoms of depression and suicidal ideation in young adulthood: results from the National Longitudinal Study of Adolescent Health. *Depression and Anxiety 30*(10), 955-64.

Dunn, E. C., Nishimi, K., Powers, A. & Bradley, B. (2017). Is developmental timing of trauma exposure associated with depressive and post-traumatic stress disorder symptoms in adulthood? *Journal of Psychiatric Research 84*, 119-27.

Dunn, E. C., Soare, T. W., Raffeld, M. R., Busso, D. S., Crawford, K. M., Davis, K. A., Fisher, V. A., Slopen, N., Smith, A., Tiemeier, H. & Susser, E. S. (2018). What life course theoretical models best explain the relationship between exposure to childhood adversity and psychopathology symptoms: recency, accumulation, or sensitive periods? *Psychological Medicine 48*(15), 2562-72.

Dvir, Y., Ford, J. D., Hill, M. & Frazier, J. A. (2014). Childhood maltreatment, emotional dysregulation, and psychiatric comorbidities. *Harvard Review of Psychiatry 22*(3), 149-61.

Evans, G. W., Li, D. & Whipple, S. S. (2013). Cumulative risk and child development. *Psychological Bulletin 139*(1), 342-96.

Finkelhor, D., Turner, H. A., Shattuck, A. & Hamby, S. L. (2013). Violence, crime, and abuse exposure in a national sample of children and youth: an update. *JAMA Pediatr 167*(7), 614-21.

Forbes, E. E. & Dahl, R. E. (2010). Pubertal development and behavior: Hormonal activation of social and motivational tendencies. *Brain and Cognition 72*(1), 66-72.

Ford, E., Clark, C. & Stansfeld, S. A. (2011). The influence of childhood adversity on social relations and mental health at mid-life. *Journal of Affective Disorders 133*(1-2), 320-7.

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Davey Smith, G., Henderson, J., Macleod, J., Molloy, L., Ness, A., Ring, S., Nelson, S. M. & Lawlor, D. A. (2013). Cohort Profile: the Avon Longitudinal Study of Parents and Children: ALSPAC mothers cohort. *Int J Epidemiol 42*(1), 97-110.

Frith, C. D. & Frith, U. (2007). Social Cognition in Humans. *Current Biology 17*(16), R724-R32.

Germine, L., Dunn, E. C., McLaughlin, K. A. & Smoller, J. W. (2015). Childhood adversity is associated with adult theory of mind and social affiliation, but not face processing. *PLoS One 10*(6), e0129612.

Germine, L. T., Duchaine, B. & Nakayama, K. (2011). Where Cognitive Development and Aging Meet: Face Learning Ability Peaks after Age 30. *Cognition 118*(2), 201-10.

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

Green, M. F., Olivier, B., Crawley, J. N., Penn, D. L. & Silverstein, S. (2005). Social Cognition in Schizophrenia: Recommendations from the Measurement and Treatment Research to Improve Cognition in Schizophrenia New Approaches Conference. *Schizophrenia Bulletin 31*(4), 882-7.

Gur, R. C., Richard, J., Calkins, M. E., Chiavacci, R., Hansen, J. A., Bilker, W. B., Loughead, J., Connolly, J. J., Qiu, H., Mentch, F. D., Abou-Sleiman, P. M., Hakonarson, H. & Gur, R. E.

(2012). Age group and sex differences in performance on a computerized neurocognitive battery in children age 8-21. *Neuropsychology 26*(2), 251-65.
Happé, F. & Frith, U. (2014). Annual Research Review: Towards a developmental neuroscience of atypical social cognition. (pp. 553-77).
Hibbeln, J. R., Davis, J. M., Steer, C., Emmett, P., Rogers, I., Williams, C. & Golding, J. (2007). Maternal seafood consumption in pregnancy and neurodevelopmental outcomes in childhood (ALSPAC study): An observational cohort study. *The Lancet 369*(9561), 578-85.
Kilford, E. J., Garrett, E. & Blakemore, S.-J. (2016). The development of social cognition in adolescence: An integrated perspective. *Neuroscience and Biobehavioral Reviews 70*(C), 106-20.
Kline, R. B. (2010). *Principles and practice of structural equation modeling.* New York, NY: Guilford Press.
Kuh, D., Ben-Shlomo, Y., Lynch, J., Hallqvist, J. & Power, C. (2003). Life course epidemiology. *Journal of Epidemiology and Community Health 57*(10), 778-83.
Ladegaard, N., Larsen, E. R., Videbech, P. & Lysaker, P. H. (2014). Higher-order social cognition in first-episode major depression. *Psychiatry Research 216*(1), 37-43.
Lang, C. A., Cox, M. J. & Flores, G. (2013). Maltreatment in multiple-birth children. *Child abuse & neglect 37*(12), 1109-13.
Lawrence, K., Campbell, R. & Skuse, D. (2015). Age, gender, and puberty influence the development of facial emotion recognition. *Front Psychol 6*, 761.
Lockhart, R., Taylor, J., Tibshirani, R. J. & Tibshirani, R. (2014). A significance test for the LASSO. *Ann Stat 42*(2), 413-68.
Luke, N. & Banerjee, R. (2013). Differentiated associations between childhood maltreatment experiences and social understanding: A meta-analysis and systematic review. *Developmental Review 33*(1), 1-28.
Magiati, I., Tay, X. W. & Howlin, P. (2014). Cognitive, language, social and behavioural outcomes in adults with autism spectrum disorders: A systematic review of longitudinal follow-up studies in adulthood. *Clinical psychology review 34*(1), 73-86.
Manly, J. T., Kim, J. E., Rogosch, F. A. & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and psychopathology 13*(04), 759-82.
McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M. & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication II: Associations with persistence of DSM-IV disorders. *Archives of General Psychiatry 67*(2), 124-32.
Messinger, D. S., Young, G. S., Webb, S. J., Ozonoff, S., Bryson, S. E., Carter, A., Carver, L., Charman, T., Chawarska, K., Curtin, S., Dobkins, K., Hertz-Picciotto, I., Hutman, T., Iverson, J. M., Landa, R., Nelson, C. A., Stone, W. L., Tager-Flusberg, H. & Zwaigenbaum, L. (2015). Early sex differences are not autism-specific: A Baby Siblings Research Consortium (BSRC) study. *Mol Autism 6*, 32.
Mills, K. L., Lalonde, F., Clasen, L. S., Giedd, J. N. & Blakemore, S.-J. (2014). Developmental changes in the structure of the social brain in late childhood and adolescence. *Social Cognitive and Affective Neuroscience 9*(1), 123-31.
Munafò, M. R. & Davey Smith, G. (2018). Robust research needs many lines of evidence. *Nature 553*(7689), 399.

Oliver, B. R., Barker, E. D., Mandy, W. P. L., Skuse, D. H. & Maughan, B. (2011). Social Cognition and Conduct Problems: A Developmental Approach. *Journal of the American Academy of Child & Adolescent Psychiatry 50*(4), 385-94.

Palmier-Claus, J., Berry, K., Darrell-Berry, H., Emsley, R., Parker, S., Drake, R. & Bucci, S. (2016). Childhood adversity and social functioning in psychosis: Exploring clinical and cognitive mediators. *Psychiatry Research 238*, 25-32.

Piskulic, D. & Addington, J. (2011). Social cognition and negative symptoms in psychosis. *Psychiatry Res 188*(2), 283-5.

Prino, L. E., Rollè, L., Sechi, C., Patteri, L., Ambrosoli, A., Caldarera, A. M., Gerino, E. & Brustia, P. (2016). Parental Relationship with Twins from Pregnancy to 3 Months: The Relation Among Parenting Stress, Infant Temperament, and Well-Being. *Frontiers in Psychology 7*, 1628.

Rose, A. J. & Rudolph, K. D. (2006). A Review of Sex Differences in Peer Relationship Processes: Potential Trade-Offs for the Emotional and Behavioral Development of Girls and Boys. *Psychological Bulletin 132*(1), 98-131.

Rowe, R., Maughan, B., Worthman, C. M., Costello, E. J. & Angold, A. (2004). Testosterone, antisocial behavior, and social dominance in boys: pubertal development and biosocial interaction. *Biological psychiatry 55*(5), 546-52.

Rubin, D. B. (1987). *Multiple imputation for nonresponse in surveys.* New York, NY: J. Wiley & Sons.

Sameroff, A. J., Lewis, M. & Miller, S. M. (2000). Handbook of developmental psychopathology.

Sasson, N. J., Nowlin, R. B. & Pinkham, A. E. (2013). Social Cognition, Social Skill, and the Broad Autism Phenotype. *Autism: The International Journal of Research and Practice 17*(6), 655-67.

Sergi, M. J., Rassovsky, Y., Nuechterlein, K. H. & Green, M. F. (2006). Social Perception as a Mediator of the Influence of Early Visual Processing on Functional Status in Schizophrenia. *American Journal of Psychiatry 163*(3), 448-54.

Skuse, D. H., James, R. S., Bishop, D. V. M., Coppin, B., Dalton, P., Aamodt-Leeper, G., Bacarese-Hamilton, M., Creswell, C., McGurk, R. & Jacobs, P. A. (1997). Evidence from turner's syndrome of an imprinted x-linked locus affecting cognitive function. *Nature 387*(12), 705-8.

Smith, A. D., Hardy, R., Heron, J., Joinson, C. J., Lawlor, D. A., Macdonald-Wallis, C. & Tilling, K. (2016). A structured approach to hypotheses involving continuous exposures over the life course. *Int J Epidemiol 45*(4), 1271-9.

Smith, A. D., Heron, J., Mishra, G., Gilthorpe, M. S., Ben-Shlomo, Y. & Tilling, K. (2015). Model Selection of the Effect of Binary Exposures over the Life Course. *Epidemiology 26*(5), 719-26.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research 25*, 173-80.

Stoltenborgh, M., Bakermans-Kranenburg, M. J., Alink, L. R. A. & Ijzendoorn, M. H. (2015). The Prevalence of Child Maltreatment across the Globe: Review of a Series of Meta-Analyses. *Child Abuse Review 24*(1), 37-50.

Teicher, M. H., Andersen, S. L., Polcari, A., Anderson, C. M., Navalta, C. P. & Kim, D. M. (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neurosci Biobehav Rev 27*(1-2), 33-44.

Tucker, L. R. & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika 38*, 1-10.

van Buuren, S. & Groothuis-Oudshoorn, K. (2011). mice: Multivariate Imputation by Chained Equations in R. *Journal of Statistical Software 45*, urn:issn:1548-7660.
Van Der Graaff, J., Branje, S., De Wied, M., Hawk, S., Van Lier, P. & Meeus, W. (2014). Perspective Taking and Empathic Concern in Adolescence: Gender Differences in Developmental Changes. *Developmental Psychology 50*(3), 881-8.
Vauth, R., Rüsch, N., Wirtz, M. & Corrigan, P. W. (2004). Does social cognition influence the relation between neurocognitive deficits and vocational functioning in schizophrenia? *Psychiatry Research 128*(2), 155-65.
Vetter, N. C., Leipold, K., Kliegel, M., Phillips, L. H. & Altgassen, M. (2013). Ongoing development of social cognition in adolescence. *Child Neuropsychology 19*(6), 615-29.
Yves, R. (2012). lavaan : An R Package for Structural Equation Modeling. *Journal of Statistical Software 48*(2).

Table 1. Exposure to childhood maltreatment in the analytic sample, stratified by type, sex, and age at exposure (n=4438)

	Sexual or physical abuse (by anyone)				Physical or emotional abuse			
	Female		Male		Female		Male	
	N	(%)	N	(%)	N	(%)	N	(%)
Unexposed	1964	89.03	1884	84.41	1812	82.14	1852	82.97
Exposed	242	10.97	348	15.59	394	17.86	380	17.03
Age at Exposure								
Infancy								
Age 8 mo.	---	---	---	---	82	3.81	70	3.24
Age 1.5/1.75	48	2.22	52	2.39	89	4.18	72	3.36
Age 2.5/2.75	59	2.81	83	3.88	110	5.22	123	5.79
Preschool								
Age 3.5	51	2.39	71	3.29	---	---	---	---
Age 4/4.75	48	2.27	84	3.91	115	5.42	85	3.95
Age 5/5.75	40	1.92	63	2.97	138	6.60	136	6.40
Middle Childhood								
Age 6/6.75	40	1.89	74	3.47	123	5.89	93	4.36
Age 8/9	42	2.00	95	4.45	74	3.51	84	3.95

Percentages for each age represent proportions of children exposed out of the total analytic sample.
--- indicates that the variable was not assessed at the corresponding time point

Table 2. Results of the SLCMA for each measure of maltreatment on social cognition

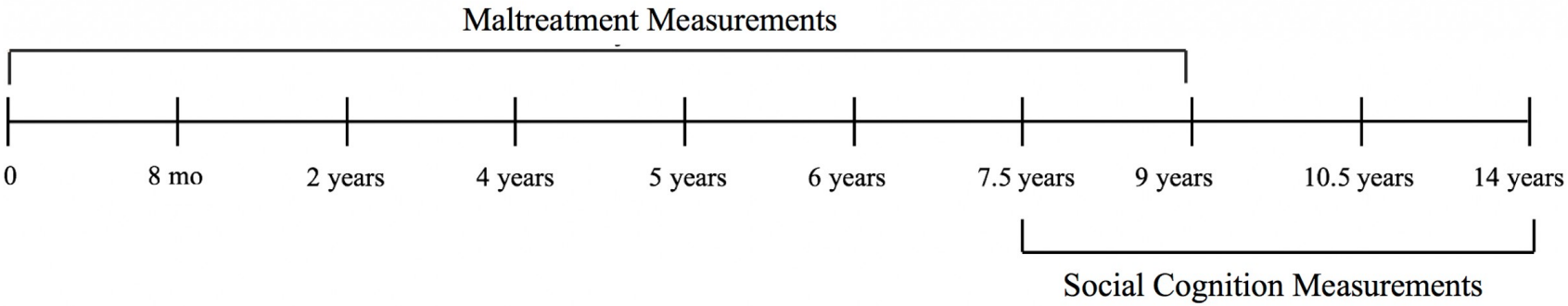
Sexual or physical abuse (by anyone)

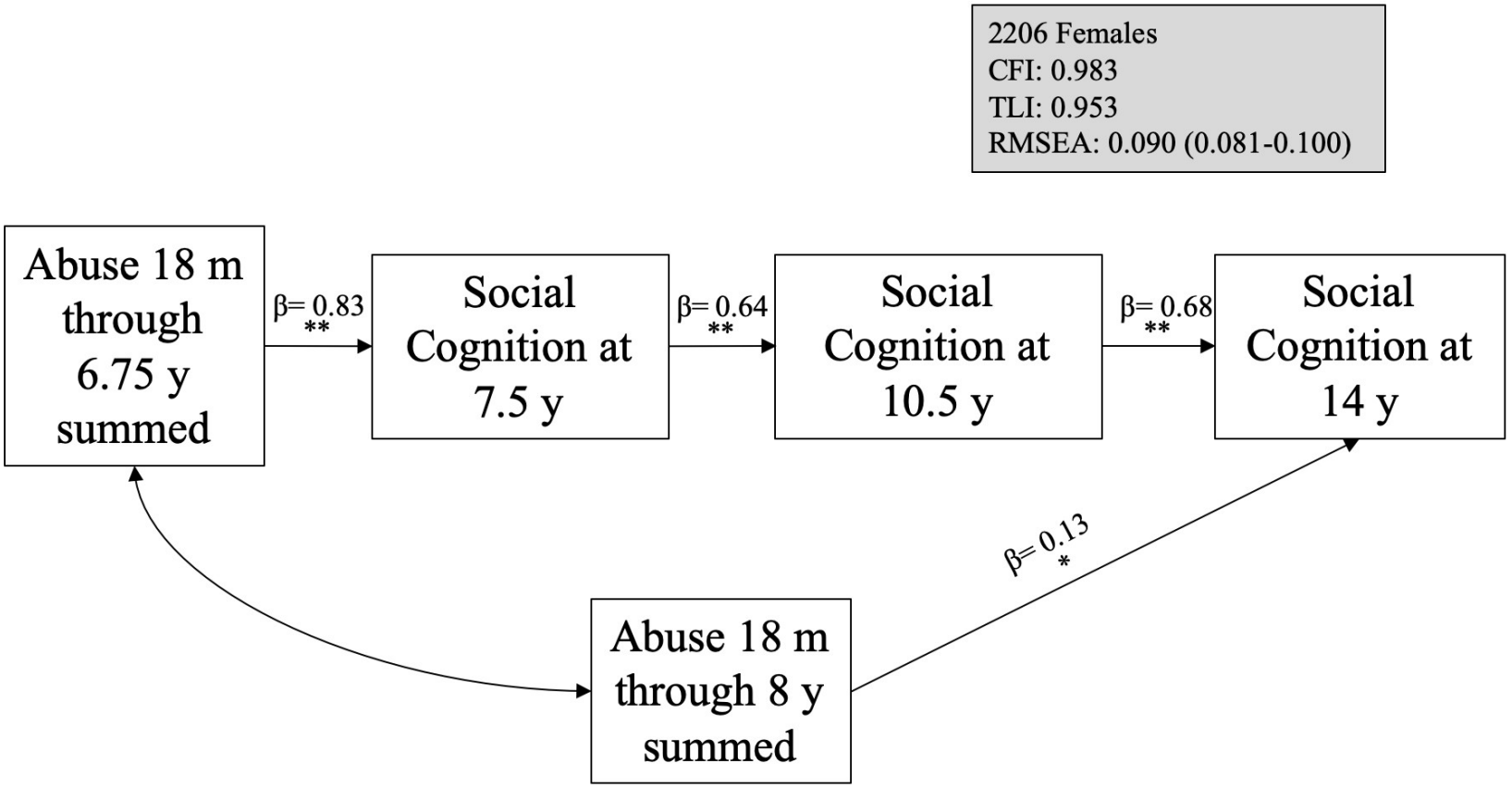
	Stage 1			Stage 2			
	Model(s) selected	R^2	P Value	β	S.E.	Lower CI	Upper CI
Female(N=2206)							
Age 7.5	Accumulation	0.71%	<0.01	0.66	0.13	0.41	0.91
Age 10.5	Accumulation	0.22%	0.08	0.60	0.12	0.30	0.90
Age 14	Accumulation	0.81%	<0.01	0.65	0.13	0.39	0.92
Male (N=2232)							
Age 7.5	None						
Age 10.5	None						
Age 14	None						

Physical or emotional abuse by caregiver

	Stage 1			Stage 2			
	Model(s) selected	R^2	P Value	β	S.E.	Lower CI	Upper CI
Female (N=2206)							
Age 7.5	None						
Age 10.5	Accumulation	0.36%	0.01	0.27	0.07	0.14	0.40
Age 14	None						
Male (N=2232)							
Age 7.5	Ever Exposed	0.43%	<0.01	0.86	0.23	0.41	1.31
Age 10.5	Accumulation	0.23%	0.06	0.44	0.10	0.21	0.68
Age 14	None						

Stage 1 cell entries are r2 values, p-values. Stage 2 cell entries are betas, standard errors, and p-values derived from multiple linear regression (one regression for each type of maltreatment and social cognition measurement). Models were only reported at Stage 2 when the covariance test p-value was below the threshold of 0.1.


Maltreatment Measurements
0
8 mo
2 years
4 years
5 years
6 years
7.5 years
9 years
10.5 years
14 years
Social Cognition Measurements


2206 Females
CFI: 0.983
TLI: 0.953
RMSEA: 0.090 (0.081-0.100)
Abuse 18 m through 6.75 y summed
β= 0.83 **
Social Cognition at 7.5 y
β= 0.64 **
Social Cognition at 10.5 y
β= 0.68 **
Social Cognition at 14 y
Abuse 18 m through 8 y summed
β= 0.13 *

Supplemental Materials

Analytic Sample

In **Supplemental Table 1**, we describe the distribution of study covariates by our analytic sample and others. Specifically, we compared the distributions of covariates between participants included in the total analytic sample (n=4438) and three other subsamples of ALSPAC: (1) participants who had at least one measure of social cognition, but were excluded based on other selection criteria (n=5239); (2) subset of the analytic sample who were exposed to sexual or physical abuse before age 10 (n=590); and (3) subset of the analytic sample who were exposed to caregiver physical or emotional abuse before age 10 (n=774).

Data Selection

Out of ALSPAC's 14,901 enrolled children alive at 1 year of age, there were 9677 children who had at least one measure of social cognition out of the three timepoints in which it was measured (ages 7.5, 10.5 and 14 years). With this base set of 9677 children, we then applied our exclusion criteria to identify the analytic sample. In a stepwise fashion, we first excluded children who did not have outcome measures at all three timepoints as would be needed for our life course modeling approach (5222 children remained). As the social experience of twins likely differs from singletons, we then excluded an additional 96 multiple-birth children. Lastly, as we restricted the analyses to only those children who had mothers and maternal figures as the sole reporters of their social cognition skills over the three timepoints of assessment to minimize reporter inconsistency, 688 children were additionally removed, yielding a total sample of 4,438 children.

We compared the distribution of covariates and outcome scores between the analytic sample (n=4438) and the subset of excluded participants with complete social cognition outcome data at all three time points, but who lacked consistent maternal reports (n=688). These two samples had largely similar sociodemographic characteristics and social cognition scores at all three time points; however, the excluded sample (without consistent maternal reports) were more likely to be born to mothers with slightly higher education (**Supplemental Table 2**).

Measures

ALSPAC was established to better understand how genetic and environmental features influence health and development of children (Fraser, 2013). Due to the breadth of this research question, specific scales or measures may not have been included at every time point of assessment. In **Supplemental Table 3**, we describe how each of the child maltreatment variables, described below was specially constructed including questions and time periods that were covered.

Child Maltreatment

To measure physical or sexual abuse by anyone, two items from an inventory assessing exposure to a series of life events were used. Specifically, mothers reported whether or not the child had been either "sexually abused" or "physically hurt by someone." If they answered "yes" on either of the two items, the child was coded as exposed. To measure caregiver physical or emotional abuse, both the mother and the partner provided responses to the following four items: 1) your partner was physically cruel to your children; 2) you were physically cruel to your children; 3) your partner was emotionally cruel to your children; 4) you were emotionally cruel

to your children. If either the mother or the partner answered affirmatively to any of the four questions above, the child was coded as exposed. The participants were assured that their responses were confidential and no information would be reported to child welfare agencies, as no mandatory reporting laws were in place in the UK at the time of data collection (Bell, 1994; Khan, 2018). We note that because of the questionnaire wording both measures of child maltreatment ("Caregiver physical or emotional abuse" and "Sexual or physical abuse") could double-count caregiver physical abuse. The specific time periods covered by these questions are described in **Supplemental Table 3.**

Correlations between caregiver physical and emotional abuse items are shown in **Supplemental Table 4**. Correlations between the two types of maltreatment examined in this study are shown in **Supplemental Table 5**. Of note, while the prevalence of being ever exposed to sexual abuse before age 10 was much lower in the analytic sample (0.4%) compared to the prevalence of being ever exposed to physical abuse before age 10 (13.1%), the two exposures were moderately correlated ($r_{tetrachoric} = 0.39$).

Social Cognition

The distribution of social cognition scores across time, stratified by child sex, are shown in **Supplemental Table 6.**

Covariates

We controlled for the following covariates, measured at the time of the child's birth: *child race/ethnicity* (0=non-White; 1=White); *number of previous pregnancies* (between 0-3+); *maternal marital status* (0=never married; 1=widowed/divorced/separated; 2=married); *highest level of maternal education* (1=less than O-level, 2=O-level, 3=A-level, 4=Degree or above); *maternal age* (0=ages 15-19, 1=ages 20-35, 2=age>35); *homeownership* (0=mortgage/own home; 1=rent home; 2=other); *parent social class* (i.e. the highest social class of either parent: 1=professional; 2=managerial and technical; 3=skilled, non-manual; 4=skilled, manual; 5=semi-skilled, manual; 6=unskilled manual/other); and *maternal depressive symptoms* (measured by total scores on the Edinburgh Postnatal Depression Scale; scores ranged from 0-30 with higher scores indicating higher levels of depressive symptoms) (Adkins et al., 2011; Anney et al., 2010; Baker, Taylor & The Alspac Survey Team, 1997; Chen et al., 2013; Wood, White & Royston, 2008).

LARs Variable Selection and Structural Modeling

We achieved a single dataset for analysis by implementing LARs on the covariance structure among all variables, estimated by averaging the covariance structure across all multiply imputed datasets. This allowed us to avoid potential problems arising from different model selections across multiply imputed datasets (Wood et al., 2008).

We then evaluated the relative importance of these maltreatment variables using a two-stage structured lifecourse modeling approach (SLCMA) originally developed by Mishra (Mishra et al., 2009) for analyzing repeated, binary exposure data across the lifecourse. Relative to a more traditional regression model, the main advantage of the SLCMA is that it provides a structured and unbiased way to compare multiple competing theoretical models simultaneously and identify the most parsimonious explanation for the observed outcome variation.

In the first stage, we followed the approach of Smith (Smith et al., 2015) and entered the set of maltreatment variables described previously into a Least Angle Regression (LARs)

procedure (Efron et al., 2004) in order to identify, separately for each type of maltreatment, the single theoretical model (or potentially more than one theoretical models working in combination) that explained the most variability in child social cognitive difficulties. We used a covariance test (Lockhart et al., 2014) and examined elbow plots (**Supplemental Figure 1**) to determine whether the selected models were supported by the ALSPAC data. Compared to other variable selection procedures, including stepwise regression, the SLCMA has been shown to not over-inflate effect size estimates (Efron et al., 2004) or bias hypothesis tests (Lockhart et al., 2014). Compared to other methods for the structured approach, LARs has been shown to have greater statistical power and not bias subsequent stages of analysis (Smith et al., 2015). To adjust for potential confounding, we regressed each encoded variable on the covariates and implemented LARs on the regression residuals (Smith et al., 2016).

In the second stage, the theoretical models determined by a covariance test p-value threshold of 0.05 in the first stage (which appeared before the elbow; see **Supplemental Figure 1**) was carried forward to a single multiple regression framework, where measures of effect would have been estimated for all selected hypotheses. The goal of this second stage was to determine the contribution of a selected theoretical model after adjustment for covariates as well as other selected theoretical models, in instances where more than one theoretical model was chosen in the first stage.

Multiple Imputation

As noted above, there were 4,438 children with complete outcome data at all three time points who met our inclusion criteria. However, a small proportion of these 4,438 children had missing exposure or covariate data; rates of missingness for exposure or covariate data ranged per variable from 4.3% (n=279 for maternal birth age) to 19.1% (n=1244 for presence versus absence of maternal psychopathology at 6 years).

To reduce potential bias and minimize loss of power due to attrition, we performed multiple imputation, separately for each exposure, using logistic regression in 20 datasets with 25 iterations each among all children with complete outcome data. In addition to imputing exposures, we also imputed covariates as described here. Of note, variables were included in the imputation models following the guidance of van Buuren and colleagues (van Buuren, Boshuizen & Knook, 1999; van Buuren & Groothuis-Oudshoorn, 2011) as well as prior research with imputation in the ALSPAC dataset (Evans et al., 2012; Ramchandani et al., 2008). The following variables were allowed to enter the imputation models: all covariates and exposures to the specific type of maltreatment from ages 0-8. Variables uncorrelated with the missing variable ($r<0.10$) were excluded from the imputation model (van Buuren et al., 1999; van Buuren & Groothuis-Oudshoorn, 2011). Imputation was performed with chained equations (Azur et al., 2011) with the *mice* package in R (van Buuren & Groothuis-Oudshoorn, 2011). To reduce noise in estimation of effect estimates, we did not impute the outcome (White, Royston & Wood, 2011). For each maltreatment, we assessed the convergence of the imputation model and the distribution of imputed data as compared to the observed data.

Results

Study results after winsorizing social cognition scores are shown in **Supplemental Table 7**.

Exploring the Possibility that Social Cognition Predicts Child Maltreatment

A primary hypothesis tested in this paper was that childhood maltreatment predicts future social cognitive skills. However, children with poor social cognitive skills may also be more likely than their peers to be exposed to child maltreatment. To explore this possibility, we performed a secondary analysis to examine the association between social cognition and child maltreatment. The first assessment of social cognition was available at age 7.5 years, which preceded the last two assessments of child maltreatment that we included in the analysis: sexual or physical abuse by anyone at 8 years and caregiver physical or emotional abuse at 9 years. We therefore fitted logistic regression models to test whether being abused later on (at 8 or 9 years) was predicted by levels of social cognition at 7.5 years. All baseline covariates included in our original analysis were also adjusted for here. Specifically, we assessed the associations between social cognition measured at age 7.5 years and odds of being exposed to each type of maltreatment separately in sex-stratified analyses (i.e., a total of four logistic regression models were fitted). We did not differentiate between incident cases of exposure to maltreatment at 8 or 9 years and cases with prior history of exposure, to preserve statistical power and keep the model parsimonious.

Among youth exposed to caregiver physical or emotional abuse at 9 years (n=158), there were 65 children whose parents had reported incident maltreatment, meaning children who had experienced new instances of caregiver physical or emotional abuse. Among youth exposed to physical or sexual abuse (by anyone) at 8 years (n=137), there were 59 were incident cases.

As shown in **Supplemental Table 8**, we found that poorer earlier social cognition skills were generally associated with lower levels of exposure to maltreatment. Specifically, the odds of being exposed to maltreatment were lower by 6-11% for each one-point increase on the social cognition scale (or worsening of social cognition scores). For example, for female participants, each one-point increase in social cognition at age 7.5 years was associated with a 9% decrease in the odds for being exposed to sexual or physical abuse by anyone at 8 years (*OR*=0.91, *p*=0.012). Similarly, each one-point increase in social cognition at age 7.5 was linked to a 11% decrease in the odds of being exposed to caregiver physical or emotional abuse at 9 years (*OR*=0.89, *p*=0.0001).

However, for boys, social cognition scores were only associated with sexual or physical abuse. Taken together, these findings do not suggest the possibility that children with poor social cognitive skills are at a substantially higher risk than their peers to be exposed to child maltreatment.

Supplemental Table 1. Comparisons of baseline sociodemographic characteristics in the total analytic sample versus among three subsamples of ALSPAC participants

	Total analytic sample (n=4438)		Excluded from the analytic sample (n=5239)			Exposed to sexual or physical abuse (n=590)		Exposed to physical or emotional abuse (n=774)	
	%	N	%	N	*p*-value	%	N	%	N
Gender					0.2				
Males	50.29	2232	51.63	2705		58.98	348	49.1	380
Females	49.71	2206	48.37	2534		41.02	242	50.9	394
Race					<0.01				
White	97.12	4186	94.69	4265		96.19	555	95.74	720
Non-White	2.88	124	5.31	239		3.81	22	4.26	32
Maternal Education					<0.01				
Less than O-level	18.2	794	29.96	1381		16.41	96	17.23	132
O-level	36.35	1586	34.34	1583		32.31	189	34.99	268
A-level	27.5	1200	22.99	1060		29.91	175	30.42	233
Degree or Above	17.95	783	12.71	586		21.37	125	17.36	133
Maternal Marital Status					<0.01				
Never Married	11.96	523	17.49	822		14.95	87	13.12	100
Widowed/Divorced/Separated	4.73	207	5.43	255		5.5	32	6.96	53
Married	83.31	3644	77.08	3622		79.55	463	79.92	609
Home Ownership					<0.01				
Mortgage/own home	85.63	3724	75.81	3539		81.83	473	80.4	607
Rent home	11.89	517	21.1	985		15.57	90	15.76	119
Other	2.48	108	3.08	144		2.6	15	3.84	29
Age of Mother at Child Birth					<0.01				
Ages 15-19	1.4	62	3.49	170		1.86	11	0.78	6
Ages 20-35	89.78	3978	89.1	4341		87.8	518	89.52	692
Age >35	8.82	391	7.41	361		10.34	61	9.7	75
Parental Social Class					<0.01				
Professional	15.4	683	10	524		15.25	90	16.02	124
Managerial and technical	39.39	1748	30.65	1606		42.54	251	40.44	313
Skilled, non-manual	21.43	951	19.18	1005		20.34	120	18.6	144

Skilled, manual	5.5	244	6.74	353		5.25	31	6.59	51
Semi-skilled, manual	1.35	60	2.02	106		1.02	6	1.42	11
Unskilled manual/other	16.94	752	31.4	1645		15.59	92	16.93	131
Number of previous pregnancies					<0.01				
0	47.7	2072	44.35	2063		47.47	272	38.76	293
1	36.07	1567	35.32	1643		34.21	196	39.15	296
2	12.22	531	14.6	679		13.61	78	16.93	128
3+	4.01	174	5.74	267		4.71	27	5.16	39

We compared the distributions of baseline characteristics between participants included in the total analytic sample (n=4438) and three other subsamples of ALSPAC: (1) participants who had at least one measure of social cognition, but were excluded based on other selection criteria (n=5239); (2) subset of the analytic sample who was exposed to sexual or physical abuse before age 10 (n=590); and (3) subset of the analytic sample who was exposed to caregiver physical or emotional abuse before age 10 (n=774). Notably, the original eligible sample (N=9677) consisted of all children that had at least one measure of social cognition. We restricted these analyses to singleton births with complete outcome data who had mothers and maternal figures as the sole reporters of their social cognition skills over the three timepoints of assessment.
p-values were determined from chi-squared tests, assessing whether the distributions of categorical covariates were different across samples. Values corresponding to education level are presented in rank order from lowest education level (less than O or Ordinary level) to Degree.

Supplemental Table 2. Distributions of covariates and social cognition scores in the analytic sample versus the sample of participants who were excluded due to having non-maternal reports

	Analytic sample (i.e., participants with maternal reports at all three time points) (n=4438)	Participants with complete but non-maternal reports of social cognition scores (n=688)	Compared to the total analytic sample
	N (%)	N (%)	*p*-value
Gender			0.535
Males	2232 (50.3)	364 (51.6)	
Females	2206 (49.7)	341 (48.4)	
Race			0.069
Non-White	124 (2.9)	29 (4.3)	
White	4186 (97.1)	653 (95.7)	
Maternal Education			<0.001
Less than O-level	794 (18.2)	139 (20.1)	
O-level	1586 (36.4)	186 (26.9)	
A-level	1200 (27.5)	189 (27.4)	
Degree or Above	783 (17.9)	177 (25.6)	
Maternal Marital Status			0.388
Never Married	523 (12.0)	74 (10.7)	
Widowed/Divorced/Separated	207 (4.7)	27 (3.9)	
Married	3644 (83.3)	588 (85.3)	
Home Ownership			0.312
Mortgage/own home	3724 (85.6)	581 (84.9)	
Rent home	517 (11.9)	91 (13.3)	
Other	108 (2.5)	12 (1.8)	
Age of Mother at child birth			0.808
Ages 15-19	62 (1.4)	8 (1.1)	
Ages 20-35	3978 (89.8)	630 (89.6)	
Age >35	391 (8.8)	65 (9.2)	
Parental Social Class			0.049
Professional	683 (15.4)	140 (19.9)	
Managerial and technical	1748 (39.4)	274 (38.9)	
Skilled, non-manual	951 (21.4)	127 (18.0)	
Skilled, manual	244 (5.5)	38 (5.4)	
Semi-skilled, manual	60 (1.4)	9 (1.3)	
Unskilled manual/other	752 (16.9)	117 (16.6)	

Number of previous pregnancies			0.729
0	2072 (47.7)	342 (49.6)	
1	1567 (36.1)	236 (34.3)	
2	531 (12.2)	81 (11.8)	
3+	174 (4.0)	30 (4.4)	
	Mean (SD)	Mean (SD)	*p*-value
Maternal depressive symptoms	5.03 (4.43)	4.95 (4.44)	0.688
Social cognition scores 7.5 years	2.69 (3.53)	2.54 (3.32)	0.321
Social cognition scores 10 years	14.19 (3.38)	13.98 (2.90)	0.121
Social cognition scores 14 years	14.43 (3.56)	14.46 (3.56)	0.866

We compared the distributions of baseline characteristics between participants included in the total analytic sample (n=4438) and a subset of excluded participants who had complete outcome data at all three time points but non-maternal reports (n=688).
p-values were determined from chi-squared tests and t-tests assessing the differences between the distributions of baseline covariates and social cognition skills in the two samples.

Supplemental Table 3. Summary of the two maltreatment measures and the time periods covered by each item

	Description	Time period covered
Sexual or Physical Abuse	Exposure to sexual or physical abuse was determined through an item asking the mother to indicate whether or not the child had been exposed to either sexual or physical abuse from anyone. This question was included at seven time-points: child ages 1.5, 2.5, 3.5, 4.75, 5.75, 6.75, and 8 years.	1.5y: 0.5-1.5y 2.5y: 1.5-2.5y 3.5y: 1-3.5y 4.75y: 3-4.74y 5.75y: 1.25-5.75y 6.75y: 5-6.75y 8.5y: 7-8y
Caregiver Physical or Emotional Abuse	Exposure to physical or emotional abuse was determined through mailed questionnaires administered separately to the mother and the mother's partner. Children were coded as having been exposed to physical or emotional abuse if the mother, partner, or both responded affirmatively to any of the following items assessed over seven time-points: (1) Your partner was physically cruel to your children; (2) You were physically cruel to your children; (3) Your partner was emotionally cruel to your children; (4) You were emotionally cruel to your children. The seven-time points were: 8 months, 1.75, 2.75, 4, 5, 6, and 9 years.	8m: birth to 8m 1.75y: 8m-1.75y 2.75y: 1.5-2.75y 4y: 2.5-4y 5y: 4-5y 6y: 5-6y 9y: 6-9y

Supplemental Table 4. Tetrachoric correlations between caregiver physical and emotional abuse items

	Parental physical abuse						
	Age	8 mo	1.75y	2.75y	4y	5y	6y	9y
Parental emotional abuse	8 mo	0.78	--	--	--	--	--	--
	1.75y	0.60	0.73	--	--	--	--	--
	2.75y	0.45	0.67	0.75	--	--	--	--
	4y	0.36	0.48	0.56	0.78	--	--	--
	5y	0.47	0.50	0.56	0.58	0.79	--	--
	6y	0.45	0.40	0.41	0.61	0.55	0.70	--
	9y	0.36	0.36	0.41	0.41	0.41	0.41	0.77

Tetrachoric correlation coefficients are presented in each cell to show the pairwise correlation between caregiver physical and emotional abuse at each time point. Notably, the two measures, when measured at the same tie point (see the diagonal), were strongly correlated (*rho* > 0.7).

Supplemental Table 5. Tetrachoric correlations between types of childhood maltreatment

Physical or emotional abuse (N=3677)							
Age	8 mo	1.75	2.75	4	5	6	9
8 mo	1	--	--	--	--	--	--
1.75	0.72	1	--	--	--	--	--
2.75	0.59	0.72	1	--	--	--	--
4	0.46	0.64	0.73	1	--	--	--
5	0.51	0.55	0.61	0.63	1	--	--
6	0.49	0.58	0.56	0.64	0.68	1	--
9	0.44	0.49	0.39	0.42	0.50	0.51	1

Sexual or physical abuse (by anyone) (N=3689)							
Age	1.5	2.5	3.5	4.75	5.75	6.75	8
1.5	1	--	--	--	--	--	--
2.5	0.5	1	--	--	--	--	--
3.5	0.36	0.39	1	--	--	--	--
4.75	0.33	0.44	0.44	1	--	--	--
5.75	0.4	0.43	0.47	0.52	1	--	--
6.75	0.3	0.4	0.35	0.46	0.63	1	--
8	0.46	0.37	0.39	0.44	0.54	0.59	1

Note. These results were generated using non-imputed datasets.

Supplemental Table 6. Social cognition scores across time

	Girls		Boys	
Age	Mean	S.D.	Mean	S.D.
7.5	2.28	3.04	3.09	3.93
10.5	1.83	2.84	2.45	3.79
14	2.19	3.19	2.56	3.86

Note. At each time period of measurement, there was a significant difference ($p<0.001$) between boys' and girls' scores

Supplemental Table 7. Results of the SLCMA for each measure of maltreatment on social cognition that were winsorized at the 90% percentile to address data skewness

Sexual or physical abuse (by anyone)

	Stage 1			Stage 2			
	Model(s) selected	R^2	P Value	β	S.E.	Lower CI	Upper CI
Girls (N=2206)							
Age 7.5	Accumulation	0.36%	0.01	0.39	0.10	0.19	0.59
Age 10.5	Accumulation	0.46%	<0.01	0.33	0.08	0.18	0.48
Age 14	Accumulation	0.78%	<0.01	0.43	0.91	0.25	0.61
Boys (N=2232)							
Age 7.5	None						
Age 10.5	Accumulation	0.65%	<0.01	0.28	0.06	0.16	0.39
Age 14	Accumulation	0.72%	<0.01	0.35	0.07	0.21	0.49

Physical or emotional abuse

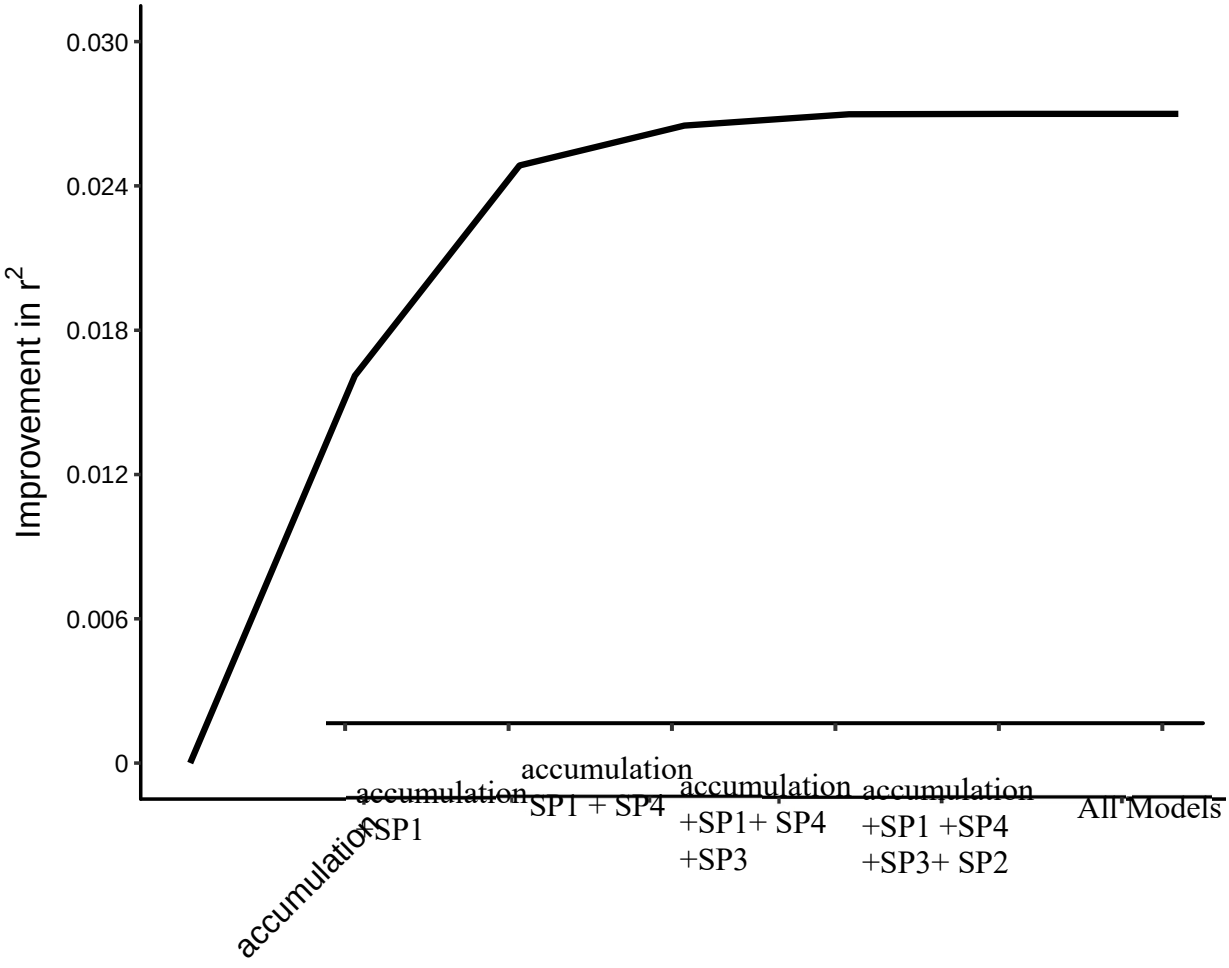
	Stage 1			Stage 2			
	Model(s) selected	R^2	P Value	β	S.E.	Lower CI	Upper CI
Girls (N=2206)							
Age 7.5	None						
Age 10.5	None						
Age 14	None						
Boys (N=2232)							
Age 7.5	Ever Exposed	0.70%	<0.01	0.58	0.14	0.31	0.85
Age 10.5	Accumulation	0.28%	0.03	0.23	0.05	0.12	0.33
Age 14	None						

Stage 1 cell entries are r^2 values and p-values. Stage 2 cell entries are betas, standard errors, and p-values derived from multiple linear regression (one regression for each type of maltreatment) and social cognition measurement). Models were only reported at Stage 2 when the covariance test p-value was below the threshold of 0.1 .

Supplemental Table 8. Social cognition scores at age 7.5 predicting exposure to sexual or physical abuse at 8 years and caregiver physical or emotional abuse at 9 years

	Maltreatment Type	Beta	SE	P-value	OR	OR Lower CI	OR Upper CI
Female	Sexual or physical abuse 8 years	-0.10	0.04	0.0119	0.91	0.84	0.98
	Caregiver physical or emotional abuse 9 years	-0.12	0.03	0.0001	0.89	0.84	0.94
Male	Sexual or physical abuse 8 years	-0.06	0.02	0.0071	0.94	0.90	0.98
	Caregiver physical or emotional abuse 9 years	-0.01	0.03	0.7883	0.99	0.94	1.05

Supplemental Figure 1. Example elbow plot illustrating LARs variable selection procedure testing two life course models: accumulation and sensitive periods



LARs begins by first identifying the single variable with the strongest association to the outcome; it then identifies the combination of two variables with the strongest association, followed by three variables, and so on, until all variables are included. LARs therefore achieves parsimony by identifying the smallest combination of encoded variables that explain the most amount of outcome variation. In addition to a covariance test, which is calculated at each stage of the LARs procedure and tests the null hypothesis that adding the next encoded variable does not improve r^2, results can also be summarized in an "elbow plot," showing the increase in overall model r^2 as additional predictors are added to the model. The point where this plot levels off indicates the point of diminishing marginal improvement to the model goodness-of-fit from adding additional predictors, suggesting that the predictors included in the model at this point represent an optimal balance of parsimony and thoroughness. In this example, both accumulation and sensitive period 1 were selected in the best fitting models. SP =Sensitive Period.

References

Adkins, D. E., Aberg, K., McClay, J. L., Bukszar, J., Zhao, Z., Jia, P., Stroup, T. S., Perkins, D., McEvoy, J. P., Lieberman, J. A., Sullivan, P. F. & van den Oord, E. J. (2011). Genomewide pharmacogenomic study of metabolic side effects to antipsychotic drugs. *Mol Psychiatry 16*(3), 321-32.

Anney, R., Klei, L., Pinto, D., Regan, R., Conroy, J., Magalhaes, T. R., Correia, C., Abrahams, B. S., Sykes, N., Pagnamenta, A. T., Almeida, J., Bacchelli, E., Bailey, A. J., Baird, G., Battaglia, A., Berney, T., Bolshakova, N., Bolte, S., Bolton, P. F., Bourgeron, T., Brennan, S., Brian, J., Carson, A. R., Casallo, G., Casey, J., Chu, S. H., Cochrane, L., Corsello, C., Crawford, E. L., Crossett, A., Dawson, G., de Jonge, M., Delorme, R., Drmic, I., Duketis, E., Duque, F., Estes, A., Farrar, P., Fernandez, B. A., Folstein, S. E., Fombonne, E., Freitag, C. M., Gilbert, J., Gillberg, C., Glessner, J. T., Goldberg, J., Green, J., Guter, S. J., Hakonarson, H., Heron, E. A., Hill, M., Holt, R., Howe, J. L., Hughes, G., Hus, V., Igliozzi, R., Kim, C., Klauck, S. M., Kolevzon, A., Korvatska, O., Kustanovich, V., Lajonchere, C. M., Lamb, J. A., Laskawiec, M., Leboyer, M., Le Couteur, A., Leventhal, B. L., Lionel, A. C., Liu, X. Q., Lord, C., Lotspeich, L., Lund, S. C., Maestrini, E., Mahoney, W., Mantoulan, C., Marshall, C. R., McConachie, H., McDougle, C. J., McGrath, J., McMahon, W. M., Melhem, N. M., Merikangas, A., Migita, O., Minshew, N. J., Mirza, G. K., Munson, J., Nelson, S. F., Noakes, C., Noor, A., Nygren, G., Oliveira, G., Papanikolaou, K., Parr, J. R., Parrini, B., Paton, T., Pickles, A., Piven, J., Posey, D. J., Poustka, A., Poustka, F., Prasad, A., Ragoussis, J., Renshaw, K., Rickaby, J., Roberts, W., Roeder, K., Roge, B., Rutter, M. L., Bierut, L. J., Rice, J. P., Salt, J., Sansom, K., Sato, D., Segurado, R., Senman, L., Shah, N., Sheffield, V. C., Soorya, L., Sousa, I., Stoppioni, V., Strawbridge, C., Tancredi, R., Tansey, K., Thiruvahindrapduram, B., Thompson, A. P., Thomson, S., Tryfon, A., Tsiantis, J., Van Engeland, H., Vincent, J. B., Volkmar, F., Wallace, S., Wang, K., Wang, Z., Wassink, T. H., Wing, K., Wittemeyer, K., Wood, S., Yaspan, B. L., Zurawiecki, D., Zwaigenbaum, L., Betancur, C., Buxbaum, J. D., Cantor, R. M., Cook, E. H., Coon, H., Cuccaro, M. L., Gallagher, L., Geschwind, D. H., Gill, M., Haines, J. L., Miller, J., Monaco, A. P., Nurnberger, J. I., Jr., Paterson, A. D., Pericak-Vance, M. A., Schellenberg, G. D., Scherer, S. W., Sutcliffe, J. S., Szatmari, P., Vicente, A. M., Vieland, V. J., Wijsman, E. M., Devlin, B., Ennis, S. & Hallmayer, J. (2010). A genome-wide scan for common alleles affecting risk for autism. *Hum Mol Genet 19*(20), 4072-82.

Azur, M. J., Stuart, E. A., Frangakis, C. & Leaf, P. J. (2011). Multiple imputation by chained equations: what is it and how does it work? *Int J Methods Psychiatr Res 20*(1), 40-9.

Baker, D., Taylor, H. & The Alspac Survey Team, H. (1997). The relationship between condition-specific morbidity, social support and material deprivation in pregnancy and early motherhood. *Social Science & Medicine 45*(9), 1325-36.

Chen, Y.-a., Lemire, M., Choufani, S., Butcher, D. T., Grafodatskaya, D., Zanke, B. W., Gallinger, S., Hudson, T. J. & Weksberg, R. (2013). Discovery of cross-reactive probes and polymorphic CpGs in the Illumina Infinium HumanMethylation450 microarray. *Epigenetics 8*(2), 203-9.

Efron, B., Hastie, T., Johnstone, I. & Tibshirani, R. (2004). Least angle regression. *The Annals of Statistics 32*(2), 407-99.

Evans, J., Melotti, R., Heron, J., Ramchandani, P., Wiles, N., Murray, L. & Stein, A. (2012). The timing of maternal depressive symptoms and child cognitive development: a longitudinal study. *Journal of child psychology and psychiatry 53*(6), 632-40.

Lockhart, R., Taylor, J., Tibshirani, R. J. & Tibshirani, R. (2014). A significance test for the LASSO. *Ann Stat 42*(2), 413-68.

Mishra, G., Nitsch, D., Black, S., De Stavola, B., Kuh, D. & Hardy, R. (2009). A structured approach to modelling the effects of binary exposure variables over the life course. *International Journal of Epidemiology 38*(2), 528-37.

Ramchandani, P. G., O'Connor, T. G., Evans, J., Heron, J., Murray, L. & Stein, A. (2008). The effects of pre- and postnatal depression in fathers: a natural experiment comparing the effects of exposure to depression on offspring. *J Child Psychol Psychiatry 49*(10), 1069-78.

Smith, A. D., Hardy, R., Heron, J., Joinson, C. J., Lawlor, D. A., Macdonald-Wallis, C. & Tilling, K. (2016). A structured approach to hypotheses involving continuous exposures over the life course. *Int J Epidemiol*.

Smith, A. D., Heron, J., Mishra, G., Gilthorpe, M. S., Ben-Shlomo, Y. & Tilling, K. (2015). Model Selection of the Effect of Binary Exposures over the Life Course. *Epidemiology 26*(5), 719-26.

van Buuren, S., Boshuizen, H. C. & Knook, D. L. (1999). Multiple imputation of missing blood pressure covariates in survival analysis. *Stat Med 18*(6), 681-94.

van Buuren, S. & Groothuis-Oudshoorn, K. (2011). mice: Multivariate Imputation by Chained Equations in R. *Journal of Statistical Software 45*, urn:issn:1548-7660.

White, I. R., Royston, P. & Wood, A. M. (2011). Multiple imputation using chained equations: Issues and guidance for practice. *Stat Med 30*(4), 377-99.

Wood, A. M., White, I. R. & Royston, P. (2008). How should variable selection be performed with multiply imputed data? *Stat Med 27*(17), 3227-46.