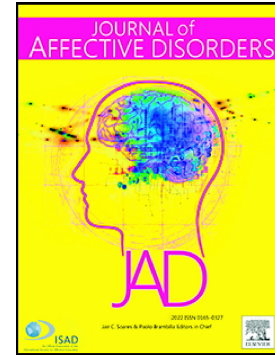


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# Sex differences in internalising problem trajectories of autistic and non-autistic youth across childhood and adolescence

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## Abstract

**Background:** Autistic children and adolescents experience high rates of internalising problems such as anxiety and depression. In the general population, female children and adolescents typically show higher trajectories of internalising symptoms – but little is known about sex differences among autistic youth. This study examined sex

differences in the developmental course of internalising symptoms among autistic and non-autistic youth across childhood and adolescence.

**Methods:** Participants included autistic (n = 573) and non-autistic (n = 15,945) individuals from the Millennium Cohort Study. Internalising symptoms were assessed via parent-report Strengths and Difficulties Questionnaire at six timepoints (ages 3-17). Latent Growth Curve Modelling (LGCM) was used to model trajectories, and growth factors were regressed onto autism diagnosis, sex, and their interaction. Additionally, separate LGCMs were fitted to autistic and non-autistic male and female groups to describe internalising trajectories.

**Results:** Autism diagnosis was associated with higher baseline internalising symptoms and steeper increases over time. Female sex was linked to steeper increases in internalising symptoms, despite slightly lower initial levels. The interaction between sex and autism diagnosis was not statistically significant, indicating no additional combined effect on trajectories. Overall, autistic youth had higher internalising trajectories than non-autistic youth, with group differences emerging earlier in males (age 7) than females (age 11).

**Conclusion:** This study showed that that sex differences in developmental trajectories of internalising problems among autistic youth parallel those seen in the general population, but with increased severity.

## Keywords

Autism; Internalising Problems; Mental Health; Sex Differences; Adolescence; Millennium Cohort Study; Developmental Trajectories.

## Introduction

Autism is a lifelong neurodevelopmental condition characterised by differences in social communication and sensory processing, intense or focused interests, and a preference for certainty, routines, and sameness (American Psychiatric Association, 2013). Mental health difficulties are common among autistic individuals, with internalising problems such as anxiety and depression particularly prevalent. Estimated rates of anxiety and depressive disorders in this population range from 20–39% and 11–21% respectively – substantially higher than global estimates of 6.5% and 2.6% in the general population (Lai et al., 2019; Micai et al., 2023; Polanczyk et al., 2015). These internalising difficulties typically emerge during childhood, and are linked to serious adverse outcomes for autistic individuals, including higher risks of suicide and premature mortality (Cassidy et al., 2018; Schendel et al., 2016). They are also associated with behavioural challenges such as greater social difficulties, aggression, and self-injurious behaviour (Bellini, 2004; Stewart et al., 2006; Wood et al., 2009).

Sex and gender are key factors known to influence internalising problems in the general population. Sex refers to the biological differences between being male, female, or intersex, whereas gender reflects socially constructed roles and identities (Chrisler & Lamer, 2016). Ideally, the impacts of sex and gender on mental health would be examined separately; however, many studies do not make this distinction, and the two are difficult to disentangle due to gendered socialisation from birth (Christiansen et al.,

2022; Lai et al., 2015). Therefore, we use the term "sex/gender" when reviewing the literature. Research consistently shows that females are at greater risk of developing internalising problems and tend to experience them with greater severity (Altemus et al., 2014; Christiansen et al., 2022; Martel, 2013). Although the underlying mechanisms are not fully understood, contributing factors may include biological influences (e.g., sex hormones), gender-based discrimination, societal pressures related to appearance, and the challenges of balancing economic and political participation with traditional gendered roles and expectations (Baños & Miragall, 2024; Byrne et al., 2017; Campbell et al., 2021).

The role of sex/gender in shaping internalising problems among autistic individuals remains poorly understood. Rates of autism diagnosis are substantially lower in females than in males, with a female-to-male ratio of approximately 1:4 (Halladay et al., 2015). One potential explanation is that autistic females more often engage in 'camouflaging' – suppressing or masking autistic traits to fit in socially – which can make autism harder to identify in this group, and is incidentally associated with worse mental health (Hull et al., 2020). A recent meta-analysis identified only eight studies on sex/gender differences in internalising problems among autistic children, with mixed results wherein some reported higher levels in females, others in males, and several studies showed no differences (Natoli et al., 2023). Although the meta-analysis reported no significant overall sex/gender differences, the small evidence base available may have limited statistical power. Moreover, the included studies largely focused on early childhood (ages 1–6), precluding investigation of differences that

emerge later. In the general population, sex/gender differences in internalising problems widen over time, particularly in adolescence when females show heightened vulnerability (Altemus et al., 2014). Thus, further research spanning childhood through adolescence is required.

A key limitation of existing research is its reliance on cross-sectional designs, which overlooks developmental stage, and may partly explain inconsistent findings. Longitudinal studies in the general population mainly show that females experience a steeper incline in internalising symptoms across childhood and adolescence compared to males (Papachristou & Flouri, 2020; Sterba et al., 2007; Toumbourou et al., 2011). Although research on internalising trajectories is increasing, few studies have examined sex differences within autistic samples. These generally mirror the general population, with autistic females showing higher baselines, steeper increases, and higher overall trajectories than males (Corbett et al., 2024; Vaillancourt et al., 2017; Wright et al., 2023). However, these studies largely focus on narrow age-ranges in childhood, with limited investigation of changes across adolescence and beyond. One study examined changes between 6 and 24 years, finding that autistic females showed greater increases in internalising symptoms throughout adolescence than males, though sex/gender differences were no longer observed by young adulthood (Gotham et al., 2015). However, this and several of the earlier studies often relied on clinical samples with few female participants (12–18%), likely reflecting lower diagnosis rates in females (Gotham et al., 2015; Vaillancourt et al., 2017; Wright et al., 2023). They also frequently lacked

non-autistic comparison groups, limiting assessment of whether observed patterns are autism-specific or reflect broader developmental trends.

Understanding how internalising problems develop in autistic populations, and whether these trajectories differ from those of non-autistic individuals, is important for clarifying underlying mechanisms and identifying individuals at greatest risk before difficulties become entrenched (Colizzi et al., 2020). However, few studies have examined these patterns systematically across childhood and adolescence, particularly within general population cohorts where sex/gender-based diagnostic disparities may be less pronounced than in clinical samples

The primary aim of this study was to characterise and compare developmental trajectories of internalising problems among autistic and non-autistic males and females in the Millennium Cohort Study (MCS) – a large, population-based cohort of children and young people in the United Kingdom. Specifically, our objectives were 1. to assess the influence of autism diagnosis and sex on the initial levels and developmental changes in internalising problems across childhood and adolescence, 2. to characterise internalising problem trajectories in autistic and non-autistic males and females, and 3. to compare levels of internalising problems between autistic and non-autistic males and females across childhood and adolescence.

## **Methods**

### **Participants**

The sample came from the UK Millennium Cohort Study (MCS), a population-based cohort of ~19,500 children born between September 2000 and January 2002, with data collected at 9 months and ages 3, 5, 7, 11, 14, and 17. Eligibility required UK residence at 9 months and receipt of Child Benefit (universal benefit at the time for UK children). Autism diagnosis was parent-reported at ages 5, 7, 11, and 14 (“Has a doctor or other health professional ever told you that your child had Autism, Asperger’s Syndrome or other autistic spectrum disorder?”). Participants with a stable diagnosis (reported autistic at the final available sweep) were classified as autistic, while unstable cases ( $n = 50$ ) were coded non-autistic. After excluding 2,967 with missing diagnostic data, the analytic sample included 573 autistic and 15,945 non-autistic participants. Sex at birth was parent-reported at 9 months (or 5 years if missing in first sweep), producing four groups: autistic males, autistic females, non-autistic males, and non-autistic females. Ethical approval for the MCS was granted by NHS committees at each sweep, with written parental consent obtained. The present study was approved by the UCL Research Ethics Committee (19439/001).

## Measures

### Internalising problems

Internalising problems were assessed with the parent-report Strengths and Difficulties Questionnaire (SDQ) a validated and reliable screening tool for emotional and behavioural difficulties for child and adolescent populations, including autistic samples (Goodman, 1997; Goodman & Scott, 1999; Simonoff et al., 2013). The SDQ



includes five subscales: emotional problems, peer problems, conduct problems, inattention and hyperactivity, and prosocial behaviour. Although internalising is sometimes measured by summing emotional and peer problems, autistic youth consistently score higher on peer problems (Petrina et al., 2014), which may inflate internalising estimates. Therefore, we used only the emotional problems subscale (0–10), which has demonstrated good validity and reliability in the MCS ( $\alpha = 0.71$ – $0.79$  across ages 3–17 (Gutman & Codioli McMaster, 2020)).

### **Covariates**

Analyses included selected sociodemographic and perinatal covariates from based on prior evidence linking them to autism and internalising difficulties (see Appendix 1 for details). Sociodemographic factors comprised ethnicity, total banded household income, and highest parental education (Essex et al., 2006; Lund et al., 2010). Perinatal factors included birth weight, delivery type (e.g., vaginal, caesarean), admission to a special baby unit, gestational age, and maternal smoking or alcohol use during pregnancy (Dekel et al., 2019; Getahun et al., 2017). Models were also adjusted for ADHD diagnosis and IQ, given their established associations with autism and internalising symptoms (Edirisooriya et al., 2021; Mayes et al., 2012; Reiersen & Todd, 2008; Steinhausen et al., 2006).

### **Statistical analysis**

Latent growth curve modelling (LGCM) was used to estimate the trajectories of internalising problems at ages 3, 5, 7, 11, 14, and 17. To assess the influence of autism diagnosis, sex, and their interaction on internalising problem trajectories, the intercept and slope factors were regressed on sex, autism diagnosis, and their interaction in the full cohort. Although sex/gender differences in internalising problem trajectories have previously been identified, the evidence largely comes from cohort studies based outside the UK that are mostly focused on adolescence or adulthood (Musliner et al., 2016; Shore et al., 2018). This is insufficient to base an assumption that that similar patterns would necessarily emerge over a period encompassing childhood as well as adolescence (3-17 years) in a UK general population cohort. Therefore, rather than estimating separate trajectories for males and females, we initially analysed the full sample. This approach also conserved statistical power and yielded a more parsimonious model structure which is recommended since accuracy and stability of LGCM parameter estimates are strongly influenced by sample size (Diallo et al., 2014; Fan, 2003). Further, via the modelling of the autism diagnosis-by-sex interaction term, our approach allowed us to directly test whether any observed sex differences differed between young people with and without an autism diagnosis. Linear, quadratic, and cubic forms were tested using SRMR and  $RMSEA < 0.06$  and  $CFI > 0.90$  (Kline, 2023). The linear model fit well and was chosen for parsimony (Appendix 2).

To explore descriptive patterns, separate LGCMs were fitted for autistic and non-autistic males and females. Cubic models fit best overall, but convergence issues arose in the non-autistic groups. Therefore, cubic models were retained for autistic groups,

and quadratic models were retained for the non-autistic groups (Appendix 3). Group comparisons used model-predicted means at each age, with t-tests assessing sex and diagnostic differences.

Covariates were added stepwise (Appendix 4): Step 1 unadjusted; Step 2 adjusted for sociodemographic and perinatal factors (ethnicity, income, parental education, birth weight, delivery type, special baby unit, gestation, maternal smoking and alcohol use); Step 3 additionally adjusted for ADHD and IQ. Step 3 served as the final model, with earlier steps reported in Appendices 5–8.

Sensitivity analyses included participants with unstable diagnoses, producing larger autistic samples and similar results (Appendices 9–12). Data cleaning and descriptive analyses were conducted in Stata 18.0 (StataCorp, College Station, TX, USA), and LGCM in Mplus 8.0 (Muthen, 2002).

### **Missing data**

Missing data on internalising problem scores across the six time points were handled using Full Information Maximum Likelihood (FIML) estimation, the default approach in MPlus for latent growth curve modelling. This method utilises all available data in the sample, including both fully observed and partially missing cases to estimate parameters by maximising the likelihood function (Enders & Bandalos, 2001). Therefore, participants with at least one time point of internalising data were included when estimating trajectories. Across the whole sample, the rates of missingness ranged between 23.4 - 35.2% for the first three time points and 41.4-51.9% for the

subsequent time points. In addition, multiple imputation was conducted in MPlus to handle missing covariate data with 50 datasets generated, and results pooled across datasets.

## Results

### Sample characteristics

The demographic characteristics and sample sizes for each group are displayed in Table 1. The sample consisted of 433 (2.7%) autistic males, 132 (0.8%) autistic females, 7912 (48.4%) non-autistic males, and 7864 (48.1%) non-autistic females. Rates of ADHD diagnosis within the autistic male (34.4%) and female (17.4%) groups were aligned with rates reported in other studies (Bougeard et al., 2021). Similarly, ADHD diagnosis rates in the non-autistic male (2.3%) and female (0.6%) groups were in line with UK diagnostic rates (Hire et al., 2018).

Demographic characteristics were compared between overall autistic and non-autistic groups, overall males and females, and between males and females within autistic and non-autistic groups (see Appendix 13 for full comparisons). In brief, compared to the overall non-autistic group, the autistic group had significantly lower representation of ethnically minoritised individuals, higher proportion of parents in low-income brackets, higher ADHD diagnosis rates, and lower IQ. Additionally, non-autistic females had significantly higher IQ than non-autistic males.

## Trajectories of internalising problems

### Regression of internalising problem trajectories on sex and autism diagnosis

The complete results for the regression analyses are presented in Table 2. The overall trajectory of internalising problems in the full cohort showed a slight decline over time, as indicated by a small but significant negative linear slope ( $M_{\text{intercept}} = 2.10$ ,  $SE = 0.09$ ,  $p < 0.001$ ;  $M_{\text{linear}} = -0.08$ ,  $SE = 0.03$ ,  $p < 0.05$ ). As shown in Table 2, regressing the growth factors on to autism diagnosis revealed that autistic individuals had significantly higher starting levels of internalising symptoms and a steeper increase over time compared to non-autistic individuals. Sex was also significantly associated with both growth parameters such that females started with slightly lower internalising scores than males but showed a significantly steeper increase over time. The interaction between sex and autism diagnosis was not significantly associated with either the intercept or slope, indicating that the combined effect of sex and autism did not significantly explain variation in initial symptom levels or rates of change, beyond the individual contributions of each factor.

### Sex differences in internalising problem trajectories within the autistic group

Trajectories of internalising problems for autistic and non-autistic males and females are presented in Figure 1. The internalising problem trajectory in autistic males initially showed a trend of declining symptoms between ages 3 and 5, followed by a gradual increase through mid-adolescence, and a sharp decline from ages 14 to 17. In contrast, autistic females showed a steady increasing trend in internalising symptoms

from early childhood through adolescence. The trajectory for autistic females initially appeared lower, but then surpassed autistic males after age 5, with divergence becoming more pronounced from age 11 onwards. These patterns were broadly reflected in the growth parameter estimates for each group, but the slope estimates did not reach statistical significance (see Table 3 for full estimates). Similarly, although model-predicted mean differences in internalising scores between autistic males and females appeared to widen over time, these differences were not significant (Table 4).

### **Sex differences in internalising problem trajectories within the non-autistic group**

Non-autistic males had a trajectory of low internalising problems which remained stable over time, as indicated by the non-significant linear ( $M_L = 0.16$ ,  $SE = 0.13$ ,  $p \geq 0.05$ ; Table 3) and quadratic ( $M_{Q2} = -0.33$ ,  $SE = 0.025$ ,  $p \geq 0.05$ ; Table 3) slope parameter estimates. Non-autistic females also initially displayed a low and stable trajectory, but a trend of gradual acceleration was observed starting from age 11 going into adolescence. In line with this, there were initially no significant differences in model-predicted mean internalising problems between the two groups (see Table 4 for full estimates), but non-autistic males had significantly lower scores than non-autistic females at ages 14 ( $M_{diff} = -0.93$ ,  $p < 0.001$ , 95% CI [-1.38, -0.49]) and 17 years ( $M_{diff} = -1.75$ ,  $p < 0.001$ , 95% CI [-2.37, -1.12]).

### **Sex differences in internalising problem trajectories between autistic and non-autistic groups**

There was a shared trend across autistic and non-autistic groups of females initially showing similar levels of internalising symptoms to males in early childhood, followed by a divergence in trajectories at approximately age 11, with females then showing greater increases in internalising symptoms and surpassing males going into adolescence. However, internalising trajectories appeared generally higher for autistic males and females compared to non-autistic males and females. Among females, significant differences in internalising symptoms between autistic and non-autistic groups first emerged at age 11, with autistic females showing higher scores ( $M_{\text{diff}} = 3.25$ ,  $p = 0.004$ , 95% CI [1.02, 5.44]; Table 4). These differences remained significant at each subsequent time point, peaking at age 17 ( $M_{\text{diff}} = 4.63$ ,  $p = 0.02$ , 95% CI [0.69, 8.48]; Table 4). Among males, significant differences in internalising symptoms between autistic and non-autistic groups emerged earlier, at age 7, with autistic males exhibiting higher scores ( $M_{\text{diff}} = 1.14$ ,  $p = 0.02$ , 95% CI [0.19, 2.64]; Table 4). These differences persisted across all subsequent time points, reaching their peak at age 14 ( $M_{\text{diff}} = 3.43$ ,  $p < 0.001$ , 95% CI [2.03, 4.65]; Table 4).

## Discussion

This study characterised sex differences in trajectories of internalising problems across childhood and adolescence, both within and between autistic and non-autistic groups, using data from a large, nationally representative cohort. We found that having an autism diagnosis was associated with higher baseline levels of internalising symptoms, and steeper increases over time. Similarly, female, compared to male sex

also predicted steeper increases in internalising problems over time, though it was associated with lower initial levels. Notably, we did not find evidence that the combination of being both autistic and female had a more severe impact on trajectories beyond the independent effects of each, which was somewhat unexpected. Since this may reflect limited power, we characterised and compared internalising problem trajectories between autistic and non-autistic males and females to capture group-level descriptive patterns. Visually, internalising problem trajectories of autistic males and females appeared overall higher than non-autistic males and females starting from a young age. We also observed significant group differences between autistic and non-autistic groups, with these differences emerging earlier in males (at age 7) than in females (at age 11). Overall, this study provides new evidence that sex differences in developmental trajectories of internalising problems among autistic youth may resemble those observed in the general population, albeit at a higher level.

Our findings contribute to a growing body of evidence showing that autistic young people experience greater internalising difficulties than their non-autistic peers starting from childhood and persisting into adolescence (Colvert et al., 2022; Cronshaw & Midouhas, 2023; Mandy et al., 2022; Rai et al., 2018). We found that differences in internalising symptoms between autistic and non-autistic groups may emerge in late childhood (age 7) among males, and early adolescence (age 11) among females. The earlier emergence of group differences in males compared to females could potentially be explained by the fact that higher camouflaging in females may result in more subtle or socially masked internalising symptoms earlier in childhood, with difficulties



becoming more apparent only as the social and emotional demands of adolescence increase (Bargiela et al., 2016). Supporting this, studies have shown that autistic individuals who camouflage more appear to experience fewer difficulties to external observers compared to their self-reported levels of mental ill health (Corbett et al., 2021; Livingston et al., 2019). Furthermore, the increasing complexity of social interactions during adolescence may pose greater challenges for autistic females than males, as adolescent girls often face heightened expectations regarding social performance during this period (Tomlinson et al., 2020). Overall, this suggests that both autistic males and females will benefit from close monitoring for internalising problems such as depression and anxiety starting from childhood.

We also observed that males may, on average, begin with higher levels of internalising problems in early childhood compared to females. Although this pattern likely reflects a complex interplay between biopsychosocial factors, one plausible explanation may involve gender socialisation, research shows that girls are more often encouraged to express sadness than boys (Cassano et al., 2007; Chaplin et al., 2005). As a result, boys may be socialised to suppress expressions of internalising difficulties, such as anxiety and sadness, as they grow older, potentially leading to lower parental ratings of these symptoms. Although less is known about emotion socialisation specifically in autistic children and young people, evidence suggests that parents of autistic and non-autistic children engage in similar emotion socialisation behaviours (Jordan et al., 2021).

There was a steep increase in internalising problems in females from older childhood to adolescence. In general, adolescence is a developmental period associated with a range of challenges that may exacerbate vulnerability to internalising difficulties such as increased social pressures, heightened sensitivity to peer rejection, and transitions to more demanding environments such as secondary school (Andrews et al., 2021). In females, this is further intensified by earlier pubertal maturation, which is associated with heightened sensitivity to interpersonal stress, increased risk of sexual harassment and abuse, body image issues, and poorer emotion regulation – factors that may interact to exacerbate internalising difficulties during adolescence (Angold et al., 1998; Rodriguez-Tomé et al., 1993; Rudolph & Flynn, 2007; Skoog & Özdemir, 2016). This is likely to be even more pronounced in autistic girls compared to non-autistic girls, given that they experience higher levels of peer problems, are more vulnerable to victimisation, and are reported to start puberty earlier on average (Corbett et al., 2022; Douglas & Sedgewick, 2024; Petrina et al., 2014). Supporting this, one study found that the effects of age and autism on increasing internalising problems in adolescents was partially mediated by the interaction between autism and puberty, whereby depressive scores were elevated in autistic youth in early puberty and decreased with advancing pubertal stage (Corbett et al., 2024).

Although group differences between autistic males and females did not reach statistical significance, we observed a crossover trend, whereby males initially showed slightly higher symptoms in early childhood, but females demonstrated a sharper increase, overtaking males by adolescence when male symptoms declined. It is

possible that this pattern may reflect meaningful underlying mechanisms that were not detectable due to limited statistical power. Indeed, a similar pattern of internalising symptoms was also observed in another study, although the crossover in trajectories occurred later – between ages 17 and 18 – compared to age 11 in the current study (Gotham et al., 2015). This crossover effect may help explain why previous studies – many of which included wide age ranges – have reported inconsistent or non-significant sex differences in internalising problems among autistic individuals, since it is possible that opposing trends may cancel each other out (Nasca et al., 2020).

The observed trend of steep increases in internalising symptoms among autistic females compared to autistic males during adolescence is also consistent with findings from several other studies reporting similar patterns (Corbett et al., 2024; Fombonne et al., 2022; Gotham et al., 2015; Penner et al., 2022, 2022; Solomon et al., 2012). There are several reasons to expect that autistic females may experience more challenges over this period. For instance, insistence on sameness – referring to preference for order, predictability, or routine – is a common feature of autism that is thought to increase with age, and aspects of cognitive inflexibility such as insistence on sameness are shown to exacerbate both social difficulties and internalising problems (Baribeau et al., 2021; Richler et al., 2010). This may be even more pronounced in autistic females, since they are reported to be more socially motivated, but also experience greater increases in social difficulties than autistic males in adolescence (Mandy et al., 2018; Sedgewick et al., 2016). Furthermore, autistic females are also thought to be more susceptible to interpersonal stress compared to autistic males (Nenniger et al., 2021).

Compounded, these factors could result in autistic females facing more social rejection, resulting in higher levels of interpersonal stress, and consequently experiencing greater internalising difficulties compared to autistic males.

The above factors may also contribute to the higher likelihood of camouflaging among autistic females to gain social acceptance (Bernardin et al., 2021; Hull et al., 2020; Wood-Downie et al., 2021). However, camouflaging is associated with increased internalising problems, and there is evidence that autistic females find it particularly stressful (Hull et al., 2020). Another consequence of increased camouflaging is the potential delay in receiving an autism diagnosis. In line with this, autistic girls are often diagnosed later than autistic boys (Gould & Ashton-Smith, 2011; Zener, 2019). This could mean that by adolescence, autistic girls have received less support and school-based adjustments compared to autistic boys, which could negatively impact school performance and self-esteem, thus, further fuelling internalising problems (Deniz & Toseeb, 2023; Seers & Hogg, 2021; Stagg & Belcher, 2019).

### **Strengths, limitations, and future directions**

This study is among the first to apply advanced longitudinal methods such as LCGM to examine sex differences in trajectories of internalising problems in autistic and non-autistic children and adolescents within a large community-based sample. It sought to help fill a key gap in the literature by including a relatively large autistic sample and incorporating a non-autistic comparison group, enabling a contextualised investigation of sex-based developmental patterns.

Despite these strengths, several limitations should be acknowledged. First, the relatively small number of autistic females compared to autistic males and non-autistic participants may have reduced power to detect significant interaction effects between sex and autism diagnosis on both intercepts and slopes. Our autistic female group ( $n = 132$ ) barely reached the threshold for the recommended ideal minimum sample size of 150 participants for non-linear LGCMs with six time points (Diallo et al., 2014), suggesting that power limitations were likely.

We were also unable to explore the separate contributions of sex and gender identity. Although the MCS did collect gender identity data, it was measured at a single time point at a late stage in the study (age 17). Therefore, it is highly likely that attrition would significantly affect the available data, and the number of gender-diverse participants with complete data would likely be too small to support meaningful statistical analysis in growth models. Differentiating the effects of sex and gender could help clarify the respective biological and social influences on internalising problems. Therefore, this remains a crucial area for future research, particularly given the high rates of gender diversity among autistic individuals (Corbett et al., 2023; Warrier et al., 2020).

Furthermore, the MCS relied on passive case ascertainment for autism diagnosis (i.e. parent report of existing diagnoses). Active case ascertainment, such as administering autism screening questionnaires to all participants, could improve diagnostic accuracy and sample representation (Loomes et al., 2017). Relying solely on

parent report also introduces potential diagnostic uncertainty, increasing the risk of misclassification between autistic and non-autistic groups.

Additionally, research shows that camouflaging in autistic individuals is associated with reduced accuracy of observer-reported mental health symptoms – that is, external observers report those who camouflage more to experience fewer difficulties compared to self-reported levels of mental health symptoms (Corbett et al., 2021; Livingston et al., 2019). Therefore, relying solely on parent-reported measures of internalising problems in the current study may have underestimated the extent of difficulties experienced. Future research on mental health in autistic populations should incorporate self-report measures to improve accuracy.

Finally, whilst this study reflects the top research priority of the autism community, identified in a recent consultation exercise (Autistica, 2017), it was not co-produced with autistic individuals. As such, it may not fully reflect the priorities or lived experiences of the autistic community. Future work should adopt a co-production approach to ensure the research is meaningful, inclusive, and responsive to the needs of the autistic population.

Future research could build on these findings by examining both potential driving factors (e.g., insistence on sameness, peer victimisation, social difficulties, and interpersonal stress) that may directly contribute to the development of internalising problems, and potential moderating factors (e.g., social support, camouflaging behaviours, and gender norms) that may shape the strength or direction of these

associations. Such work could help clarify the mechanisms underlying sex differences in internalising problems among autistic youth

## **Conclusion**

The findings from this study suggest that sex differences in internalising trajectories among autistic youth may mirror those observed in the general population, underscoring the need for early identification and tailored mental health support. Future research should aim to disentangle the roles of sex and gender, include self-reported measures of mental health, and explore mechanisms that drive sex-specific risk for internalising difficulties in autism.

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**Table 1** Demographic characteristics of autistic and non-autistic participants in the whole sample. Sample sizes may vary depending on data availability for each characteristic.

	Autistic		Non-autistic	
	Males	Females	Males	Females
Total <i>n</i>	433	132	7,912	7,864
Received ADHD diagnosis <i>n</i> (%)	149 (34.4)	23 (17.4)	196 (2.3)	50 (0.6)
Ethnicity <i>n</i> (%)				
White	384 (88.7)	117 (88.6)	6,487 (82)	6,452 (82.0)
Minoritised ethnicity	46 (10.6)	13 (9.8)	1,376 (17.4)	1,367 (17.4)
Banded household income <i>n</i> (%)				
£ 0 - £16,500	163 (37.6)	44 (33.3)	2,386 (35.8)	2,447 (31.1)
£16,500.01 - £28,000	119 (27.5)	39 (29.5)	2,049 (25.9)	2,011 (25.6)
> £28,000	105 (24.2)	31 (23.5)	2,335 (29.5)	2,284 (29.0)
Parental highest educational qualification <i>n</i> (%)				
NVQ levels 1-2 (GCSE level)	184 (42.5)	49 (37.1)	2,947 (37.3)	2,962 (37.7)
NVQ levels 3, 4, 5 (A-level - higher education)	187 (43.2)	59 (44.7)	3,567 (45.1)	3,482 (44.3)
Delivery type <i>n</i> (%)				
Vaginal	288 (66.5)	86 (65.2)	5,262 (66.5)	5,450 (69.3)
Assisted or surgical (forceps, caesarean, etc)	139 (32.1)	44 (33)	2,588 (32.7)	2,369 (30.1)
Admitted to special baby unit at birth – answered yes <i>n</i> (%)	55 (12.7)	21 (15.9)	821 (10.4)	632 (8.0)
Maternal alcohol use during pregnancy – answered yes <i>n</i> (%)	153 (35.3)	38 (28.8)	2,587 (32.7)	2,493 (31.7)
Maternal smoking during pregnancy – answered yes <i>n</i> (%)	172 (39.7)	36 (27.2)	2,218 (28.0)	2,071 (26.3)
IQ score at age 7 years – mean (SD)	95.35 (15.6)	96.0 (16.5)	99.4 (15.4)	100.9 (14.4)
Gestational age (days) – mean (SD)	253.2 (17.1)	272.9 (18.1)	275.4 (14.3)	275.8 (13.8)
Birth weight (kg) – mean (SD)	3.36 (0.66)	3.22 (0.71)	3.40 (0.60)	3.28 (0.58)

**Table 2** Standardised coefficients from regression of intercept and slope factors on covariates in the whole sample latent growth curve model.

Predictor	$\beta_{\text{intercept}}$ (SE)	$\beta_{\text{slope}}$ (SE)	<sup>1</sup> Ethnicity coding: 1 = White, 0 =
Autism	0.750 (0.080) ***	0.368 (0.086) ***	
Female sex	- 0.056 (0.235) **	0.167 (0.009) ***	
Sex*Autism	-0.300 (.174)	0.079 (0.064)	
ADHD	0.143 (0.095)	0.203 (0.036) ***	
IQ (computed factor score)	-0.135 (0.024) ***	-0.032 (0.009) **	
Ethnicity - White <sup>1</sup>	0.454 (0.037) ***	-0.113 (0.012) ***	
Total banded family income - > £28,000 <sup>2</sup>	-0.292 (0.026) ***	-0.018 (0.010)	
Highest parent education – A-level/higher education <sup>3</sup>	-0.212 (0.027) ***	-0.001 (0.010)	
Baby in special baby unit	0.153 (0.046) **	-0.006 (0.018)	
Gestational age (days)	0.034 (0.025)	-0.021 (0.009) *	
Birthweight (kg)	-0.078 (0.023) ***	0.014 (0.008)	
Alcohol during pregnancy	0.013 (0.024)	-0.018 (0.009) *	
Smoking during pregnancy	0.081 (0.029) **	0.033 (0.011) **	
Delivery type - Vaginal birth <sup>4</sup>	-0.032 (0.025)	-0.004 (0.009)	

Minoritised ethnicity;

<sup>2</sup> Banded family income coding: 1 = > £28,000, 0 = £ 0 - £28,000;<sup>3</sup> Parent education level coding: 1 = NVQ levels 3, 4, 5 (A-level - higher education), 0 = NVQ levels 1-2 (GCSE level);<sup>4</sup> Delivery type coding: 1 = Vaginal, 0 = Surgical/assisted.

\* p &lt; 0.05. \*\* p &lt; 0.01. \*\*\* p &lt; 0.001.

**Table 3** Standardised growth parameter estimates for Step 3 latent growth curve models of internalising symptoms trajectories in autistic and non-autistic males and females.

Growth parameter	Autistic males	Autistic females	Non-autistic males	Non-autistic females
Intercept – Mean (SE)	3.038 (0.690) ***	2.023 (0.920) <sup>*</sup>	2.118 (0.139)***	2.158 (0.136)***
Intercept – Variance (SE)	2.043 (0.754) **	2.121 (1.002) <sup>*</sup>	1.012 (0.070)***	1.079 (0.068)***
Linear – Mean (SE)	-1.245 (1.244)	-0.114 (1.723)	0.160 (0.125)	-0.086 (0.123)
Linear – Variance (SE)	2.006 (1.316)	1.811 (1.688)	0.433 (0.049)***	0.454 (0.05)***
Quadratic – Mean (SE)	1.118 (0.651)	0.753 (0.897)	-0.033 (0.025)	0.084(0.026)**
Quadratic – Variance (SE)	0.424 (0.246)	0.177 (0.275)	0.015 (0.002)***	0.021 (0.002)***
Cubic – Mean (SE)	-0.162 (0.089)	-0.094 (0.122)	-	-
Cubic – Variance (SE)	0.008 (0.005)	0.003 (0.005)	-	-

\* p < 0.05. \*\* p < 0.01. \*\*\* p < 0.001

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**Table 4** Step 3 model-predicted differences in internalising symptom scores between autistic (A) and non-autistic (N) males (M) and females (F).

		3 years	5 years	7 years	11 years	14 years	17 years
Autistic males	Mean estimate	3.04	2.75	3.72	4.99	5.57	4.50
Autistic females	Mean estimate	2.02	2.57	4.05	5.91	7.57	8.47
Non-autistic males	Mean estimate	2.12	2.25	2.31	2.30	2.23	2.10
Non-autistic females	Mean estimate	2.16	2.16	2.32	2.66	3.17	3.84
Autistic males vs autistic females	Mean difference [95% CI]	1.02 [-1.64, 3.66] p = 0.452	0.18 [-2.83, 3.19] p = 0.905	-0.33 [-3.40, 2.74] p = 0.835	-0.92 [-4.23, 2.39] p = 0.586	-2.00 [-5.55, 1.55] p = 0.270	-3.97 [-8.47, .53] p = 0.083
df=563		M = F	M = F	M = F	M = F	M = F	M = F
Non-autistic males vs non-autistic females	Mean difference [95% CI]	-0.04 [-0.42, 0.34] p = 0.838	-0.09 [-0.24, 0.41] p = 0.591	-0.02 [-0.39, 0.36] p = 0.929	-0.36 [-0.76, 0.04] p = 0.078	-0.93 [-1.38, -0.49] p < 0.001	-1.75 [-2.37, -1.12] p < 0.001
df= 15774		M = F	M = F	M = F	M = F	M < F	M < F
Autistic vs non-autistic females	Mean difference [95% CI]	-0.14 [-2.23, 1.96] p = 0.899	0.41 [-1.33, 2.16] p = 0.644	1.73 [-0.29, 3.73] p = 0.092	3.25 [1.05, 5.44] p = 0.004	4.40 [1.81, 6.99] p < 0.001	4.63 [0.77, 8.49] p = 0.018
df= 7994		A = N	A = N	A = N	A > N	A > N	A > N
Autistic vs non-autistic males	Mean difference [95% CI]	0.92 [-0.29, 2.13] p = 0.135	0.50 [-0.57, 1.58] p = 0.356	1.14 [0.19, 2.64] p = 0.023	2.69 [1.41, 3.96] p < 0.001	3.34 [2.03, 4.65] p < 0.001	2.40 [0.67, 4.14] p = 0.007
df = 8343		A = N	A = N	A > N	A > N	A > N	A > N

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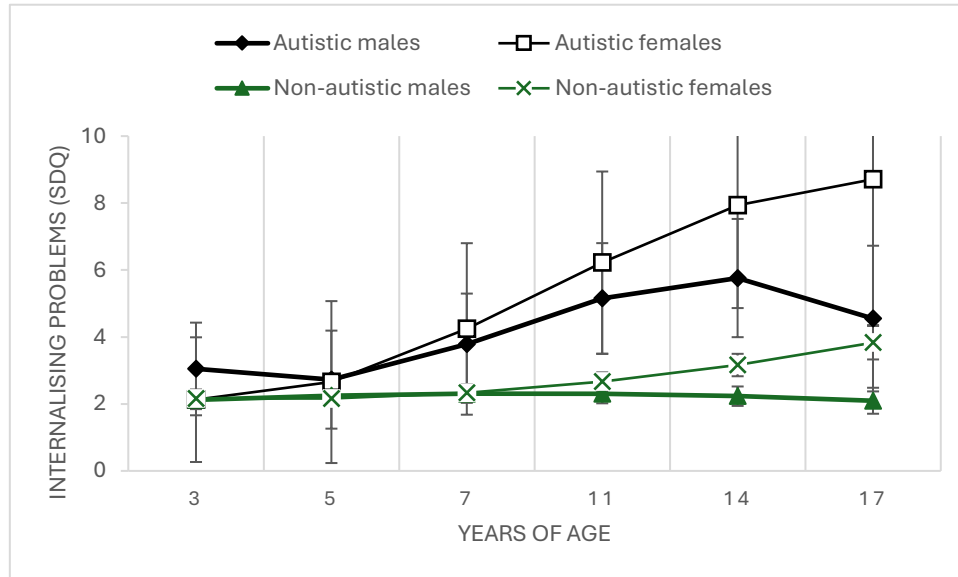
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**Figure 1** Mean trajectories of internalising symptoms for autistic males and females and non-autistic males and females estimated from Step 3 latent growth curve models. Error bars represent 95% confidence intervals.



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## Highlights

- Autistic (n=573) and non-autistic (n=15,945) participants from a UK cohort study.
- Latent Growth Curve Modelling used to track internalising trajectories.
- Autism linked to higher and steeper increases in internalising symptoms.
- Females showed steeper increases despite lower baseline symptoms.
- Autistic vs non-autistic differences emerged earlier in males than females.

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