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Psychopathology trajectories of children with autism spectrum disorder: The role of family poverty and parenting

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| Abstract: | <p>Objective Children with autism spectrum disorder (ASD) are reported to have high rates of co-occurring psychopathology. Little is known about risk factors that might contribute to this psychopathology. This study modelled the effect of family poverty on psychopathology trajectories in young children with ASD, and examined whether home organisation and maternal warmth and involvement can buffer the effect of poverty on children's psychopathology.</p> <p>Method The sample comprised 209 children with ASD who participated in the UK's Millennium Cohort Study, a population birth cohort study. Individual trajectories of psychopathology at ages 3, 5 and 7 years were analysed using growth curve models. Psychopathology was assessed with the Strengths and Difficulties Questionnaire.</p> <p>Results Children with ASD exhibited increasingly high rates of psychopathology over time. Family poverty was associated with broad and specific (emotional and conduct problems) psychopathology, but not with changes in psychopathology over time. Warmth, involvement and home organisation did not buffer the association of family poverty with psychopathology. However, low warmth explained the relationship between poverty and broad psychopathology, and predicted annual changes in broad psychopathology. Warmth was associated with fewer conduct problems and less hyperactivity, and with an annual decrease in peer and conduct problems. Household chaos was a risk factor for conduct problems as was maternal involvement for peer problems.</p> <p>Conclusions Family poverty, low maternal warmth and household chaos are risk factors for externalising problems in children with ASD. Maternal warmth may be a key</p> |

target for intervention, particularly in poorer families of children with ASD.

Running head: Psychopathology of children with ASD

Psychopathology trajectories of children with autism spectrum disorder: The role of family poverty and parenting

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Objective Children with autism spectrum disorder (ASD) are reported to have high rates of co-occurring psychopathology. Little is known about risk factors that might contribute to this psychopathology. This study modelled the effect of family poverty on psychopathology trajectories in young children with ASD, and examined whether home organisation and maternal warmth and involvement can buffer the effect of poverty on children’s psychopathology. **Method** The sample comprised 209 children with ASD who participated in the UK’s Millennium Cohort Study, a population birth cohort study. Individual trajectories of psychopathology at ages 3, 5 and 7 years were analysed using growth curve models. Psychopathology was assessed with the Strengths and Difficulties Questionnaire. **Results** Children with ASD exhibited increasingly high rates of psychopathology over time. Family poverty was associated with broad and specific (emotional and conduct problems) psychopathology, but not with changes in psychopathology over time. Warmth, involvement and home organisation did not buffer the association of family poverty with psychopathology. However, low warmth explained the relationship between poverty and broad psychopathology, and predicted annual changes in broad psychopathology. Warmth was associated with fewer conduct problems and less hyperactivity, and with an annual decrease in peer and conduct problems. Household chaos was a risk factor for conduct problems as was maternal involvement for peer problems. **Conclusions** Family poverty, low maternal warmth and household chaos are risk factors for externalising problems in children with ASD. Maternal warmth may be a key target for intervention, particularly in poorer families of children with ASD.

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Introduction

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2 Autism spectrum disorder (ASD) describes a group of pervasive developmental disorders, including
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4 autism and Asperger's syndrome.¹ Children with ASD exhibit a range of problems, including
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6 communication difficulties, impairments in reciprocal social interaction, limited imagination, and
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8 repetitive and restricted patterns of behaviours. Currently, ASD affects roughly 1% of the child
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10 population.² Recent research has demonstrated that children and adolescents with ASD can exhibit
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12 additional psychiatric symptoms and disorders, such as anxiety disorders, oppositional defiant disorder
13
14 and ADHD, that do not form part of the diagnostic criteria for ASD.³⁻⁵ This co-occurring
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16 psychopathology^a may severely impact on children with ASD and their families.
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20 Although the aetiology of ASD is unclear, genetic risk factors are known to contribute to its development.
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22 Twin studies consistently reveal a high concordance rate amongst monozygotic compared to dizygotic
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24 twins with autism. However, rates are not 100%,⁷ highlighting the potential importance of environmental
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26 factors. One line of investigation into environmental influences addresses the role of family and
27
28 contextual factors for psychopathology in children with ASD.⁴ These factors play little role in ASD
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30 symptomatology itself. Bettelheim's⁸ proposition that autism may result from poor parenting behaviours
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32 has been discredited. However, environmental factors including aspects of parenting and parent-child
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34 interaction may contribute to the co-occurrence of psychopathology and ASD.⁹⁻¹²
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38 Family socio-economic disadvantage (SED) is widely acknowledged as a risk factor of psychopathology
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40 in the general population of children.¹³ However, research elucidating its role in psychopathology of
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42 children with ASD is generally limited and has produced mixed results.¹⁴⁻¹⁵ In a cross-sectional study
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44 using a population-representative sample of 10- to 14-year olds with ASD, family material deprivation
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46 was not associated with children's psychiatric disorders.⁴ However, in a follow up study, deprivation
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48 predicted less improvement in emotional symptoms over time¹⁶. Using a more detailed measure of family
49
50 SED and a large longitudinal population sample, we aimed to explore the role of family poverty in the
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52 development of psychopathology in children with ASD from preschool age to middle childhood.
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54 Longitudinal data allowed us to get closer to estimating causal relations than would cross-sectional data.
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56 We also explored the role in this association of three factors related to resilient outcomes in typically
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60 ^a Contrary to the DSM-IV-TR hierarchical rules, DSM-5 allows a diagnosis of ADHD alongside an ASD
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62 diagnosis.⁶
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developing children: maternal warmth, maternal involvement, and home organisation. Examining the role of the above factors in the development of psychopathology in the early years in children with ASD may help professionals identify at-risk ASD children early. Interventions for these children may be most useful during early development.¹⁷

Maternal warmth, maternal involvement, and home organisation

There is much evidence for the role of home organisation and maternal warmth and involvement in mental health outcomes in typically developing children.¹⁸⁻²² However, there is little research on their role in psychopathology in atypical populations. With regard to warmth, there is evidence that its effect on behaviour problems in autistic children may be non-linear, with behavioural difficulties being linked both with parenting that is highly intrusive and lacking in warmth and responsiveness, and with emotional over-involvement.²³⁻²⁴ However, warm, sensitive, facilitative parenting has been associated with optimal behavioural development in adolescents and adults with ASD.¹⁰ Regarding involvement, although Osborne et al.²⁵ found no relationship between parental involvement and behaviour problems in autistic children, more recently, Osborne and Reed⁹ found strong evidence of involvement interacting bi-directionally with parenting stress, a powerful risk factor of adjustment problems in children with developmental disabilities.²⁶ Finally, household chaos, a home environment characterised by high levels of noise and disorganisation and low levels of regularity and routine,²⁷ strongly predicts behavioural problems in typically developing²⁷ and autistic²⁸ children. To date, no study has investigated its role in the development of behaviour problems in ASD children. . Previous research with typically developing children has also suggested that warm and involved parenting and home organisation may not only predict adjustment but also promote resilience; that is, better than expected outcomes in children exposed to contextual risk factors^{21,29} . It is possible that similar effects may be evident in the ASD population. To date, no study has tested this.

The present study

It is difficult to isolate the causal impact of SED, as many factors might jointly determine SED and child psychopathology. To avoid attributing to SED what should be attributed to correlated determinants of both SED and psychopathology, we adjusted for maternal qualifications as a proxy for family socio-economic status and an indicator of human capital in the family. We also adjusted for children's verbal cognitive ability and low birthweight status. Verbal cognitive ability is a strong predictor of emotional and

1 behavioural adjustment among children in the general population³⁰ and among those with ASD.¹⁶ It is
2 also related to childhood family poverty.³¹ Low birthweight children are at increased risk of developing
3 psychiatric problems by middle childhood³².
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7 We hypothesised that, both concurrently and over time, SED and household chaos would contribute to
8 heightened levels of psychopathology, whereas higher parental warmth and involvement would be
9 associated with less psychopathology. Also, given their association with resilience among children in the
10 general population, we hypothesised that parental involvement, parental warmth, and home organisation
11 would moderate (i.e., 'buffer') the effect of SED on children's psychopathology.
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20 In our analysis of these relationships, we tested the role of parenting in 'mediating' and 'moderating'³³ the
21 effect of SED. A mediator is a variable that accounts for the relation between the 'independent' and
22 'dependent' variables, establishing 'how' or 'why' the relationship exists. We first examined whether the
23 association between SED (independent) and psychopathology (dependent) could be explained by less
24 maternal warmth, less involvement and less home chaos. In this case, warmth, involvement and home
25 organisation were tested as mediators. . A moderator is a variable that affects the strength and/or direction
26 of the relation between an independent and a dependent variable, establishing 'when' or 'for whom' a
27 relationship exists. We tested whether the effect of SED on psychopathology differed depending on the
28 level of maternal involvement, warmth and household organisation (the 'moderators'). For example, we
29 hypothesized that greater maternal involvement would weaken the relationship between SED and
30 psychopathology such that children with more involved mothers would have fewer problems than their
31 counterparts with less warm mothers. Moderation was examined by specifying interaction terms of SED
32 and the 'moderators'.
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48 **METHOD**

49 **Sample**

50 Data were obtained from the first four sweeps (at ages 9 months, and 3, 5 and 7 years) of the Millennium
51 Cohort Study (MCS), a population-based cohort study of children born in the UK in 2000-2002. MCS
52 was designed to over-represent families living in areas of high child poverty, areas with high proportions
53 of ethnic minority populations across England, and the three smaller UK countries.³⁴ Parent-reported data
54 were collected through interviews and self-completion questionnaires.
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2 At Sweep 1, 18522 families participated, and the numbers of productive families at Sweeps 2, 3, and 4
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4 were 15590, 15246, and 13857, respectively. For families with twins and triplets, we used information
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6 only about the first-born twin or triplet. At Sweep 4, the main caregiver was asked, “Has a doctor or
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8 health professional ever told you that [Cohort child's name] had Autism or Asperger's Syndrome?” The
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10 ‘ASD’ sample (n=209, 174 boys) included children whose main caregiver indicated ‘yes’ to this question.
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12 The ‘non-ASD’ sample of children whose main caregiver answered ‘no’ (n=13737, 6888 boys) was used
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14 for descriptive comparative analyses. Recent research provides strong evidence for the reliability of
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16 parent-reported community diagnosis of ASD.³⁵
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20 **Measures**

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22 *Child psychopathology.* Psychopathology was assessed at Sweeps 2- 4 using the main caregiver’s
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24 report of the Strengths and Difficulties Questionnaire (SDQ),³⁶ a valid and reliable measure of child
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26 psychopathology³⁷ and a useful clinical screening tool for children with autism.³⁸ Total scores were
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28 calculated for each 5-item (on a 3-point scale) subscale of emotional symptoms, conduct problems,
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30 hyperactivity/inattention, and peer relationship problems (specific psychopathologies). A total difficulties
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32 score (broad psychopathology) was calculated by summing the subscale scores. In our ASD sample,
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34 Cronbach’s alpha for total difficulties ranged from .64-.74 across sweeps, indicating adequate internal
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36 consistency. At Sweep 4, subscales had acceptable coefficients (ranging from .73 to .79); in earlier
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38 sweeps, some appeared unreliable (e.g., peer problems ($\alpha=.48$) at Sweep 2)).
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41 *Family socio-economic disadvantage.* Family socio-economic disadvantage (SED) was measured at
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43 Sweeps 2- 4 with a 4-item summative index of family poverty.³⁹ The four dichotomous items were
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45 overcrowding (>1.5 people per room excluding bathroom and kitchen), lack of home ownership, receipt
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47 of income support, and income poverty (below the poverty line). One official poverty line for equivalised
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49 net family income is set at 60% of the UK national median household income. *Parenting.* The parenting
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51 variables hypothesized to buffer poverty effects were reported by the main caregiver at Sweep 2. *Parental*
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53 *warmth* was assessed with the short form of the Child-Parent Relationship Scale.⁴⁰ The scale has 15 items
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55 rated on a 5-point Likert scale measuring closeness and conflict (reverse coded). All items were summed
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57 to form a total score ($\alpha=.77$). *Parental involvement* was assessed using five items (on a 5-point frequency
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59 scale) measuring how often the parent engages with the child in reading, helping to learn the alphabet,
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61 teaching counting, teaching songs/poems/rhymes, and painting/drawing. The level of involvement was
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computed by summing item scores ($\alpha=.60$). A higher score indicated more involvement. *Household chaos* was measured with three items (on 5-point Likert scales) from the Confusion, Hubbub, and Order Scale (CHAOS)⁴¹: “the atmosphere in my home is calm”; “I can’t hear myself think in my home” and “it is really disorganised in our home”. A total score was generated by summing the items. A higher score indicated a more chaotic household ($\alpha=.66$).

Key covariates. The covariates were gender, low birthweight (<2.5 kilos), verbal cognitive ability, and maternal education. Verbal cognitive ability was measured at Sweep 2 with the British Ability Scales II Naming Vocabulary subtest.⁴² The mother’s highest educational qualification as at Sweep 4 was measured using the National Vocational Qualification (NVQ) levels.

Statistical Analysis

To predict children’s psychopathology over time due to SED and parenting (adjusting for covariates), we modelled individual trajectories of emotional and behavioural problems using hierarchical growth curve models⁴³. This approach allowed us to estimate mean trajectories (i.e., growth) of children’s psychopathology from age 3 to 7 by specifying an independent variable for time (in this study, our time variable is age in years centred at the grand mean across sweeps (5.28 years)). Importantly, as children with ASD may differ from each other in the level of psychopathology at different ages, this approach also captures these individual differences in (both linear and non-linear) patterns over time (i.e., growth curves) and therefore deviations from mean trajectories. At the same time, these models capture ‘clustering’ of repeated measures of children’s psychopathology as an individual child’s problems will be correlated across measurement occasions. In this type of model, occasions are considered to be at ‘Level 1’ and children are at ‘Level 2’ as occasions are nested within the child. These models specify both fixed and random growth parameters. The fixed parameters are the intercept (mean psychopathology scores at the average age) and the slope (mean change in scores per annum). The random parameters simultaneously capture the variation in scores between occasions for each child (‘between-occasion variance’), between children at the average age (‘between-child intercept variance’) and the variation in their annual growth (‘between-child slope variance’). The covariance of the variances at the average age and over time tells us whether there is a relationship between children’s scores at around age 5 and their growth between ages 3 and 7.

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The individual trajectories plot of broad psychopathology indicated an overall linear pattern with a steady increase in problems over time and non-parallel trajectories, suggesting that total difficulties varied with age and between children with ASD. Therefore, a two-level linear growth model was deemed appropriate to capture the inter-individual differences in intra-individual change. Because age was centred at the grand mean across sweeps, all main effects predicted psychopathology at the second measurement occasion (around age 5).

Models (Table 1) were carried out for broad and specific psychopathology. Data were analysed using SPSS18 and MLwiN 2.25, a statistical package for multilevel modelling. In descriptive analyses, survey weights were used to account for the MCS survey design and non-response. In multilevel analyses, the stratified sampling design of MCS was recognized by modelling the design variables that accounted for the oversampling of children from particular area types.

(Table 1)

RESULTS

Children with ASD displayed significantly higher rates of broad and specific psychopathology than children without ASD at all ages. Figure 1 and Table S1, available online, show the weighted mean broad and specific psychopathology scores for the ASD and non-ASD groups. The ASD group showed an average increase in broad psychopathology across time and the non-ASD group demonstrated an average decrease. Table 2 shows the descriptive statistics for the key parenting predictors in the two groups. The ASD group had lower levels of maternal warmth and higher levels of household chaos and SED. Scores for maternal involvement did not differ significantly between groups.

(Figure 1 and Table 2)

Linear Growth Curve Regression Models

Broad psychopathology

The main effect of SED on broad psychopathology of ASD children was statistically significant (Model 1, Table 3), with an increase in one element of disadvantage being associated with an increase in roughly one parent-reported difficulty. Its effect on average annual change in difficulties was not statistically significant. Notably, there was a fairly large effect of mother's education. The significant random

1 parameters indicated variation between occasions as well as between children in both average difficulties
2 and in the average linear change over time. Markedly, there was greater between-occasion than between-
3 child variation reflecting the instability of other developmental outcomes (e.g., cognitive) in children with
4 ASD.⁴⁴ Furthermore, the significant positive covariance of the between-child intercept and slope
5 variances suggested that children with more problems at age 5 increased in their problems at a faster rate
6 over time compared with children with fewer problems at age 5.
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11 We then examined the effects of the three parenting variables in Models 2a-2d on problem trajectories.
12 Warmth was significantly associated with total difficulties at age 5 ($b=-.263$, $SE=.082$, Model 2a). A one
13 point increase on the warmth scale at age 3 was associated with an average annual increase of .065 in
14 total difficulties. A trajectories plot demonstrated that ASD children with mothers exhibiting higher levels
15 of warmth had fewer difficulties at age 3 than those with mothers with less warmth. However, at age 5 the
16 difficulties scores of children with less warm mothers appeared to drop. Therefore, the effect of low
17 maternal warmth was stronger in preschool years, weakening thereafter. Moreover, the addition of
18 warmth mediated the relationship between SED and broad psychopathology. Neither the main nor the
19 interaction (by age) effects of involvement (Model 2b) and chaos (Model 2c) were statistically significant.
20 However, the effects of warmth discussed above were robust to adjustment for both involvement and
21 chaos in Model 2d (Table 3). To assess the fit of Model 2d compared with Model 1, we examined the
22 likelihood ratio (LR) test statistic, calculated as two times the difference in the log likelihood values for
23 the two models ($LR=2*(2116.27-1348.28)=1535.98$). The 5% point of a chi-squared distribution on 6 d.f.
24 (as there are 6 parameters' difference between Models 1 and 2d) is 12.60^b. Therefore, Model 2d was a
25 statistically significant improvement on Model 1. Finally, we investigated whether warmth, involvement
26 and chaos moderated the effect of SED on total difficulties at age 5 and over time. None of the
27 interactions between parenting factors and age were statistically significant (Model 3d, Table 3). Model
28 3d (with 6 additional parameters than Model 2d) was not a statistically significant improvement on Model
29 2d ($LR=2*(1348.28-1345.27)=3.01$).
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52 (Table 3)
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59 ^b Even when applying a Bonferroni correction, Model 2d was a better fit than Model 1. Based on our corrected alpha
60 of .025, obtained by dividing the alpha of .05 by 2 for the number of model comparisons, the 2.5% point of a chi-
61 square distribution on 6 d.f. is 14.45.
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Specific psychopathology

1 We then fitted these models on the four SDQ difficulties. In Model 1, the main effect of SED was
2 statistically significant only on emotional symptoms ($b=.131$, $SE=.228$) and conduct problems ($b=.307$,
3 $SE=.109$). There were no significant interactions between SED and age in any psychopathological
4 domain. However, in Model 2d, the effect of SED on emotional symptoms was attenuated by the addition
5 of warmth, chaos, and involvement. Warmth had a significant main (negative) effect on age 5 conduct
6 problems and hyperactivity, and was positively associated with growth in both conduct ($b=.020$,
7 $SE=.009$) and peer problems ($b=.028$, $SE=.011$). Therefore, the effect of warmth on the average trajectory
8 of total difficulties was likely due to the effect of warmth on the average trajectory of conduct and peer
9 problems. In Model 2d there was also a positive main effect of chaos on conduct problems ($b=.114$,
10 $SE=.057$), and a positive main effect of involvement on peer problems ($b=.172$, $SE=.081$). Finally,
11 Models 3a-3d showed that warmth, involvement and chaos did not moderate the effect of SED on the four
12 difficulties at age 5 or on their growth over time.
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DISCUSSION

28 Following a large sample of children with ASD from age 3 to 7, we investigated the role of family
29 poverty and parenting (maternal warmth and involvement, and home organisation) in children's
30 psychopathology trajectories. We also examined the moderating role of parenting in the association
31 between poverty and child psychopathology. Compared to children without ASD, children with ASD
32 showed higher levels of psychopathology as early as age 3 which increased over time, extending previous
33 research findings on co-occurring psychiatric problems in ASD children⁴⁵⁻⁴⁶. Additionally, poverty was
34 associated with broad and specific (conduct problems and emotional symptoms) psychopathology above
35 and beyond child-level characteristics of intellectual ability and low birthweight. Poor families may face
36 barriers to accessing autism-related services⁴⁷; the relationship between poverty and psychopathology
37 may reflect this. Future research might explore the issue of governmental agencies intervening when poor
38 families have a child with ASD. However, poverty was not associated with change over time in any
39 psychopathology, which may be related to the developmental stage of our sample. Poverty has been
40 related to an increase in psychopathology in ASD populations in studies with adults¹⁵ and adolescents.¹⁶
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59 As expected, household chaos was associated with conduct problems. Children with autism require
60 structure and routine in their lives.¹ Families of children with ASD may find it difficult to manage their
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1 children's challenging behaviours and, in turn, have trouble maintaining organisation and routine at
2 home. Families of children with ASD may benefit from support or education that encourages the
3 maintenance of calmness and routine within the home. However, in line with Osborne et al.²⁵, we did not
4 find a strong association between mothers' involvement and children's psychopathology. Parental
5 involvement may operate differently within the ASD population compared with typically developing
6 children. For example, parental involvement, assessed in this study as degree of active or structured
7 interaction with the child, has also been conceptualised as parental accessibility.⁴⁸ Accessibility, not
8 measured in MCS, may be more important to children with ASD, for whom active over-involvement may
9 be disadvantageous. El-Ghoroury and Romanczyk⁴⁹ found a negative association between parental play
10 behaviours and children's social characteristics, suggesting that high levels of interaction can prevent the
11 child with autism from initiating interactions. Alternatively, the severity of children's ASD may be
12 confounded with parents' involvement as children with a more severe disorder may have both more
13 involved parents and greater psychiatric problems. Moreover, parental involvement and parental stress
14 could interact to affect children's displayed difficulties.⁹

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30 Our most notable findings were those pertaining to the role of maternal warmth. Warmth was associated
31 with lower scores in broad and specific (conduct problems and hyperactivity) psychopathology at age 5,
32 and with change in conduct and peer problems over time. These results complement findings from studies
33 indicating the importance of warmth for adjustment in young people with ASD¹⁰ and in the general
34 population.²¹ Importantly, maternal warmth fully explained the effect of poverty on broad
35 psychopathology. Hence, warmth may contribute to the poverty-psychiatric problems pathway in children
36 with ASD. Together, our findings suggest that parental warmth may have important implications for the
37 development of psychiatric symptoms in young children with ASD, especially those living in poverty.

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48 However, we found no evidence to suggest that warmth, involvement and home organisation buffered the
49 effect of poverty on psychopathology in our ASD sample. Studies within the general population have
50 indicated that these factors can promote resilience among children at this developmental stage.^{21,29} As
51 suggested by Simonoff et al.,¹⁶ it is important to consider alternative pathways that may be responsible for
52 psychopathology in children with ASD. For example, children's autistic traits themselves may contribute
53 towards additional psychiatric problems. Furthermore, children with ASD may be predisposed to
54 biological risks that affect their susceptibility to environmental influence.⁵⁰ If so, the buffering role of
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1 family factors on psychiatric symptoms may be harder to underpin. Fortunately, there is increasing
2 exploration of interactive effects within the family that can influence development of children with ASD
3 in terms of core autism symptoms and co-occurring psychopathology, and with regard to maternal well-
4 being¹¹⁻¹². The development of such an evidence-base can contribute to identifying and supporting
5 potentially vulnerable families to promote better child outcomes.
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11 There are a number of study limitations that should be acknowledged. First, children's medical diagnoses
12 of ASD were based on parent reports, which may have limited validity due to reporter bias. Nonetheless,
13 parental reports can have good reliability.³⁵ Second, the severity of autism was unknown. Severity can
14 contribute significantly to the degree of psychiatric problems displayed.⁵¹ Third, the SDQ does not
15 identify all areas of additional difficulties in the ASD population, such as sleeping problems and emotion
16 regulation deficits.⁵² Fourth, some of our measures, such as parental involvement, did not demonstrate
17 strong internal consistency and have not undergone psychometric validation. Fifth, caution should be
18 taken when generalising this study's findings to all families of children with ASD. Last, our study
19 focused on one risk factor (poverty) ignoring co-occurring adversities such as abuse and parental mental
20 illness⁵³. Future research should address the effects of multiple and cumulative risk on psychopathology
21 of children with ASD.
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36 Despite these limitations, our study has several strengths. It included a large sample of young children
37 with ASD drawn from a population rather than a clinically referred sample. Its prospective longitudinal
38 design allowed for a rigorous examination of family-level predictors of children's early trajectories of
39 psychopathology. Furthermore, the inclusion of a non-ASD group for initial exploratory analyses allowed
40 for group comparisons, and helped set the scene for further multilevel analysis within the ASD sample.
41 Finally, although this study did not find that parenting buffered the effect of poverty on psychopathology,
42 it underscored the importance of family experiences in the early life of children with ASD.
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Table 1

Model summary

| Model | Specification |
|----------|--|
| Model 1 | Design variables + covariates ^a + age + SED + (SED x age) |
| Model 2a | Model 1 + warmth + (warmth x age) |
| Model 2b | Model 1 + involvement + (involvement x age) |
| Model 2c | Model 1 + chaos + (chaos x age) |
| Model 2d | Model 1 + warmth + (warmth x age) + involvement + (involvement x age) + chaos + (chaos x age) |
| Model 3a | Model 2d + (warmth x SED) + (warmth x SED x age) |
| Model 3b | Model 2d + (involvement x SED) + (involvement x SED x age) |
| Model 3c | Model 2d + (chaos x SED) + (chaos x SED x age) |
| Model 3d | Model 2d + (warmth x SED) + (warmth x SED x age) + (involvement x SED) + (involvement x SED x age) + (chaos x SED) + (chaos x SED x age) |

Note: ‘x’ indicates interaction. SED = Socio-economic disadvantage. ^aGender, verbal IQ, low birthweight, and maternal qualifications. Models 1 and 2d are nested, as are Models 2d and 3d. Likelihood ratio test comparisons were made for these two sets of models.

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Table 2

Key predictors of psychopathology at age 3 for ASD and non-ASD groups

| Variable | ASD | | | Non-ASD | | | <i>t</i> | <i>df</i> |
|-----------------------------|----------|----------|-----------|----------|----------|-----------|----------|-----------|
| | <i>n</i> | <i>M</i> | <i>SD</i> | <i>n</i> | <i>M</i> | <i>SD</i> | | |
| Warmth | 133 | 59.26 | 7.71 | 10386 | 64.61 | 6.76 | -7.98** | 134.62 |
| Involvement | 122 | 21.26 | 2.76 | 9480 | 21.01 | 2.80 | 0.99 | 9600.00 |
| Chaos | 181 | 7.62 | 2.45 | 12491 | 7.00 | 2.18 | 3.43* | 184.12 |
| Socio-economic disadvantage | 158 | 1.08 | 1.21 | 10661 | 0.81 | 1.13 | 2.95* | 10817.00 |

Note: * $p < .01$; ** $p < .001$.

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Table 3

Growth curve models of broad psychopathology

| Predictors | Model 1 | | Model 2d | | Model 3d | |
|-------------------------|----------------------|-----------|----------|-----------|----------|-----------|
| | b | se | b | se | b | se |
| | Fixed effects | | | | | |
| SED | 0.949* | 0.394 | 0.683 | 0.470 | -1.335 | 7.043 |
| SED x age | 0.034 | 0.170 | 0.135 | 0.209 | -2.164 | 3.032 |
| Warmth | | | -0.263** | 0.082 | -0.266* | 0.107 |
| Warmth x age | | | 0.065* | 0.032 | 0.038 | 0.041 |
| Involvement | | | 0.408 | 0.238 | 0.391 | 0.280 |
| Involvement x age | | | 0.066 | 0.090 | 0.038 | 0.110 |
| Chaos | | | 0.426 | 0.229 | 0.408 | 0.276 |
| Chaos x age | | | 0.005 | 0.088 | 0.063 | 0.104 |
| Warmth x SED | | | | | 0.013 | 0.070 |
| Warmth x SED x age | | | | | 0.028 | 0.030 |
| Involvement x SED | | | | | 0.037 | 0.224 |
| Involvement x SED x age | | | | | 0.054 | 0.095 |
| Chaos x SED | | | | | 0.040 | 0.195 |
| Chaos x SED x age | | | | | -0.069 | 0.083 |

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|-----------------------------------|-----------|-------|-----------|-------|-----------|-------|--|
| England-advantaged (<i>ref</i>) | | | | | | | |
| England-disadvantaged | 1.036 | 1.244 | -0.611 | 1.223 | -0.609 | 1.268 | |
| England-ethnic | 4.319 | 3.248 | 5.030 | 3.467 | 5.401 | 3.708 | |
| Wales-advantaged | 3.853 | 3.463 | 3.033 | 7.479 | 2.683 | 7.591 | |
| Wales-disadvantaged | 0.893 | 1.649 | 2.556 | 1.864 | 2.650 | 1.875 | |
| Scotland-advantaged | 2.942 | 1.846 | 4.529* | 1.894 | 4.548* | 1.912 | |
| Scotland-disadvantaged | -0.850 | 1.732 | -0.182 | 1.470 | 0.116 | 1.494 | |
| Northern Ireland-advantaged | -3.363 | 2.757 | -1.010 | 2.492 | -0.802 | 2.601 | |
| Northern Ireland-disadvantaged | -0.393 | 2.379 | 1.288 | 2.303 | 1.733 | 2.336 | |
| Age | 0.893*** | 0.242 | -4.490 | 2.753 | -2.275 | 3.302 | |
| Female | -2.110 | 1.466 | -1.052 | 1.471 | -1.201 | 1.519 | |
| Verbal ability | -0.074 | 0.040 | -0.091* | 0.041 | -0.088* | 0.041 | |
| Low birthweight | 0.157 | 2.361 | -3.408 | 4.253 | -3.320 | 4.276 | |
| NVQ level 1 (<i>ref</i>) | | | | | | | |
| NVQ level 2 | -5.256* | 2.354 | -10.830* | 4.428 | -11.163* | 4.469 | |
| NVQ level 3 | -3.291 | 2.493 | -9.586* | 4.502 | -10.172* | 4.573 | |
| NVQ level 4 | -5.962* | 2.412 | -11.716** | 4.442 | -12.194** | 4.485 | |
| NVQ level 5 | -8.346** | 2.883 | -14.042** | 4.744 | -14.118** | 4.778 | |
| Constant | 22.846*** | 2.919 | 33.275*** | 7.747 | 34.208*** | 9.042 | |

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| | Random effects | | | | | |
|---|-----------------------|-------|-----------|-------|-----------|-------|
| Between-child intercept variance | 18.810*** | 3.533 | 15.739*** | 3.755 | 16.422*** | 3.835 |
| Between-child slope variance | 1.269* | 0.609 | 1.389 | 0.700 | 1.334 | 0.683 |
| Between-child intercept-slope variance covariance | 2.544** | 0.955 | 5.026*** | 1.190 | 5.512*** | 1.196 |
| Between-occasion variance | 19.985*** | 2.784 | 18.360*** | 3.125 | 18.037*** | 3.068 |
| Log likelihood | 2116.274 | | 1348.282 | | 1345.271 | |

Note: *p<.05; **p<.01; ***p<.001. SED=Socio-economic disadvantage. NVQ=National Vocational Qualification. England advantaged-Northern Ireland-disadvantaged=MCS design variables.

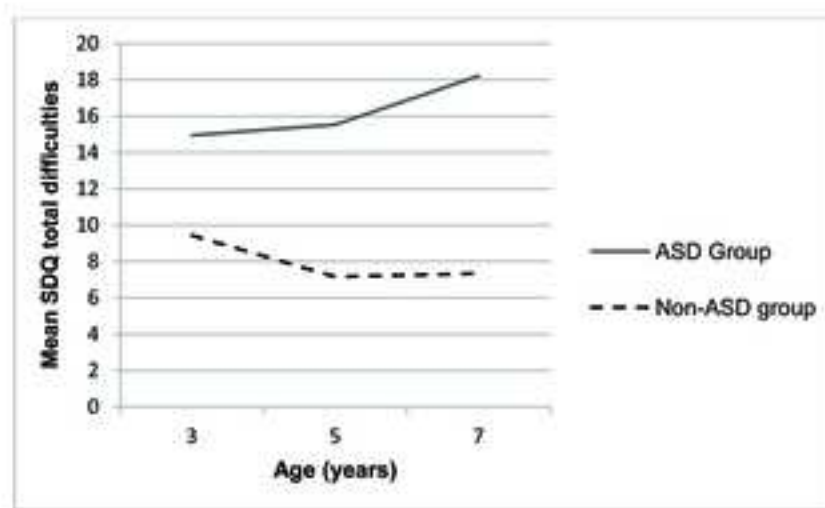


Figure 1

Weighted mean trajectories of broad psychopathology for children in the ASD and non-ASD groups

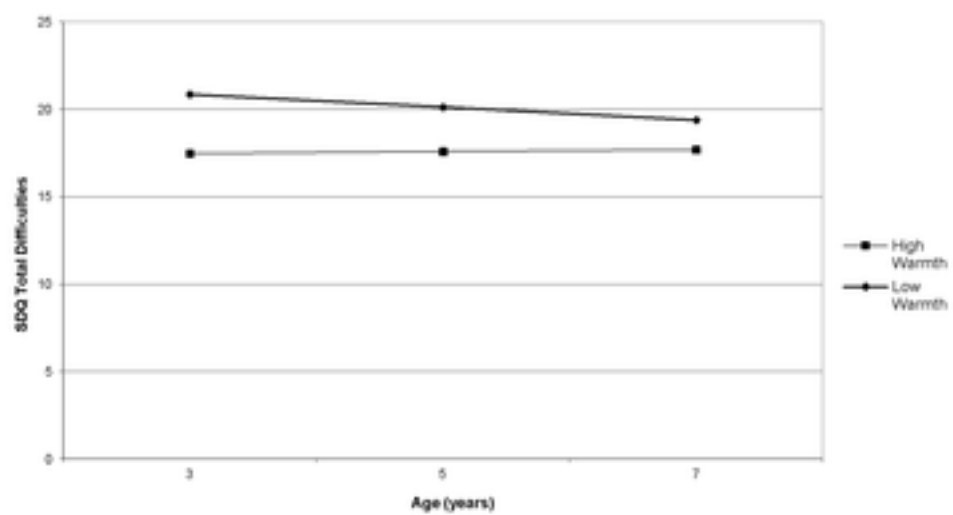


Figure S1.
Predicted trajectories of children with ASD by high/low warmth

Table S1

Broad and specific psychopathology at time 1 (3 years), time 2 (5 years) and time 3 (7 years) for the ASD and non-ASD groups

| Variable | ASD group | | | Non-ASD group | | | <i>t</i> | <i>df</i> | 95% <i>CI</i> |
|---------------------------|--------------------------|----------|-----------|---------------|----------|-----------|----------|-----------|---------------|
| | <i>n</i> | <i>M</i> | <i>SD</i> | <i>n</i> | <i>M</i> | <i>SD</i> | | | |
| Time 1 | Broad Psychopathology | | | | | | | | |
| Total Difficulties | 170 | 14.95 | 6.04 | 11705 | 9.44 | 5.20 | 11.83* | 172.66 | [4.59, 6.43] |
| | Specific Psychopathology | | | | | | | | |
| Emotional Symptoms | 175 | 1.93 | 1.82 | 11959 | 1.35 | 1.47 | 4.17* | 177.39 | [0.30, 0.85] |
| Conduct Symptoms | 175 | 3.91 | 2.10 | 11980 | 2.78 | 2.05 | 7.25* | 12153.00 | [0.82, 1.44] |
| Hyperactivity/Inattention | 175 | 6.09 | 2.56 | 11866 | 3.85 | 2.34 | 11.46* | 178.32 | [1.85, 2.62] |
| Peer Problems | 170 | 3.11 | 2.16 | 11892 | 1.51 | 1.57 | 9.64* | 171.59 | [1.27, 1.93] |
| Time 2 | Broad Psychopathology | | | | | | | | |
| Total Difficulties | 190 | 15.53 | 7.03 | 12647 | 7.16 | 4.83 | 16.36* | 191.69 | [7.36, 9.38] |
| | Specific Psychopathology | | | | | | | | |
| Emotional Symptoms | 192 | 2.60 | 2.36 | 12747 | 1.37 | 1.57 | 7.22* | 193.57 | [0.70,1.60] |
| Conduct Symptoms | 194 | 2.90 | 1.77 | 12763 | 1.48 | 1.48 | 11.12* | 197.12 | [1.17,1.68] |
| Hyperactivity/Inattention | 194 | 6.49 | 2.80 | 12696 | 3.22 | 2.33 | 16.20* | 197.10 | [2.87, 3.67] |
| Peer Problems | 192 | 3.63 | 2.43 | 12740 | 1.12 | 1.40 | 14.24* | 192.90 | [2.16, 2.85] |
| Time 3 | Broad Psychopathology | | | | | | | | |

| | | | | | | | | | |
|---------------------------|-----|-------|------|-------|------|------|--------|----------|---------------|
| Total Difficulties | 201 | 18.21 | 7.34 | 13326 | 7.34 | 5.27 | 20.91* | 203.12 | [9.84, 11.89] |
| Specific Psychopathology | | | | | | | | | |
| Emotional Symptoms | 204 | 3.65 | 2.59 | 13403 | 1.51 | 1.74 | 11.75* | 205.79 | [1.78, 2.50] |
| Conduct Symptoms | 204 | 3.21 | 2.20 | 13432 | 1.37 | 1.52 | 11.90* | 205.94 | [1.54, 2.15] |
| Hyperactivity/Inattention | 204 | 7.16 | 2.56 | 13382 | 3.31 | 2.48 | 22.00* | 13584.00 | [3.51, 4.19] |
| Peer Problems | 201 | 4.23 | 2.43 | 13415 | 1.19 | 1.50 | 17.72* | 202.28 | [2.70, 3.38] |

Note: *p<.001.