Relationships between parental mental health, peer victimisation and internalising symptoms in children with autism

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

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OVERVIEW

This thesis focuses on improving our understanding of the development of internalising symptoms, such as anxiety and depression, for autistic children and adolescents.

Part 1 is a systematic review of the literature examining potential environmental risk factors for internalising symptoms, within samples of autistic adolescents and young people. The systematic review identified 29 studies, all of which were included in a narrative synthesis. Of these, 23 were also included in six meta-analyses. Findings indicated that eight themes of potentially-modifiable environmental factors showed meaningful associations with internalising symptoms experienced by autistic young people: parental mental health or stress; peer victimisation; parenting behaviour or family interaction; socioeconomic status; negative life events; social interaction; social support and pet ownership.

Part 2 describes the empirical paper, which uses secondary data from the Millennium Cohort Study to examine the longitudinal and bi-directional relationships between two hypothesised risk factors (parental mental health, and peer victimisation) with child internalising symptoms. Data was available for 560 young people with a diagnosis of Autism Spectrum Disorder, across 6 timepoints from birth to 17-years-old. Random-Intercept Cross-Lagged Panel Models identified consistent cross-sectional associations between risk factors and child internalising symptoms at each timepoint, and several cross-lagged associations between risk factors and child internalising symptoms, including a bi-directional effect.

Part 3 is a critical appraisal of the research, and focuses on three themes: the implications of being a non-autistic researcher conducting research about autism spectrum disorder; the opportunities and limitations of using secondary data from longitudinal cohort studies; and the utility and interpretation of Random-Intercept Cross-Lagged Panel Models.

IMPACT STATEMENT

The findings from this research have implications for both clinical practice and academic research within the field of mental health difficulties experienced by autistic children and young people.

Part one of this thesis systematically reviews the literature to identify potential environmental risk factors for internalising symptoms in autistic adolescents and young people. Environmental risk factors were chosen as they may be more directly amenable to prevention and intervention (compared with individual risk factors). This focus on the environmental context also aligns with the neurodiversity conceptualisation of autism, prioritising improvements to the person-environment fit for autistic people. The quality of evidence in support of each category of environmental risk was reviewed, and environmental risk factors with consistent associations with adolescent internalising symptoms were identified. Clinical implications include the prioritisation of identified environmental risk factors for prevention or intervention, in both public health policy, and routine clinical settings. Additionally, implications for academic research include further study to understand these risk factors in more detail, extending this systematic review to earlier childhood or adulthood, or making comparisons with typically-developing samples to understand whether these environmental risk factors have a general association with internalising symptoms, or have an autism-specific effect.

Part two of the thesis uses longitudinal cohort data to measure the associations between internalising symptoms and two hypothesised risk factors between birth and 17-years-old in a sample of autistic children. Random-Intercept Cross-Lagged Panel Models were used to measure longitudinal associations between time-points, and bi-directional associations across time points. Clinical implications include suggestions for the prevention of child internalising symptoms, by acting on the environmental risk factors of parental mental health and peer victimisation in early childhood. These early predictive associations may inform public health policy, as well as clinical practice with young autistic children and their families. Clinicians working with autistic children and young people may consider screening for parental mental health difficulties or peer victimisation at

assessment and throughout treatment, so that preventative intervention may be offered. Additionally, a later bidirectional association was found, with youth internalising symptoms predicting parental mental health difficulties. Clinicians could consider this when working with autistic adolescents experiencing internalising symptoms, so that parents may be offered access to mental health support where indicated. Implications for academia include replication of these effects in other countries and cultures, given that this data is based on a British birth cohort. Further study will be important to understand whether internalising symptoms (known to be more prevalent in autistic children than the general population) occur with greater sensitivity in response to similar risk factors as for typically-developing children (as suggested by this study), or whether further autism-specific risk factors may explain the increased prevalence.

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Part 1: Literature Review

A systematic review of environmental factors associated with internalising symptoms in adolescents and young people with autism

ABSTRACT

Background: Internalising symptoms such as anxiety and depression are common in young people with autism. However, it is not yet fully understood how environmental factors may contribute to the development of internalising symptoms in this population, or act as protective factors. This systematic review focuses on environmental factors (existing outside the young person with autism) as opposed to individual factors, in order to emphasise the influence of the environmental context, rather than locating causes of mental health difficulties within the individual.

Methods: In this systematic review, we searched EMBASE, MEDLINE, PsycINFO, Web of Science and ASSIA databases for studies investigating environmental factors in relation to internalising symptoms experienced by autistic young people. A narrative synthesis of studies was conducted by theme of environmental factor, and environmental factors with sufficient studies were further analysed by meta-analysis. This systematic review was pre-registered with PROSPERO (CRD42021243913).

Findings: Of 3049 unique studies identified, 116 were selected for full-text review, of which 29 were eligible for inclusion in narrative synthesis, and 23 studies were included in six meta-analyses. Environmental factors identified in the studies were grouped into 11 themes, with findings indicating that eight themes showed a meaningful association with internalising symptoms experienced by autistic young people: parental mental health or stress; peer victimisation; parenting behaviour or family interaction; socioeconomic status; negative life events; social interaction; social support and pet ownership. Meta-analyses were conducted for five environmental factors: peer victimisation (mean effect size r = 0.19 (95%-CI 0.00 – 0.36) for parent-reported internalising symptoms; mean effect size r = 0.37 (95%-CI 0.12 – 0.57) for young person reported internalising symptoms), parental mental health or stress (mean effect size r = 0.44 (95%-CI 0.33 – 0.54)), parenting behaviour (mean effect size r = -0.13 (95%-CI -0.36-0.10)), socioeconomic status (mean effect size r = 0.26 (95%-CI 0.12-0.39), and negative life events (mean effect size r = 0.23 (95%-CI 0.09 – 0.36)).

Interpretation: Several potentially-modifiable environmental factors were consistently associated with worsened internalising symptoms for autistic young people: parental mental health or stress; peer victimisation; low socioeconomic status and negative life events. Environmental factors suggesting a potentially protective association with internalising symptoms were also identified: social interaction; social support and pet ownership. Clinical and research implications of the findings are discussed.

INTRODUCTION

Autism Spectrum Disorder is a diagnosis used to describe individuals displaying a characteristic set of difficulties in social communication, sensory sensitivity, restricted and repetitive behaviours and highly restricted interests (American Psychiatric Association, 2013). These characteristic difficulties begin early in life, but may not be recognised or diagnosed until mid- to late-childhood, or even beyond. Autism Spectrum Disorder is a life-long neurodevelopmental condition, and has an estimated prevalence of 1.1% in the UK (NHS, 2012).

This review concerns the mental health of young people diagnosed with Autism Spectrum Disorder. We are aware that a number of terms have been used to refer to and communicate about autism spectrum disorder in community, clinical and research settings, for example 'people with autism', 'ASD', 'autistic', or 'on the autism spectrum'. Within this review, we will use the terms 'people [or children, or young people] with autism' as well as 'autistic'. This is in order to remove the emphasis on autism as a 'disorder', and balance the use of 'person-first' (person with autism) and 'identity-first' language (autistic person). These frames of reference were most highly endorsed by a 2015 study of autistic people, parents, families and friends, and professionals working with autistic people (Kenny et al., 2016). This is in line with guidance from Autism Europe (2017), and The National Autistic Society UK (2015). For the purpose of this review, the words 'autism' or 'autistic' will be used to refer to the clinical or research diagnosis of autism spectrum disorder, or associated historic diagnoses such as Asperger's syndrome and Pervasive Developmental Disorder.

There is high prevalence of mental health disorders in autistic young people – with estimates of 70% of 10-14 year olds meeting criteria for at least one mental health disorder, and 41% meeting criteria for two or more (Simonoff et al., 2008). Studies of children with autism have shown that co-occurring psychological difficulties are associated with increased impairment (including social and academic impairment) and reduced quality of life for both the child and their families (Kaat, Gadow & Lecavalier, 2013; Posserud et al., 2018).

A key distinction in the field of children's mental health is between 'externalising' and 'internalising' disorders or symptoms (Achenbach, 1978), where externalising refers to hyperactivity, aggression and anti-social behaviour, and internalising refers to withdrawn, anxious, inhibited, and depressed behaviours. Internalising symptoms (IS) are known to be very common in autistic children, and prevalence studies have found them to manifest in common diagnosable difficulties such as social anxiety disorder (Simonoff et al., 2008), generalised anxiety disorder and phobias (Salazar et al., 2015). Research has also shown that difficulties with affect and anxiety in childhood often persist into early adulthood for people with autism (Simonoff et al., 2013; Gotham et al., 2015), and are associated with lower life satisfaction and emotional regulation (Gotham et al., 2015). Internalising symptoms are also a concern for autistic people themselves, as put forward in a 2016 priority-setting initiative (Autistica & James Lind Alliance, 2016). Out of the top ten priorities for autism research, both the first-ranked priority ("Which interventions improve mental health or reduce mental health problems in people with autism?") and the fourth-ranked priority ("Which interventions reduce anxiety in autistic people?") concern internalising symptoms in the form of mental health problems such as anxiety, depression and obsessive-compulsive disorder. For the above reasons, the present study is focused on internalising symptoms experienced by autistic children.

A risk factor can be defined as "any factor or situation that increases a [person's] chance of developing negative health or behavioural outcomes" (Grizenko & Fisher, 1992). In this systematic review, environmental risk factors will be therefore be considered as any factor external to the young person which may increase their chance of developing negative health or behavioural outcomes. Examples of environmental factors external to the young person may include for example, parental mental health, poverty, or exposure to negative life events. This is opposed to individual risk factors, such as general intelligence, or expression of autistic traits. The identification of environmental risk factors has clinical utility in that these factors are potentially modifiable, and increase the focus on the impact of the environmental and social context that autistic people are

living within, as opposed to locating the risk factors for difficulties within the person (Pellicano & den Houting, 2021).

Environmental factors have been proposed to contribute to the co-occurrence of IS in autistic children and young people; with a variety of risk factors, including social and familial factors, being identified (Midouhas et al., 2013; Storch et al., 2012; Scherff et al., 2013; Maljaars et al., 2014). Considerable evidence has emerged for the impact of parental mental health difficulties on the development of child IS, both in children with autism, as well as neuro-typical (NT) children (Simonoff et al., 2013, Bayer et al., 2011; Fitzimons et al., 2017; Bøe et al., 2014). An additional risk factor proposed by the literature to contribute to child IS is peer victimisation (or bullying). Whilst this has been observed in NT samples (Reijntjes et al., 2010) this may be particularly important for autistic young people, who are at a particularly high risk of peer victimisation due to the nature of their social communication difficulties (Adams et al., 2014; Bishop et al., 2007; Schroeder et al., 2014).

Given the background of existing research, this review aims to identify and understand which environmental factors (that is – factors external to the young person) are associated with IS in autistic young people. We have chosen to focus this review on 'young people' as opposed to younger children, in order to encompass the developmental stage of adolescence, and given that this age range is comparatively under-studied for autistic people.[1] Focusing on this older age group may also allow the inclusion of more female participants, who are more likely to receive an autism diagnosis at a later age (Rutherford et al., 2016), and therefore may be disproportionally missed in studies of autism in children. For this review we used the definition of 'young people' provided by the World Health Organisation, spanning from 10-24 years old (WHO, n.d.*).*

[1] *A search on the Web of Science database returns 27,103 results for the search terms 'autism' AND 'child*', compared with 4,690 results for the terms 'autism' AND ('adolescen*' OR 'young person' OR 'young people')).*

This systematic review therefore aims to answer the following questions:

1. What environmental factors are associated with internalising symptoms in autistic young people?

2. What is the strength of association between these environmental factors and internalising symptoms in autistic young people?

METHOD

Search Strategy

The systematic review was registered on Prospero before searches were conducted (PROSPERO 2021 CRD42021243913).

A systematic search of the literature was conducted by searching EMBASE, MEDLINE, PsycINFO, Web of Science and ASSIA databases. Search terms focused on four areas: age range (young people), autism, internalising symptoms, and environmental factors (see Table 1).

Table 1

Age range	**Autism**	**Internalising symptoms**	**Environmental factors**
adolescen*	Autism	depress*	explain*
teen*	Autistic	anxi*	explanatory
young adult*	ASD	phobi*	predict*
youth*	Asperger*	internali*	associat*
young person	Pervasive development*	emotion*	risk
young people	disorder*	somatic	factor*
	PDD	psychosomatic	
		psychopathology	
		psychiatric	

Literature review search terms

The search terms in Table 1 were combined so that the terms within each area were separated by 'OR' functions, and columns separated by 'AND' functions, so that only articles which contained at least one term in all four areas were retrieved.

No date limits were set for the search, which was performed on the 14th June 2021.

The retrieved articles were reviewed against the inclusion and exclusion criteria set out below. To determine which articles met inclusion criteria, titles and abstracts were reviewed initially. Those of potential relevance were then reviewed in full. This is described in detail in section 3, alongside a PRISMA diagram.

Inclusion and Exclusion criteria

Inclusion and exclusion criteria were set for type of study, participants within sample, and type of exposure. These are laid out in Table 2 below.

Table 2

Type of Study	*Inclusion*	1. Studies that have been peer-reviewed 2. Cross-sectional or longitudinal studies 3. Studies investigating an intervention (only baseline data will be extracted) 4. Studies using a validated measure of adolescent or young person internalising symptoms
	Exclusion	1. Review articles, conference presentations, or discussion papers. 2. Studies using exclusively qualitative data or analyses 3. Retrospective studies 4. Studies investigating only parent or sibling internalising symptoms 5. Studies investigating adolescent / young person externalising symptoms only
Participants	*Inclusion*	1. 10-24 year olds, with a diagnosis of autism spectrum disorder, diagnosed using recognized diagnostic criteria at the time of the publication.
	Exclusion	1. Studies in which samples have a mean age of 25 years or older 2. Studies in which samples have a mean age of 9 years or younger 3. Studies in which samples include participants without a clinical or research diagnosis of autism spectrum disorder.
Exposure	*Inclusion*	1. Any environmental factor that is being investigated for potential association with internalising symptoms.
	Exclusion	1. Environmental factors where the exposure was prior to birth (such as in utero) 2. Factors internal to the young person (such as personality traits, IQ, autism spectrum disorder presentation). This study is not investigating the effect of interventions. If we find studies that include an intervention for environmental factors or internalising symptoms, we will only include data from the baseline assessment.

Inclusion and Exclusion criteria for study selection following searches

Quality Assessment

The methodological quality of all included studies was assessed by means of a critical appraisal checklist designed for the evaluation of cross-sectional studies. The chosen critical appraisal checklist was the JBI Critical Appraisal Checklist for Analytical Cross-Sectional Studies (Moola et al., 2020), given its ease of use and interpretation, and that it has good utility for observational studies without a control group (as was the case for most studies identified through this systematic review). Prior research has compared the JBI tool with the Risk Of Bias In Non-randomised Studies-of Interventions (ROBINS-I)) tool, and found them comparable in assessing risk of bias (Glasgow et al., 2020). Whilst a JBI cohort (longitudinal) critical appraisal checklist is also available, the cross-sectional appraisal tool was used for all included studies given that where the study was longitudinal, data for only one timepoint was extracted, and where the study was an RCT, only baseline data were extracted. It therefore felt most appropriate to evaluate the cross-sectional element of each study for the purposes of the quality appraisal.

Each study was assessed against this checklist, which evaluates 8 domains; inclusion criteria, description of subjects and settings, measure of exposure, measure of condition, identification of confounding factors, appropriate techniques to deal with confounding factors, outcome measures, and statistical analysis. See Appendix 1 for a copy of the full checklist. For the purposes of this systematic review, autism was considered to be the 'condition', measure of IS was considered to be the 'outcome measure' and measure of environmental factor was considered to be the 'exposure'.

Each study was evaluated in each of the 8 domains, and marked as either high quality in this domain ('Yes'), low quality ('No'), or that it was not possible to evaluate quality in this domain from the study information ('Unclear'), see Table 4.

It should be noted that in the case of the exposure (measure of environmental factor), the JBI checklist would rate any non-validated measure of environmental factor as low quality, however reporting a non-validated measure of environmental factor was not an exclusion criterion for this

review, in order to capture a pragmatic, clinically-relevant range of environmental influences. For this reason, a number of studies are marked as 'low quality' or 'missing information' in this domain but are suitable for inclusion in the review.

Data Extraction

Data was extracted from each included study using a Microsoft Excel-based extraction template, which included study design, sample size, country, mean age of sample, participant characteristics, method of ASD diagnosis, measure of IS, type and measure of environmental factors, statistical analyses and results.

Where studies reported more than one measure of exposure, or measure of IS that met our inclusion criteria, all were extracted. All relevant statistical analyses and results were extracted. In the case of treatment studies (including RCTs), only analyses based on the baseline data were extracted. In the case of longitudinal studies, all data where participants were within the age range inclusion criteria were extracted.

Data Synthesis

The identified studies were grouped into environmental factor 'themes' based on shared environmental factor characteristics. All studies were considered for inclusion in meta-analyses where possible, and all studies were included in narrative synthesis by environmental factor theme.

Inclusion in the meta-analysis

Studies were included in the meta-analyses if: 1) there were at least two studies in a given theme 2) the study measured sufficiently similar variables to the other studies to provide a fair comparison, and 3) the study reported an effect size measure that could be converted to *r*. The correlation coefficient *r* was used as this was the most commonly-reported effect size statistic.

In some cases, studies eligible for meta-analysis reported the results of more than one relevant analysis. In deciding which effect size to extract, we chose where possible to match on the following

characteristics across studies within one meta-analysis: 1) by reporter-identity (such as parent, or self-report), 2) by type of internalising measure where there was a choice (such as choosing to use the effect size for anxiety rather than depression measures, where other studies more commonly report anxiety measures), 3) by measurement of environmental factor (where there were measurement differences).

Whilst most studies featuring in the meta-analyses were cross-sectional, a small number of longitudinal studies were included. Where these studies reported analyses for several timepoints within their sample, we chose the earliest timepoint in which the study participants fall within the review inclusion criteria of between 9- and 25-years-old.

Meta-analytic procedures

Meta-analyses were carried out in R, using the 'meta' package and 'metacor' function (Balduzzi et al., 2019). Fisher's Z-transformations were completed within the metacor function. A random effects model was used because we expected considerable heterogeneity across studies. Q and I^2 statistics were calculated as indicators of heterogeneity, where an I^2 value of 0% indicates no observed heterogeneity and larger values indicate greater heterogeneity (25%=low, 50%=moderate, 75%=high).

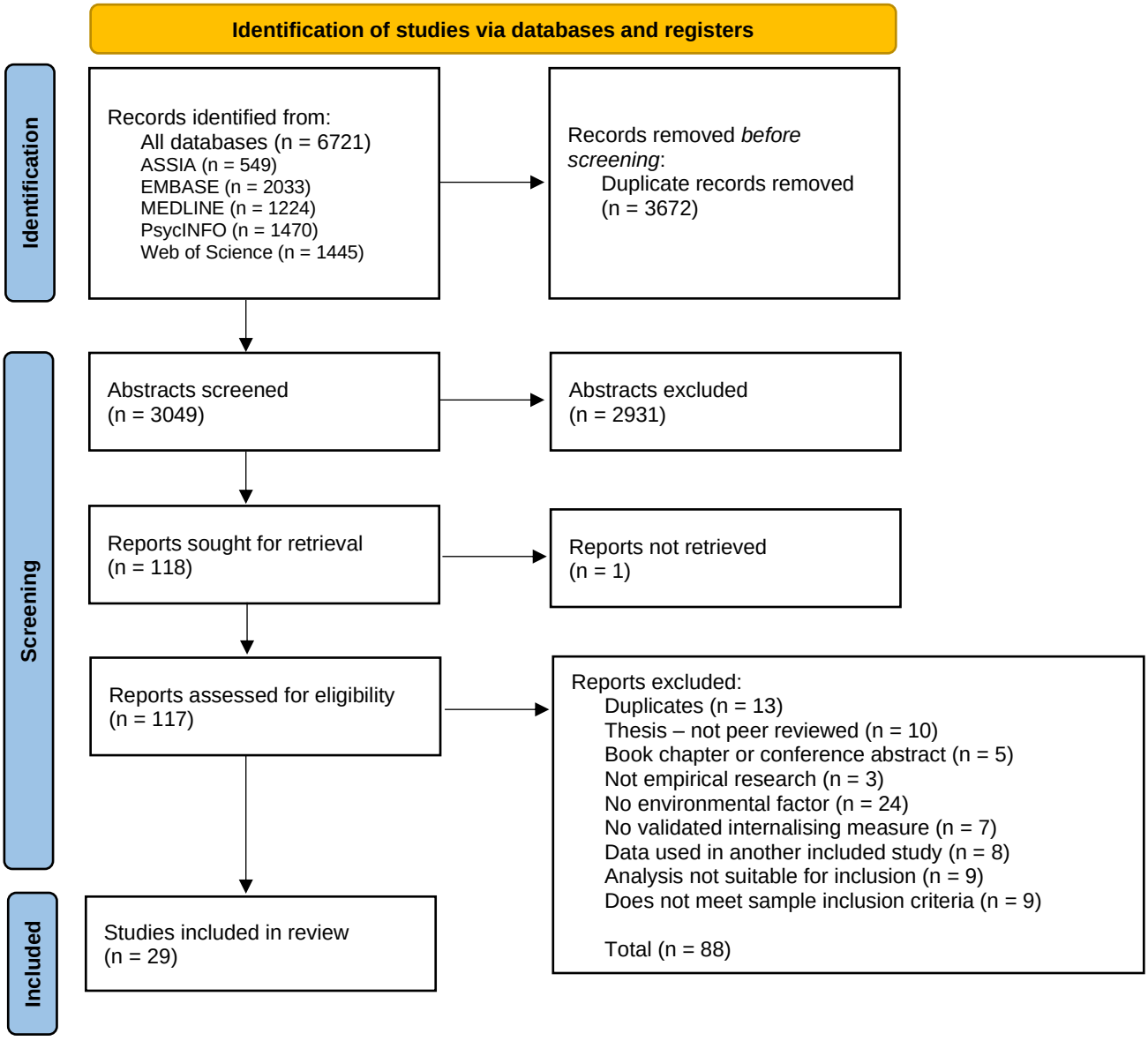
Narrative Synthesis

In addition to being considered for meta-analysis, all studies were synthesised by narrative synthesis within their environmental factor theme. This included a primary synthesis of the findings (tabulated sample characteristics, research design and results), exploring relationships in the data both within and between studies, and considering the impact of study design, sample, and measurement tools in explaining differences within and between studies.

RESULTS

The database searches identified a total of 6721 articles. 3672 duplicates were identified, leaving 3049 unique articles for title and abstract review. 2931 abstracts did not meet the inclusion criteria, leaving 118 abstracts for further investigation. Full-text articles were sought for these studies (not available for 1 study), and were read through for further evaluation. Of these 117 studies, 29 studies met the full inclusion criteria and were included for this review (Figure 1).

Figure 1



PRISMA diagram

Study characteristics

Summaries of the included studies are provided in Table 3. The studies were conducted in the following countries: USA (n = 15), Canada (n = 3), Netherlands (n = 3), Spain (n = 2), Australia, UK , Belgium, Taiwan, Italy, and Singapore (all n = 1). Study sample sizes ranged from 17 to 1202, with a total of 3,999 autistic young people represented in this review. Most studies were cross-sectional (21), with an additional three RCTs (baseline data only), and five longitudinal (cohort) studies.

Participant characteristics

As per the inclusion criteria, all participants had a research or clinical diagnosis of autism. There were no exclusion criteria for comorbidity, therefore participants in some studies had additional diagnoses such as intellectual disability or attention deficit hyperactivity disorder (ADHD). Whilst most studies did not recruit participants specifically based on their anxiety levels, three studies (Storch et al., 2012; Frank et al., 2020; Reaven et al., 2015), included only participants with autism and a clinical level of anxiety. Participant samples ranged in mean age from 9.1 to 19.9 years old. Percentage of male adolescents in study samples ranged from 62% to 100%.

Environmental factor characteristics

The environmental factors investigated by each study are shown in Table 3. Whilst each study measured and reported their own selection of environmental factors, resulting in a very heterogeneous set, we allocated the reported environmental factors into a set of environmental factor theme clusters derived from identified studies, in order to aid analysis. These consisted of peer victimisation (n = 11), parental stress or MH (n = 6), parenting behaviour or family interaction (n = 6), socioeconomic status (n = 4), negative life events (n = 3), social interaction (n = 2), and social support, Covid-19 pandemic, pet ownership, school placement, and parental education (all n = 1). A number of studies investigated the impact of one or more environmental factors, so these do not sum to 29. Table 4 shows which studies are represented within each environmental factor theme.

Risk of bias within studies

All included studies were evaluated using the JBI Quality Appraisal checklist tool. 12 studies were evaluated as high quality across all eight domains, 6 studies included one or more domains in which quality was evaluated as low, and the remaining 11 studies included one or more domains in which quality appraisal information was missing (Table 5). Although the overall quality of included studies was good, methodological issues in several domains were more common: 1) fully describing inclusion or exclusion criteria for their sample of autistic young people or caregivers (n = 8), 2) identification of potentially confounding factors (n = 7), or 3) techniques to deal with confounding factors (n = 8).

Table 3

	Study type	Country	Sample size	Percentage male	Mean age	Measure of internalising symptoms	Environmental factor
Adams et al., 2014	Cross-sectional	USA	54	100%	14.6	Youth Self Report, Children's Depression Inventory & Child Behaviour Checklist	Peer Victimisation
Dallman et al., 2021	Cross-sectional	USA	17	100%	14.0	Child Depression Inventory 2nd Ed.	Level & Quality of Social interaction
Dieleman et al., 2017	Cohort	Belgium	139	83%	10.2 (Timepoint 1 used in review)	Internalising sub-scale of Child Behaviour Checklist	Parenting behaviour
Fink et al., 2018	Cross-sectional	Netherlands	120	91%	15.6	Emotional problem sub-scale of Strengths & Difficulties Quest.	Bullying behaviour Peer victimisation
Frank et al., 2020	RCT (baseline)	USA	168	80%	9.9	Pediatric Anxiety Rating Scale	Youth accommodation (by parents)
Fung & Weiss, 2015	Cross-sectional	Canada	91	82%	13.4	Glasgow Depression Scale – Carer supplement	Negative life events Parental stress
Gray et al., 2012	Cohort	Australia	119	82.4 – 80.9% (dependent on timepoint)	12.8 (Timepoint 2 used in review)	Anxiety sub-scale of Developmental Behaviour Checklist	Socioeconomic status
Greenberg et al., 2008	Cohort	USA	149	76%	19.9	Problem behaviour sub-scale of Scales of Independent Behaviour revised	Maternal expressed emotion Quality of Mother-Child relationship
Greenlee & Johnson, 2020	Cross-sectional	USA	176	73%	13-17 (No mean provided)	Revised Child Anxiety and Depression scale	Family functioning Peer victimisation
Kerns et al., 2015	Cross-sectional	USA	59	78%	10.6	Anxiety Disorder Interview Schedule	Parental Stress
Liu et al., 2021	Cross-sectional	Taiwan	219	88%	13.7	Social Anxiety sub-scale of Taiwanese ver. Multidimensional Anxiety Scale for children	Bullying behaviour Peer victimisation
Lugo-Marin et al., 2021	Cohort	Spain	37	87%	10.7	Internalising sub-scale of Child Behaviour Checklist	Covid-19 lockdown
Maljaars et al., 2014	Cross-sectional	Netherlands & Belgium	536	83%	11.7	Internalising sub-scale of Strengths & Difficulties questionnaire	Parenting behaviour
Mazurek et al., 2010	Cross-sectional	North America	1202	86%	9.1	Child Behaviour Checklist	Dyadic friendship
Pouw et al., 2013	Cross-sectional	Netherlands	63	100%	11.7	Child Depression Inventory	Bullying behaviour Peer victimisation
Reaven, et al., 2015	RCT (baseline)	USA	31	Primary age cohort: 100%	13.8	Screen for Child Anxiety and Related Emotional Disorders	Parental anxiety

				Adolescent cohort: 62%			
Rosa et al., 2016	Cross-sectional	Spain	50	92%	12.0	Schedule for Affective Disorders & Schizophrenia school-aged children	SES
Scibelli et al., 2021	Cross-sectional	Italy	101	84%	12.7	Anxiety, Depression and Somatic sub-scales of Child Behaviour Checklist	Parent Stress
Simonoff et al., 2013	Cohort	UK	81	93%	12.0 (Timepoint 1 used in review)	Emotional problem sub-scale of Strengths & Difficulties questionnaire	Parent SES Parental education Maternal mental health Parenting stress
Storch et al., 2012	Cross-sectional	USA	60	80%	12.2	Child Behaviour Checklist Revised Children's Anxiety and Depression scale	Peer victimisation
Taylor & Gotham, 2016	Cross-sectional	USA	36	83%	18.7	Schedule for Affective Disorders & Schizophrenia school-aged children	Negative Life Events
Ting & Weiss, 2017	RCT (baseline)	Canada	51	88%	10.0	Internalising sub-scale of Behaviour Assessment System for children	Parent co-regulation Parent scaffolding
Ung et al., 2016	Cross-sectional	USA	81	77%	11.9	Revised Child Anxiety and Depression scale	Peer victimisation
Van Gerrit et al., 2018	Cross-sectional	USA	35	65%	16.4	Multidimensional Anxiety Scale for children	Bullying behaviour Peer victimisation
Ward et al., 2017	Cross-sectional	USA	73	88%	13.9	Youth self-report Depression Scale Depression sub-scale of Child Behaviour Checklist	Pet Ownership
Weiss et al., 2015	Cross-sectional	Canada	101	75%	14.5	Insecure-Anxious sub-scale of Nisonger Child Behaviour Rating form	Parent Stress Negative Life Events Peer victimisation Household income
Wright, 2017	Cross-sectional	USA	128	89%	11 – 16 (no mean provided)	Centre for Epidemiological Studies Depression scale Multidimensional Anxiety Scale for children	Peer victimisation Parent mediation of technology use
Zainal, 2019	Cross-sectional	Singapore	96	Mainstream cohort = 81% Special school = 88%	Mainstream cohort = 10.9 Special school = 10.9	Spence Children's Anxiety scale Developmental Behaviour Checklist	School environment Learning / Behavioural support
Zeedyk et al., 2014	Cross-sectional	USA	44	89%	13.0	Internalising sub-scale of Child Behaviour Checklist	Peer victimisation Bullying behaviour

Summary of included studies

Table 4

Peer Victimisation		Parental Mental Health & Stress		Parenting & Family Behaviour		Socioeconomic Status (SES)	
Adams et al.	2014	Fung & Weiss	2015	Dieleman et al.	2017	Gray et al.	2012
Fink et al.	2018	Kerns et al.	2015	Frank et al.	2020	Rosa et al.	2016
Van Gerrit et al.	2018	Reaven et al.	2015	Greenberg et al.	2008	Simonoff et al.	2013
Greenlee & Johnson	2020	Scibelli et al.	2021	Greenlee & Johnson	2020	Weiss et al.	2015
Liu et al.	2021	Simonoff et al.	2013	Maljaars et al.	2014	***N = 4***	
Pouw et al.	2013	Weiss et al.	2015	Ting & Weiss	2017		
Storch et al.	2012	***N = 6***		***N = 6***			
Ung et al.	2016						
Weiss et al.	2015	**School Placement**		**Social Interaction**		**Social Support**	
Wright	2017	Zainal et al.	2019	Dallman & Harrop	2021	Ung et al.	2016
Zeedyk	2014	Simonoff et al.	2013	Mazurek	2010	***N = 1***	
N = 11		***N = 2***		***N = 2***			
Covid-19 Pandemic		**Negative Life Events**		**Pet Ownership**		**Parent Education**	
Lugo-Marin et al.	2021	Fung & Weiss	2015	Ward et al.	2017	Simonoff	2013
N = 1		Taylor	2016	***N = 1***		***N = 1***	
		Weiss et al.	2015				
		N = 3					

Environmental Factors by Theme

Table 5

	Defined inclusion criteria	Subjects & Setting described	Valid & Reliable exposure measure	Objective, standard measure of condition	Confounding factors identified	Strategies to deal with confounding factors	Valid, Reliable outcome measure	Appropriate statistical analysis
Gray et al., 2012	🟡	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Adams et al., 2014	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Dallman et al., 2021	🟢	🟢	🟡	🟢	🟡	🟡	🟢	🟢
Dieleman et al., 2017	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Fink et al., 2018	🟡	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Frank et al., 2020	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Fung & Weiss, 2015	🟡	🟢	🟢	🟡	🟢	🟢	🟢	🟢
Van Schalkwyk et al., 2018	🔴	🟢	🟢	🟢	🟢	🟡	🟢	🟢
Greenberg et al., 2008	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Greenlee et al., 2020	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Kerns et al., 2015	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Liu et al., 2021	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢
Lugo-Marin et al., 2021	🟢	🟢	🟡	🟢	🔴	🔴	🟢	🟢
Maljaars et al., 2014	🟢	🟢	🟢	🟢	🟢	🟢	🟢	🟢

Mazurek et al., 2010								
Pouw et al., 2013								
Reaven, et al., 2015								
Rosa et al., 2016								
Scibelli et al., 2021								
Simonoff et al., 2013								
Storch et al., 2012								
Taylor & Gotham, 2016								
Ting & Weiss, 2017								
Ung et al., 2016								
Ward et al., 2017								
Weiss et al., 2015								
Wright, 2017								
Zainal, 2019								
Zeedyk et al., 2014								

Quality Appraisal summary by study. Green indicates evaluation as high quality ('Yes'), Red indicates evaluation as low quality ('No'), and Yellow indicates that the quality appraisal was unclear from the study information available ('Unclear').

Synthesis of results by environmental factor

A summary of the 11 environmental factor themes can be found in Table 4 above. These are synthesised in the sections below, by meta-analysis (where possible) and narrative synthesis.

1. Parent mental health or stress

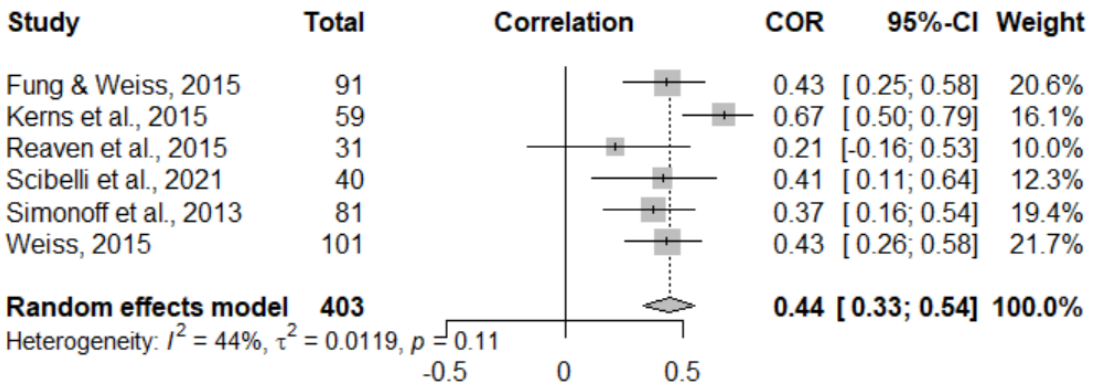
The relationship between parent MH and young person IS was investigated by six studies. Two of these studies were rated as having at least some concerns or unclear information in their quality assessments (Fung & Weiss, 2015; Reaven et al., 2015). Out of these six studies, all were suitable for meta-analysis.

In all cases parental MH or stress was reported by parents themselves; mothers only, or either parent. In this subset of studies, none measured exclusively self-reported adolescent IS, with studies measuring; parent-reported (n = 4), parent- and teacher-reported (n = 1) or joint parent & self-reported autistic adolescent IS (n = 1).

The studies report on broad conceptualisations of adolescent IS, including anxiety (Kerns et al., 2015; Reaven et al., 2015; Scibelli et al.; Weiss et al., 2015), depression (Fung & Weiss, 2015; Scibelli et al., 2021), somatising symptoms (Scibelli et al., 2021) or a broad internalising factor (Simonoff et al., 2013).

In the case of one study (Scibelli et al., 2021) the results reported adolescent IS separately as measures of anxiety or depression. The anxiety measure was chosen for inclusion in the meta-analysis to improve similarity with the other included studies, however a sensitivity analysis was conducted with the depression measure in place of the anxiety measure (see Appendix 3).

Figure 2



Meta-analysis of the associations between autistic adolescent internalising symptoms, and parental MH or stress

Six cross-sectional associations relating parental MH or stress with greater levels of adolescent IS yielded a combined $p < 0.0001$, a moderate mean effect size ($r = 0.44$) and moderate heterogeneity ($I^2 = 44\%$), see Figure 2. This indicates a highly significant moderately-sized effect, where worsened parental MH or stress is associated with increased internalising symptoms in autistic adolescents.

The sensitivity analysis conducted with Scibelli et al.'s depression measure, yielded a combined $p < 0.0001$, with a moderate mean effect size ($r = 0.43$), indicating no meaningful difference between the meta-analysis in Figure 4 and the sensitivity analysis.

Whilst the meta-analysis indicates a moderate association between increased parental MH and adolescent IS, there is a mixed pattern of association across these six studies. Reaven et al. (2015) reported the weakest association ($r = 0.21$), and is an outlier in this sample of studies as the only RCT (association reported here is from baseline assessment). Adolescents were eligible for the RCT only if they experienced clinically-significant anxiety symptoms. This narrow selection of the adolescents may have therefore weakened the association with parent MH, as compared to a community sample. Kerns et al. (2015) reported the strongest association ($r = 0.67$). This study utilised the most rigorous measure of IS, with clinicians conducting individual parent and child interviews to assign a

measure of severity. It is possible that this more thorough approach was more sensitive to child IS than other measurement approaches.

Two studies included adolescents with cognitive impairment or intellectual disability (Scibelli et al., 2021 - 39% of sample; Fung & Weiss, 2015 – 44% of sample). The remainder either excluded participants with these characteristics (3 studies), or did not specify (1 study). Fung & Weiss (2015) did not analyse their sample by intellectual disability status, but found a significant correlation between parental MH and adolescent IS in their mixed sample ($r(89) = .43$, $p < .01$). Scibelli et al. (2021) separately analysed the group with and without cognitive impairment, and found that whilst both groups showed significant correlations between parental MH and anxiety (without CI: $r(59) = .406$, $p < .001$; with CI: $r(38) = .504$, $p < .001$), for depression and somatising symptoms a significant correlation emerged only for adolescents without CI (depression: $r(59) = .352$, $p < .006$; somatising: $r(59) = .358$, $p < .005$).

This meta-analysis evidences a moderate association between adolescent IS and parental MH, where the strength of association may be impacted by sample selection and IS measure sensitivity. It appears that adolescent intellectual disability is not a key moderator of this effect for anxiety, but may be more relevant to the association when IS measures somatising or depressive symptoms.

2. Peer victimisation

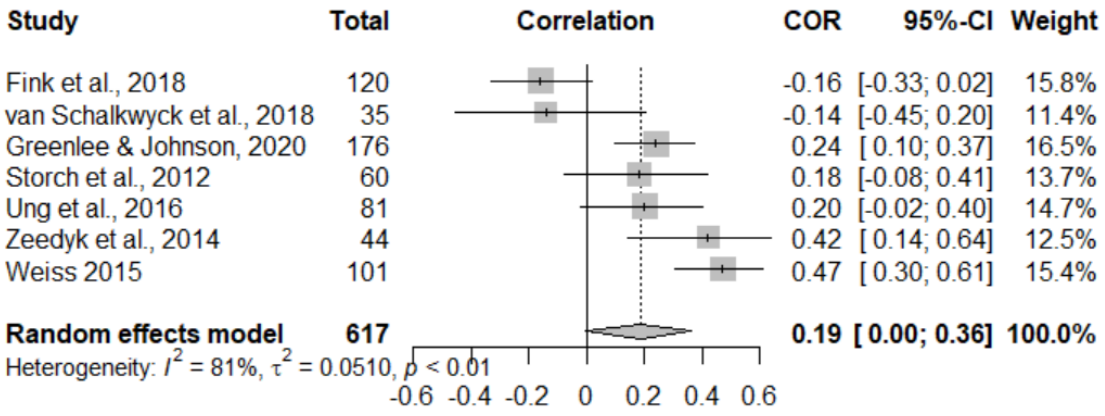
The association between peer victimisation and IS was investigated by 11 studies. Eight of the eleven studies were rated as having at least some concerns or unclear information in their quality assessments (Fink et al., 2018; Van Schalkwyk et al., 2018; Pouw et al., 2013; Storch et al., 2012; Ung et al., 2016; Weiss et al., 2015; Wright, 2017; Zeedyk et al., 2014). Nine studies were suitable for meta-analysis. The remaining two were ineligible for inclusion as they reported only un-standardised regression coefficients (Liu et al., 2021) or structural equation models (Adams et al., 2014) that could not be converted into *r* coefficients.

Given the heterogeneity of the reporter of the adolescent IS, with many studies reporting both parent-reported and young person self-reported measures, separate meta-analyses were conducted for informant (parent)- and self-reported IS. As three studies reported both, they were included in both meta-analyses. In either analysis, the vast majority of peer victimisation measures were reported by the young people themselves. It should be noted that in one study (Fink et al., 2018) young person IS were reported by the young person's teacher, with peer victimisation being reported by the young person's peers. This study was included in the informant-report meta-analysis.

Peer Victimisation – Parent-reported adolescent IS

Seven cross-sectional associations relating peer victimisation to greater levels of parent-reported IS yielded a combined p = 0.053, a small mean effect size (*r* = 0.19) and high heterogeneity (I^2 = 81%), see Figure 3. This is bordering on significance at the p < 0.05 level, and indicates a small positive effect, where increased peer victimisation is associated with increased internalising symptoms in autistic adolescents.

Figure 3



Meta-analysis of the associations between Parent-reported autistic adolescent internalising symptoms, and peer victimisation.

Whilst the meta-analysis suggests a small, positive effect, there is a mixed pattern of association. Fink et al. (2018) and van Schalkwyck et al. (2018) both studied samples with a high proportion of special school attendance or individual education plan (Fink et al., all secondary special school; van Schalkwyck et al., 83% individual education plan). It is possible that these educational contexts are associated with different patterns of victimisation than mainstream educational settings. Additionally, it is of note that Fink et al. used an unusual measure of peer victimisation, with peers using an online tool to nominate the participant's victimisation status.

The strongest associations were reported by: a study with interview-based measures of bullying, which may perhaps be more sensitive (Zeedyk et al., 2014); and a study including a higher proportion of the participants attending mainstream education (Weiss et al., 2015). It may be that this pattern seen for special *versus* mainstream education demonstrates the impact of a poor person-environment fit for autistic adolescents, with peer victimisation contributing to greater IS in mainstream settings.

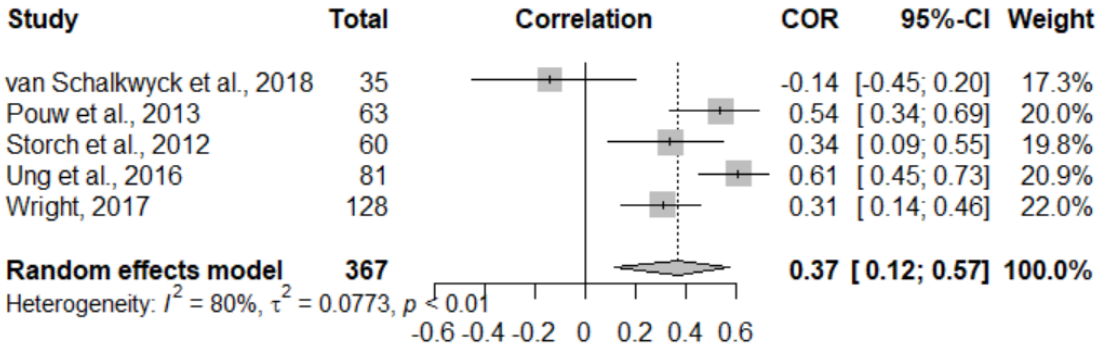
Peer Victimisation – Self-reported adolescent IS

Five cross-sectional associations relating peer victimisation to greater levels of self-reported adolescent IS yielded a combined p = 0.005, with a moderate mean effect size (r = 0.37) and high heterogeneity (I^2 = 80%), see Figure 4. This indicates a highly significant moderately-sized effect, where increased peer victimisation is associated with increased internalising symptoms in autistic young people.

Storch et al. (2012) presented study data for anxiety and depression measures separately. The anxiety measure data was extracted and inputted into the meta-analysis for the analysis in Figure 3, as anxiety was more commonly reported by the other included studies. However, we also conducted a sensitivity analysis (see Appendix 2 for the model) where the anxiety measure reported by Storch & colleagues was replaced by their reported depression measure, in order to establish the effect on the model of this choice of measure. For this sensitivity analysis, the meta-analysis of 5 studies

yielded a combined p = 0.003, and a moderate mean effect size (*r* = 0.38). The sensitivity analysis therefore reveals no meaningful differences.

Figure 4



Meta-analysis of the associations between self-reported autistic adolescent internalising symptoms, and peer victimisation.

From a narrative synthesis, the majority of studies reported that peer victimisation is related to worsened IS in adolescents with autism. Nine studies reported a significant relationship between peer victimisation and higher severity of IS (Adams et al., 2014; Greenlee et al., 2020; Liu et al., 2021; Pouw et al., 2013; Storch et al., 2018; Ung et al., 2016; Weiss et al., 2015; Wright, 2017; Zeedyk et al., 2014), with two studies reporting inconsistent relationships (Fink et al., 2018; Van Schalkwyk et al., 2018). Taking the results of the two meta-analyses, there appears to be a pattern concerning the symptom reporter, with self-reported IS having the greater effect size. Additionally, there may be an effect of who reported the peer victimisation, as is indicated by Ung et al. (2016), where a significant relationship was found between adolescent-reported peer victimisation and adolescent-reported IS, as well as between parent-reported peer victimisation and IS, however the parent- and adolescent-reported measures were not significantly associated with one another.

The eleven studies investigating peer victimisation measured IS in the form of: a broad measure of IS (Adams et al., 2014; Storch et al., 2012; Ung et al., 2016; Zeedyk et al.; 2014; Fink et al., 2018), anxiety (van Schalkwyk et al., 2018; Weiss et al., 2015; Wright, 2017), depression (Greenlee et al., 2020; Pouw et al., 2013; Storch et al., 2012; Wright, 2017), social anxiety (van Schalkwk et al, 2018;

Liu et al., 2021; Storch et al., 2012), as well as panic, separation anxiety, GAD, OCD, and panic disorder (all Storch et al., 2012). Three out of five studies investigating peer victimisation with broad internalising measures found a significant association, with peer victimisation contributing to greater internalising symptoms (Adams et al., 2014; Ung et al., 2016; Zeedyk et al., 2014). Two out of three studies investigating broad anxiety also found significant associations (Weiss et al., 2015; Wright, 2017). All studies investigating broad depression symptoms found significant associations between these symptoms and peer victimisation. For the three studies investigating social anxiety however, only Liu et al. (2021) found a significant relationship.

Several studies measured peer victimisation in finer detail. For example, Adams et al. (2014) measured verbal, relational, physical and social victimisation (all found to have significant relationships with IS when young people, but not parents, reported the victimisation). Equally, Storch et al. (2012) separately measured overt, relational and reputational victimisation (overt and relational victimisation being significantly associated with various presentations of IS across anxiety and depression). Two studies separately investigated cyber and face-to-face victimisation (Liu et al., 2021; Wright, 2017). Across these two studies, no patterns of differentiation appear between cyber or face-to-face victimisation, with both relating to increased internalising symptoms.

The meta-analyses therefore evidence a small-to-moderate association between adolescent IS and peer victimisation, depending on whether the child (stronger association) or adult informant (weaker association) is reporting the IS. This relationship does not seem to be significantly affected by the type of IS construct being measured, or the type of peer victimisation under investigation.

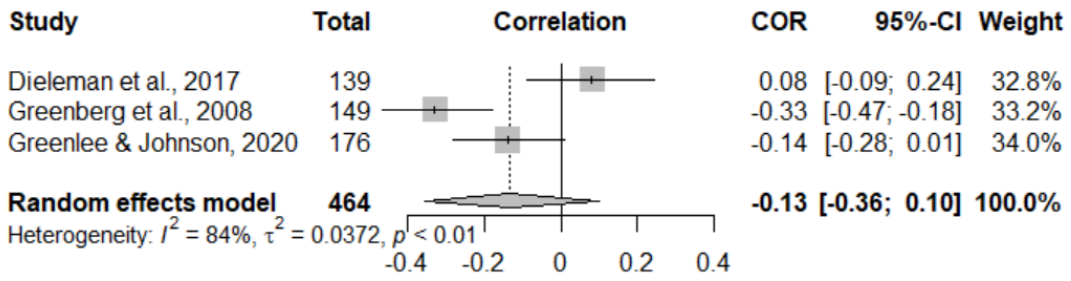
3. Parenting behaviour and/or family interaction style

The association between parenting behaviour / family interaction and young person IS was investigated by six studies. None of the six studies was rated as having concerns or missing information in the JBI quality assessments.

The measures used to investigate parenting behaviour or family interaction in these studies are highly heterogeneous, meaning the results from this subset are more difficult to synthesise. The types of parenting behaviour and family interaction measured encompass purportedly supportive parenting and family interactions, as well as more critical family and parenting styles and accommodation behaviour: 'positive parenting', rules, discipline, harsh punishment (Dieleman et al., 2017; Maljaars et al., 2014), family accommodation (Frank et al., 2020), maternal warmth and praise, quality of mother-child relationship (Greenberg et al., 2008), family functioning (Greenlee & Johnson, 2020), stimulating the development, adapting the environment (Maljaars et al., 2014), parent co-regulation, and motivational- and emotional-scaffolding (Ting & Weiss, 2017). Only Maljaars et al. (2014) measured two parenting behaviours purported to be autism-specific (stimulating and adapting the environment). Due to this heterogeneity, the results reported in the studies were sub-divided by parenting style ('warm', 'critical' and 'accommodating').

Four studies were suitable for meta-analysis, with one study (Ting & Weiss, 2017) being ineligible due to reporting measures of scaffolding and co-regulation which differed too greatly to be comparable with others in the 'warm' parenting subgroup. One additional study (Maljaars et al., 2014) did not report correlation coefficients for the non-significant associations in the 'warm' and 'critical' parenting styles and thus could not be included. As the 'critical' and 'accommodating' parenting styles only included one study each which was eligible for meta-analysis, only the meta-analysis for 'warm' parenting style is reported below.

Figure 5



Meta-analysis of the associations between parenting or family interaction styles, and autistic adolescent internalising symptoms.

Three cross-sectional associations relating warm parenting styles to greater levels of IS yielded a combined p = 0.26, a small mean effect size (*r* = -0.13) and high heterogeneity (I^2 = 84%), see Figure 5. This indicates a non-significant weak negative effect, where greater levels of warm parenting are associated with lower internalising symptoms in autistic adolescents.

A narrative synthesis of the six studies within the parenting style and family interaction theme suggests a weak or mixed relationship between parenting behaviour or family interaction, and IS. Only one study consistently reported significant relationships (Frank et al., 2020), with greater parent accommodation being positively related with child anxiety. The majority of studies reported mixed significant and insignificant (Maljaars et al., 2014; Greenberg et al., 2008; Dieleman et al., 2017) or consistently insignificant relationships (Ting & Weiss, 2017; Greenlee & Johnson, 2020).

Amongst those studies examining parenting or family behaviours hypothesised to be warm or supportive, few relationships were statistically significant, in some cases showing a small positive correlation with adolescent IS (Dieleman et al., 2017 - positive parenting, *r*(137) = 0.08; Greenberg et al., 2008 - maternal warmth, *r*(147) = 0.10). In contrast, several non-significant negative correlations were found (Greenberg et al., 2008 – maternal praise, *r*(147) = -0.09; Ting & Weiss 2017 – scaffolding of anxious, or angry emotions, *r*(51) = -0.18 and -0.14 respectively, and co-regulation (*r*(51) = -0.02 to -0.13). The largest effect was found for relationship quality, with a negative

relationship with young person IS (Greenberg et al., 2008, *r*(147) = -0.33). Interestingly, Greenberg et al. also reports longitudinal relationships between timepoints, and whilst the relationships between maternal praise, maternal warmth and adolescent IS at timepoint 2 were very weak, they show a stronger (negative) correlation with timepoint 3 adolescent IS (praise; *r*(147) = -0.36, warmth; *r*(147) = -0.72). This suggests that the effect of 'positive' parenting styles may be more observable in longitudinal designs.

In contrast, a number of studies examined the relationships between hypothesised 'harsh' or rule-based parenting and internalising symptoms, finding non-significant relationships for rules, discipline, harsh punishment (Maljaars et al., 2014), and a non-significant positive correlation between negative control and adolescent IS (Dieleman et al., 2017).

Within 'accommodating' parenting styles, several paradoxical effects were found, with family anxiety accommodation showing a small, significant positive correlation with severity of anxiety ($r(166) = 0.16$, $p < 0.001$), and Maljaars et al. (2014) reporting two autism-specific behaviours: stimulating the development, adapting the environment, both of which showed small, statistically significant positive correlations with IS (STE: $r(534) = .12$, $p < .007$; ATE: $r(534) = .17$, $p < .007$).

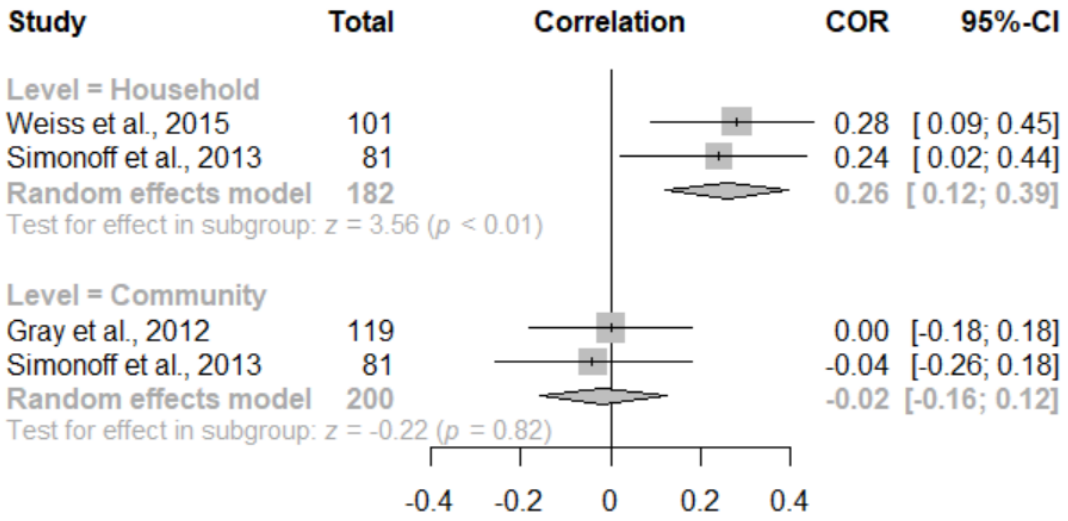
Taken together, the meta-analysis and narrative synthesis therefore evidences a mixed, and largely statistically non-significant set of relationships between parenting and family interaction style and IS. The strongest evidence was for accommodating parenting styles, consistently finding a small positive association with IS.

4. Socioeconomic status (SES)

The association between socioeconomic status and IS was investigated by four studies (Gray et al., 2012; Simonoff et al., 2013; Rosa et al., 2016; Weiss et al., 2015). Two of the four studies were rated as having some missing information in their JBI quality assessments (Gray et al., 2012; Weiss et al., 2015). Three studies were eligible for inclusion in the meta-analysis, with Rosa et al. (2016) being excluded as no coefficients were reported for the relevant analysis.

The four studies measured SES in quite different ways. Three studies measured SES directly in relation to household income or SES; by categorical household income ranging from <$25,000 to >$100,000 (Weiss et al., 2015), or based on family occupation and education, between 1 (low SES) and 63 (high SES) (Rosa et al., 2016) and in the case of Simonoff et al. (2013) using an 8-point scale of parental SES (UK Office of National Statistics, 1996). Two studies measured community-level socioeconomic disadvantage, in the case of Gray et al. (2012) this was done based on family postcode when the child with autism was aged on average 8.7 years old. Simonoff et al. (2013) measured neighbourhood socioeconomic disadvantage based on the Carstairs Index, which combines overcrowding, unemployment, proportion of the population in Registrar General social classes 4 and 5 and households without a car. They also measured family deprivation (based on car ownership and housing tenure). As a result of these two ways of measuring SES (household *versus* community level), the meta-analysis was sub-divided by measurement level.

Figure 6



Meta-analysis of the associations between community and household socioeconomic status and autistic adolescent internalising symptoms.

Examining first the relationship between parental or household SES and young person IS, both Simonoff et al. (2013) and Weiss et al. (2015) found significant positive relationships between

greater parental SES deprivation or lower household income and greater adolescent IS. This yielded a statistically-significant, small pooled effect size (r = 0.26), p < 0.01.

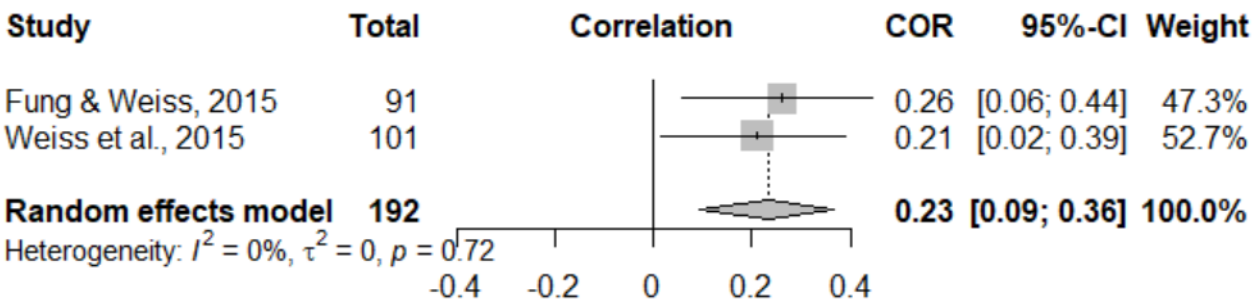
Two studies reported relationships between community SES or deprivation and adolescent IS (Gray et al., 2012; Simonoff et al., 2013). Neither study found a statistically significant relationship between neighbourhood deprivation or SES and adolescent IS. This is reflected in the pooled effect size of r = 0.02, p = 0.82.

A synthesis of the four studies therefore suggests a small, statistically-significant relationship between parental or household SES and adolescent IS, where greater deprivation or lower SES is related to greater young person IS. No relationship was found between community SES and adolescent IS.

5. Negative Life Events

The association between negative life events (NLE) and young person IS was investigated by three studies. All three studies were rated as having some missing information in their JBI quality assessments (Fung & Weiss, 2015; Taylor & Gotham, 2016; Weiss et al., 2015). Two studies were eligible for meta-analysis, with Taylor & Gotham (2015) being excluded as it did not report an effect size that could be converted to *r.*

Figure 7



Meta-analysis of the associations between negative life events and autistic adolescent internalising symptoms.

The pooled effect of the meta-analysis indicates a small, statistically-significant positive association between NLE and adolescent IS. This yielded a statistically-significant, small pooled effect size (r = 0.23), $p < 0.01$. Taylor & Gotham equally reports a significant positive relationship between NLE and IS (OR= 6.57). This suggests that experiencing a greater number of NLEs is associated with worsened IS in autistic young people.

NLE were measured in each study by parent-report, asking parents whether their child had experienced a number of NLEs within the last year (Weiss et al., 2015; Fung & Weiss, 2015), or over their lifetime (Taylor & Gotham, 2015 - only those events in which the child was said to be 'extremely' affected were included in analyses).

Fung & Weiss (2015) conducted further analyses on the number of NLE experienced by the adolescents in their sample, finding that whilst depression scores did not significantly differ between adolescents with 0-2 NLE, those experiencing 3 or more negative life events had significantly greater depression scores ($p = 0.01 – 0.06$ depending on the number of NLE).

6. Social interaction

The association between social interaction and internalising symptoms was investigated by two studies (Dallman & Harrop, 2021; Mazurek, 2010). Both studies were rated as having some missing information in their JBI Quality Assessments.

Dallman & Harrop used an 'ecological momentary analysis' study design over 7 days, where participants were prompted on their mobile phone to complete a brief questionnaire about their current activities, as well as the Positive and Negative Affect Scale (PANAS) to assess momentary positive and negative affect on several occasions per day. Social interaction was measured by momentary level of, quality of, and enjoyment of social interaction. Mazurek (2010) in contrast, measured the relationship between parent-reported IS and parent-reported dyadic friendship (as part of the Autism Diagnostic Interview – revised).

A synthesis of the two studies is tentative, given the differing designs and measures of social interaction. For this reason only narrative synthesis was used, as the measures are not sufficiently similar to pool in a meta-analysis. Mazurek (2010) reports a significant negative correlation between quality of dyadic relationships and symptoms of depression ($r(1200) = -0.15$, $p < 0.001$). Dallman & Harrop (2021) found that in their momentary ecological analysis, momentary quality of social interaction was significantly negatively related to negative affect ($p < 0.01$). Associations between level of social interaction and enjoyment of current activity were not significantly related to negative affect. Despite these studies using different methodologies to measure social interaction, both show statistically significant negative relationships between quality of social interaction and IS.

7. School Placement

The association between school placement and adolescent IS was investigated by two studies. One study was rated as having some missing information in the JBI quality assessments (Zainal, 2019). A meta-analysis was not performed for this theme as only Simonoff et al. (2013) reported a correlation coefficient.

Simonoff and colleagues asked parents to report on their child's school placement at age 12; as either mainstream or special school. Zainal and colleagues categorised adolescents as: not attending school; special education school; special class in mainstream school; mainstream school; or 'other'.

A synthesis of the two studies suggests little or weak associations between school placement and adolescent IS in these samples. Simonoff et al. found the correlation between adolescent IS and special school placement to be non-significant (r (79) = 0.04) when measured using the Strengths and Difficulties Questionnaire. Zainal and colleagues conducted Mann-Whitney U tests, to compare IS between adolescents attending mainstream *vs*. special schools. A significant difference in IS between autistic adolescents in mainstream or special schools was found for only one anxiety subscale (social anxiety) of the Spence Children's Anxiety scale, or Developmental Behaviour Checklist: $U(N_{mainstream} = 27, N_{special\ school} = 69) = 698.00$, $z = 1.92$, $p = 0.02$, with social anxiety levels

being greater at mainstream schools. All other associations between school placement and overall anxiety or specific anxiety subscales were found to be non-significant.

8. Covid-19 pandemic and public health measures

The association between the Covid-19 pandemic and internalising symptoms was investigated by one study (Lugo-Marin et al., 2021). This study was rated as having some concerns and missing information in the JBI quality assessment.

Lugo-Marin et al. measured adolescent IS in relation to the Covid-19 pandemic lockdown measures (pre-, and post-lockdown measures). Adolescent IS was measured in this study by parent-report. The mean age of the sample was relatively young within the scope of our adolescent review (10.7 years old).

The study measured the differences in adolescent IS before and after the Covid-19 lockdown began, finding no significant differences (Wilcoxon signed-rank, $Z = -1.81$, $p = > 0.05$).

9. Social support

The association between social support and adolescent IS was investigated by one study (Ung et al., 2016). This study was rated as having some concerns in the JBI quality assessment.

Social support was investigated in this study via the Social Support Scale for adolescents, a 24-item self-report questionnaire which assesses perceived social support and positive regard from parents, close friends, classmates, teachers and school staff. Young person IS were reported separately by both self- and parent-report.

Ung et al. conducted regression analyses and found a significant negative association between social support and self-reported IS (ß(77) = -0.54, p = 0.003), although the relationship between social support and parent-reported IS was marginally non-significant (ß (77) = -0.30, p = 0.07).

10. Parental education

The relationship between parental education and adolescent IS was investigated by one study (Simonoff et al., 2013). This study was rated without concerns or missing information by the JBI quality assessment.

Parental education in this study was determined by the highest household parental education on a 6-point scale, ranging from no GCSEs through to postgraduate qualifications.

The results of a correlation analysis found no significant correlation between IS and higher parental education ($r(79) = -0.05$).

11. Pet ownership

The relationship between pet ownership and adolescent IS was investigated by one study (Ward et al., 2017). This study was rated as having some missing information by the JBI quality assessment.

Pet ownership was measured by parent-reported questionnaire, asking about the number of pets owned, how long the pet has been owned, whether the child considers the pet their 'best friend', and the level of child responsibility, comfort and companionship with the pet. Both parent-reported and adolescent-reported IS were collected.

Ward et al. conducted correlation analyses for relationships between responsibility for pets, using pets for comfort, and using pets for companionship with both parent-reported and self-reported IS. For self-reported IS, significant positive correlations were found for using pets for comfort ($r(71) = 0.22$, $p < 0.001$), and using pets for companionship ($r(71) = 0.28$, $p < 0.001$). Investigating the relationships with parent-reported IS, a significant positive relationship was found with using pets for comfort ($r(71) = 0.26$, $p < 0.001$), and a significant negative relationship for taking responsibility for pets ($r(71) = -0.25$, $p < 0.05$). It should be noted that a negative correlation was also found between self-reported IS and pet responsibility, although this did not meet statistical significance.

DISCUSSION

Summary

In this systematic review we have synthesised the findings of 29 studies, encompassing 3,999 autistic young people. All studies were investigated by narrative synthesis, and six meta-analyses were conducted (including a total of 1,436 participants across 23 studies).

1. What environmental factors are associated with internalising symptoms in autistic young people?

Addressing the first question, this review found 11 themes of environmental factors in the studies that met our inclusion criteria, with eight of these showing some meaningful association with IS experienced by autistic young people; parental MH or stress, peer victimisation, parenting behaviour or family interaction, socioeconomic status, negative life events, social interaction, social support, and pet ownership. In this study sample, school placement, parental education and impact of covid-19 pandemic measures showed weak, or no, meaningful relationships with IS. It should be noted that in the case of these last three environmental factors, only between one and two studies were identified for each factor, and therefore a lack of data may be precluding the identification of a stronger association.

2. What is the strength of association between these environmental factors and internalising symptoms in autistic young people?

Taking into account the narrative synthesis as well as the meta-analyses, the greatest weight of evidence for a reliable association between the environment and IS is for parental MH or stress, peer victimisation, negative life events and socioeconomic status. As these environmental factors were investigated by a larger number of studies than other factors, this may have added to the weight of our findings. It is also clear that these environmental factors have been the subject of more research than other factors.

Both NLE and SES also showed robust trends for strength of association with IS. Environmental factors with some, but weaker, evidence of association with IS in autistic young people are social support, social interaction and pet ownership. Only two studies measured social interaction, and one each measured pet ownership and social support, so the limited number of studies available has limited the strength of associations we can identify in this study.

As described throughout the narrative synthesis, the measures used to captured environmental factors within the studies included in this review may have influenced the strength of association with IS. For example, SES showed an interesting difference in association with IS depending on whether household-level or community-level SES was being measured. Prior research in population-level samples has also reported that the associations between SES and child development or psychopathology are highly sensitive to how SES is defined and measured (Webb et al., 2017; Boelens et al., 2020). These studies consistently report material deprivation (such as lower income, lower access to material goods) to be more closely associated with child development or psychopathology, mirroring our findings for this environmental factor theme.

Additionally, as this research aim asked; "What is the strength of association between these environmental factors and internalising symptoms in *autistic young people*?", it is important to consider whether the measurement tools used were accurately capturing environmental factors as experienced by autistic young people. Whilst all participants included in this review had a research or clinical diagnosis of autism, many of the measures used to capture IS or environmental factors were not specifically designed or adapted for autistic adolescents or young people. It is therefore possible that some aspects of the 'strength' of the associations identified here have not been fully captured. Within the environmental factor theme of NLE for example, NLE were measured using standard measures that were originally developed for typically-developing samples. It is possible that young people with autism may consider other categories of life events as significantly negative to be included in a such a list, for example events that relate to sensory experiences, social-

communication, or exposure to environments designed overwhelmingly for typically-developing young people. Another example of this may be 'social support', which may function differently for autistic adolescents and young people than typically-developing individuals, or preferentially take place online as opposed to face-to-face, and therefore existing measures of social support may not capture these aspects that are most relevant to people with autism.

Finally, it should be noted that as all studies were evaluated cross-sectionally, we cannot infer longitudinal trends from this systematic review. Therefore, whilst robust associations between environmental factors and worsened IS were found for a number of environmental factor themes, we cannot infer causality. Equally, prior research has identified bi-directionality between IS and a number of environmental factors in samples of typically-developing young people (such as parental mental health, Sifaki et al., 2021; and peer victimisation, Reijntjes et al., 2010). It is possible that there may also be some bi-directionality in the associations between environmental factors and IS for young people with autism, which could not be captured within this systematic review as a result of the cross-sectional measurement.

Limitations

There are a number of limitations to this systematic review. One limitation concerns the participants within the identified studies. A substantial proportion of the participants were autistic boys or young men, as opposed to autistic girls or women, and not presenting with cognitive impairment or learning disability. Girls and women, and young people with a learning disability may experience IS differently than the predominantly higher IQ boys and young men featured in this review. Equally, where parents were reporters of the young people's experience, or reporting their own parenting behaviours, MH or stress, they were predominantly mothers. Whilst most studies did not exclude fathers, the identities of the parent taking part in each study were disproportionately reported as mothers.

The age range in our review was broad, spanning from a mean age of 9 years older, to 24 years old. Whilst this encompasses the 'young person' definition used for our inclusion criteria, and includes the developmental stage of adolescence and gradually increasing independence, there are likely to be very important differences between the internal experiences that 9- and 24-years-olds face, and the environmental factors they are subject to. It would be beneficial for future research to investigate IS and corresponding environmental factors in narrower age ranges, or longitudinally, in order to better understand these differences.

One difficulty in both narrative and meta-analysis was the heterogeneity of the studies, in terms of their measures, focus of study, and participants, limiting the possible analyses, and the conclusions that can be reliably drawn from this data. Whilst it was important for our study to cast the net widely so as not to miss key environmental influences on autistic young people, and so as to identify all potentially relevant studies, it would be beneficial for future research to focus more specifically on key participant groups, environmental factors, or components of IS (such as anxiety), in order to draw further conclusions.

Lastly, the majority of statistical analyses reported in the identified studies were bidirectional (such as correlation analyses), and as such few conclusions can be drawn about causality.

Future research:

This review is to our knowledge the first systematic review to investigate a broad set of environmental factors in relation to IS experienced by autistic young people. Our findings have identified that whilst certain environmental factors are better studied (parental MH, or peer victimisation), other important factors warrant further attention. In our study SES, NLE, social interaction, social support and pet ownership all showed significant associations with IS, however only 1-4 studies were identified in each case. In particular, those environmental factors that may relate differently to autistic young people than NT young people warrant further study in this population. Examples of this are social interaction or social support, given that one key feature of

autism is difficulty with social communication, this environmental factor may relate differently to young people with autism than what can be drawn from existing research on NT groups.

Another consideration for future research is who is included in participant samples. The studies identified in this review were heterogeneous when it came to exclusion criteria, and whilst several samples did include young people with learning disabilities or cognitive impairment, many also excluded them. This is important both in enabling us to interpret the results of the studies (only one reported results separately for young people with cognitive impairment and without, finding considerable differences in results), but also for their generalisability. We know that in clinical practice comorbid cognitive impairment or learning disability is common with autism, and therefore it would be beneficial to include this group in research too.

Reporter effects were an interesting and consistent feature of the results of this review, and suggest that future research should carefully consider the use of different reporters for different measures; such as child-reported IS in relation to parent-reported victimisation. Studies in this review which measured both parent- and self-report for all measures repeatedly found that the experiences reported by parent- *vs.* self-reported measures were quite different, and showed varying strengths of association with the environmental factor of interest.

Within this review two studies investigated cyber victimisation specifically, in addition to 'traditional' face-to-face victimisation. Whilst there were not sufficient cyber-victimisation studies identified in this review to draw further conclusions, this would be an important area for future research, as social media and internet applications become more widespread and sophisticated. This may be particularly relevant for autistic young people, who may be more attracted to socialising in an online context than their NT peers.

Additionally, only one study relating to the Covid-19 pandemic was eligible for inclusion in this review. Anecdotally, there have been many reports of the mental health impact of the Covid-19 pandemic, both for autistic and non-autistic people, although specific challenges in relation to the

pandemic may exacerbate the impact on autistic young people; such as difficulties with routine change, the impact of mask wearing on social communication difficulties or sensory differences and disproportionate disruption to school placements such as special schools where a greater proportion of autistic young people than NT young people may attend. Whilst no significant relationship with IS was found for the study included in this review, it may be that studies with a longer period of study during the pandemic, or which were not yet published at the time of our searches, will report different findings.

Clinical Implications

The majority of the environmental factors identified in this review are potentially modifiable, and therefore may be suitable targets for intervention, to improve the mental health and quality of life of young people with autism. This may be in the form of early identification and screening (such as screening for peer victimisation, parental MH or NLE), or improving access to services for individuals or families that are exposed to one or more of these factors. This may for example be done by co-locating services, for example those providing housing or financial support, within MH services (in the case of low SES), or assessing proactively for parental MH when autistic young people present with MH difficulties.

These environmental factors can also be considered from a 'preventative' lens – if the impact of some factors such as low SES, peer victimisation, parental MH or NLE are reduced or prevented, this may reduce the burden of IS on young people with autism. These findings may therefore add weight to arguments for research and resource distribution in these areas.

Several environmental factors also suggested possible 'protective' associations, such as social interaction, social support, and taking responsibility within pet ownership. Further research in these areas would be beneficial to establish whether emphasis on these factors as part of parenting advice, education programmes or social care interventions may contribute to resilience against IS in young people with autism.

One interesting clinical implication relates to the pattern that was observed for different reporters of IS or environmental factors such as peer victimisation. Parent and young person ratings of victimisation or IS often differed, and showed varying strengths of association, with young person self-reported ratings often showing stronger associations with environmental factors than parent- or teacher-report. Within this older age group (9 years old or greater), it may be that young people are increasingly able to report on their own experience, and naturally have more direct access to their everyday interactions and internal experiences than their parents or educators do. Given the trends found in this review, it is important to be aware of who the reporters are in clinical practice, for example within clinical assessment and treatment, and in clinical questionnaires, in order to correctly interpret the information given.

CONCLUSION

Several potentially-modifiable environmental factors were consistently associated with worsened internalising symptoms for autistic young people: parental mental health or stress; peer victimisation; low socioeconomic status and negative life events. Environmental factors suggesting a potentially protective association with internalising symptoms were also identified: social interaction; social support and pet ownership. The findings of this systematic review prompt further study; to understand whether these associations hold in a younger age group, to replicate the findings in those themes where the fewest studies were identified, and for further study of environmental factors most likely to relate to autism, such as environmental sensory stimuli, social support and social interaction. Reporter-effects were consistently identified where there were both self- and parent-reported measures, which may inform the use and interpretation of measures in both research and clinical practice.

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Part 2: Empirical paper

Relationships between parental mental health, peer victimisation and internalising symptoms in children with autism

ABSTRACT

Background: Anxiety and depression ('internalising symptoms') are common in children with autism, however the factors underlying this association are poorly understood. There is evidence that particular risk factors, such as parental mental health, and peer victimisation may contribute to the development of internalising symptoms for typically-developing children, but it is unknown whether these associations hold for autistic children. Additionally, prior research has identified bidirectionality between risk factors and internalising symptoms in typically-developing children. This study therefore investigates the bi-directional longitudinal relationships between two hypothesised risk-factors (peer victimisation and parental mental health) and internalising symptoms in children with autism.

Methods: Participants were 560 autistic children who participated in the Millennium Cohort Study, a population-based UK birth cohort. Internalising symptoms were measured by parent-report using the Emotional Problems sub-scale of the Strengths and Difficulties Questionnaire at ages 3, 5, 7, 11, 14 and 17. Parental mental health and peer victimisation were also measured at each timepoint by parent-report. Random-Intercept Cross-Lagged Panel Models were used to examine the bidirectional associations between parental mental health and peer victimisation with internalising symptoms across these six timepoints.

Results: An RI-CLPM of child internalising symptoms and parental mental health revealed stable longitudinal trajectories, significant associations between variables at each timepoint, and bidirectional longitudinal relationships: with parental mental health difficulties predicting child internalising symptoms from 3- and 5-years-old, to 5- and 7-years-old, and conversely, with child internalising symptoms predicting greater parental mental health difficulties from 14- to 17-years-old. The RI-CLPM of child internalising symptoms and peer victimisation equally revealed stable longitudinal trajectories and significant associations at each timepoint, however identifying only one cross-lagged association: with greater peer victimisation at 5-years-old predicting greater

internalising symptoms at 7-years-old. The implications of covariates are discussed with reference to additional RI-CLPMs, constructed using Multiple Imputation for missing covariate data.

Conclusions: Peer victimisation and parental mental health appear to be important risk factors for the development of internalising symptoms in autistic children. Additionally, child internalising symptoms in adolescence appear to be a predictor of later parental mental health. Child sex, IQ and ethnicity (entered as covariates in the RI-CLPMs) weaken the relationship between internalising symptoms and parental mental health, warranting further study of how these demographic characteristics function in the context of this relationship. Limitations and implications for further research are discussed.

INTRODUCTION

Autism Spectrum Disorder (ASD) is a diagnosis used to describe individuals displaying a characteristic set of; difficulties in social communication, sensory sensitivity, restricted and repetitive behaviours and highly restricted interests (American Psychiatric Association, 2013). The difficulties in these characteristic domains begin early in life, but may not be recognised or diagnosed until mid- to late-childhood, or even into adulthood (Hosozawa et al., 2020). ASD is a life-long neurodevelopmental condition, and has an estimated prevalence of 1.1% in the UK (NHS, 2012). It is important to stress however that the definition of ASD by these 'characteristic difficulties' has received critique for its narrow focus on perceived biological deficits and emphasis on the individual as opposed to their wider environmental and social context (Pellicano & den Houting, 2021). Recent activism, advocacy and research has considered ASD through the lens of 'neurodiversity' in order to recognise it as one facet of the range of natural diversity that exists in human neurodevelopment (Blume, 1998; Singer, 1998).

A variety of terms have been used to refer to and communicate about ASD in community, clinical and research settings, for example 'people with autism', 'ASD', 'autistic', or 'on the autism spectrum'. In order to acknowledge the variety of preferred nomenclature within the community of autistic people, their families, clinicians and researchers, in this study I will interchangeably use the terms 'autistic person' [or child, or young person] as well as 'person [or child, or young person] with autism'. This is in order to remove the emphasis on autism as a 'disorder' (as implied by 'Autism Spectrum Disorder'), and balance the use of 'person-first' (person with autism) and 'identity-first' language (autistic person). These frames of reference were most highly endorsed by a 2015 study of autistic people, parents, families and friends, and professionals working with autistic people (Kenny et al., 2015). This is in line with guidance from Autism Europe (2017), and The National Autistic Society UK (2015). For the purpose of this study, the word 'autism' or 'autistic' will refer to any

person who has received a clinical or research diagnosis of autism spectrum disorder, or associated historic diagnoses such as Asperger's syndrome and Pervasive Developmental Disorder.

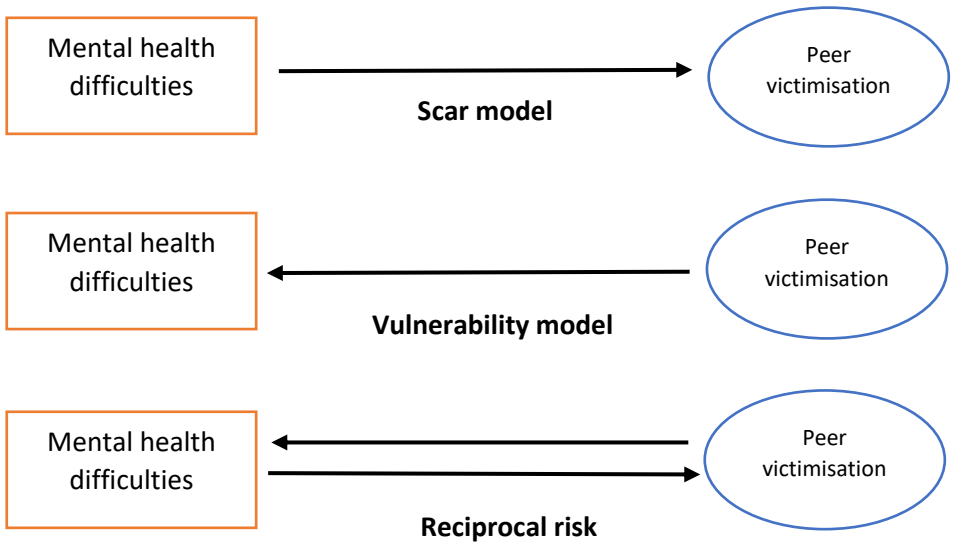
Autistic children have a higher prevalence of emotional and behavioural difficulties than non-autistic children (Lai et al., 2019). Research has estimated that 70% of 10- to 14-year-old autistic children meet criteria for at least one mental health disorder, with 41% meeting criteria for two or more (Simonoff et al., 2008). Co-occurring psychological difficulties are also associated with increased impairment and reduced quality of life for both the child and their families (Kaat, Gadow & Lecavalier, 2013; Posserud et al., 2018). This is contrasted against current emotional and behaviour difficulty prevalence estimates for typically developing children of 17 – 25% (NHS Digital, 2021; Deighton et al., 2019).

There is some empirical evidence on which factors may be associated with or predict the occurrence of mental health difficulties amongst children in the general population. Existing research has identified a wide range of hypothesised risk factors for the population of children as a whole: family socio-economic status, negative life events, parental mental health history, marital and family conflict, parent-child relationship quality, parenting behaviour, sleep problems and peer victimisation (Harland et al., 2002; Essex et al., 2006; Johnco et al., 2021; Narmandakh et al., 2021). In the case of several risk factors, bi-directional relationships have also been identified (Peer victimisation, Reijntjes et al., 2010; cyber victimisation, van den Eijnden et al., 2013; paternal psychological distress (Sifaki et al., 2021). For example, in the case of peer victimisation, it has been hypothesised that this bidirectional relationship may operate by peer victimisation acting as a risk factor for worsened mental health, with mental health difficulties additionally conferring vulnerability to bullying.

This can be considered from the perspective of the vulnerability, scar, and reciprocal risk models (as used to conceptualise the relationship between depression and low self-esteem, see Figure 1; Johnson et al., 2016; Orth & Robins, 2013). The vulnerability model proposes that an external risk

factor (such as parental criticism, or peer victimisation) may contribute to an individual's vulnerability to mental health difficulties. Conversely, the scar model proposes that mental health difficulties may cause a 'scar' that predisposes the individual to, for example, subsequent victimisation or parental criticism. Finally, the reciprocal risk model proposes a mutual association with predictive power of both variables on one-another.

Figure 8



Diagrammatic representation of Scar, Vulnerability and Reciprocal Risk models, with Peer Victimisation as example risk factor

Whilst a number of risk factors have similarly been identified for mental health problems of children with autism (see systematic review paper in previous chapter of this thesis), further research is required to understand how these risk factors operate, particularly over time and during child development, and whether any of these factors may operate bi-directionally with mental health difficulties. As in the systematic review paper presented in the previous chapter, I felt it important to focus on environmental risk factors in this study (factors existing outside of the young person), as opposed to individual risk factors (such as general intelligence, or expression of autistic traits). This felt particularly important in order to account for the wider environmental and social contexts in which autistic people find themselves, rather than locating risk factors within the individual

(Pellicano & den Houting, 2021). This approach also lends itself to identifying potentially modifiable risk factors, rather than purely identifying individuals who may be at risk as a result of individual differences.

A distinction is commonly made between 'externalising' and 'internalising' disorders or symptoms experienced by children (Achenbach, 1978), where externalising refers to hyperactivity, aggression and anti-social behaviour, and internalising refers to withdrawn, anxious, inhibited, and depressed behaviours. Internalising symptoms are known to be common in autistic children, and prevalence studies have found them to manifest in common diagnosable difficulties; social anxiety disorder (Simonoff et al., 2008), generalised anxiety disorder and phobias (Salazar et al., 2015). Research has also shown that difficulties with affect and anxiety in childhood often persist into early adulthood for people with autism (Simonoff et al., 2013; Gotham et al., 2015), and are associated with lower life satisfaction and emotional regulation (Gotham et al., 2015). Internalising symptoms are also a concern for autistic people themselves, as put forward in a 2016 priority-setting initiative (Autistica & James Lind Alliance, 2016). Out of the top ten priorities for autism research, both the first-ranked priority ("Which interventions improve mental health or reduce mental health problems in people with autism?") and the fourth-ranked priority ("Which interventions reduce anxiety in autistic people?") concern internalising symptoms in the form of mental health problems such as anxiety, depression and obsessive-compulsive disorder. For the above reasons, the present study is focused on internalising symptoms as experienced by autistic children.

Longitudinal cohort studies are an excellent resource for this type of research question, as they enable the investigation of patterns within the same population over time. A number of studies have utilised longitudinal cohort studies to answer research questions concerning autistic children. Findings included significant associations between family poverty and emotional difficulties (Midouhas et al., 2013; Flouri et al., 2015), between later age of ASD diagnosis and depression or

self-harm (Hosozawa et al., 2021), and between maternal wellbeing and child behaviour problems (Totsika et al., 2013).

One analytical method that is particularly suitable for identifying bi-directional associations in longitudinal data such as this are Cross-Lagged Panel Model (CLPM) approaches, (Sage, 2017). CLPMs and extensions of the CLPM model have now been used widely to investigate longitudinal patterns of mental health difficulties in association with hypothesised risk factors (for example: Totsika et al., 2013; Fredrik et al., 2021; Sifaki et al., 2021, Neville et al., 2021, amongst others). An advantage of the CLPM is its ability to capture change in key variables over time, the strength of association between variables at each timepoint, and at adjacent future timepoints. This allows for an estimation of the predictive power of a potential risk factor on mental health difficulties, or conversely, of mental health difficulties on the factor. Recent extensions of the CLPM have been proposed, such as the Random Intercept Cross-Lagged Panel Model (RI-CLPM) which aims to separate out stable (trait-like) between-person variation from the model by creating latent variables (Hamaker et al., 2015), so that the cross-lagged associations represent only the within-person variation over time. RI-CLPMs are now considered amongst the best approaches to an estimation of causality between factors in the absence of experimental manipulation, enabling more robust conclusions to be drawn (Orth et al., 2021)

In this study we make use of a longitudinal cohort dataset and longitudinal analytic techniques (RI-CLPM) to pursue the following research aims:

- To explore the longitudinal relationship between parental mental health and internalising symptoms in children with autism
- To explore the longitudinal relationship between peer victimisation and internalising symptoms in children with autism

METHODS

Sample

The sample was drawn from the Millennium Cohort Study (MCS, https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/), a population-representative birth cohort study in the UK, which follows the health and development of children born between September 2000 and January 2002. The original sample comprised 19,231 families. At the time of writing, data are available from 7 rounds of data collection, referred to in MCS as 'sweeps', of the cohort to date (Table 1).

Table 4

Sweep	1	2	3	4	5	6	7
Age of child	9 months	3 years	5 years	7 years	11 years	14 years	17 years

Millennium Cohort Study sweeps and ages of children

For this study, data from the MCS were accessed from the UK Data Service (https://ukdataservice.ac.uk/). Data for all available sweeps (1 – 7) were used.

The MCS itself had 3 inclusion criteria:

1) For the child to be born within eligible dates; between 1/9/2000 and 31/8/2001 (England & Wales), and between 23/11/2000 and 11/01/2002 (Scotland and Northern Ireland)
2) For the child to be alive and living in the UK at age nine months
3) For the child to be eligible to receive Child Benefit at age nine months

For the purposes of this study we have introduced one further inclusion criterion:

4) For the parent to have indicated that the child has been given a diagnosis of Autism Spectrum Disorder

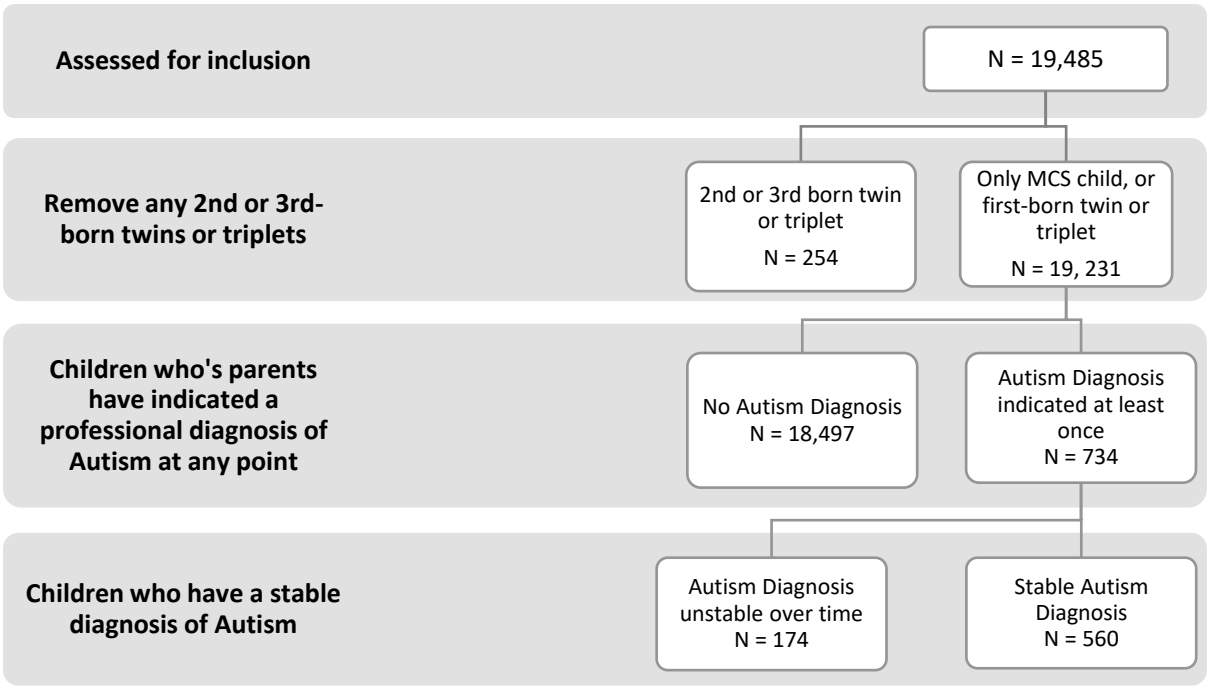
In the MCS, parents were asked 'Has a doctor or other health professional ever told you that your child had Autism, Asperger's Syndrome or other autistic spectrum disorder?' when the child was

approximately 5-, 7-, 11-, and 14-years-old. Using records for one child per family (the first-born, in the case of multiparous births), the sample therefore comprised of children whose main parent responded 'yes' to this question at least once, and who did not respond 'no' to the same question in any subsequent sweeps. This resulted in an analytic sample of 560.

Other studies concerning autistic children in the MCS sample have taken a more conservative approach to identify children with stable autism diagnoses (Mandy et al., 2022), including children only if their parent gave a valid response to this question in at least 3 out of 4 sweeps. From our sample, this results in a sample size of 415. We therefore conducted sensitivity analyses using this sample (see appendix).

See the participant flow chart in Figure 2 for a summary of the sample selection.

Figure 9



Participant flow chart

Measures

Internalising symptoms

– Internalising symptoms were measured using the parent-report Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). The questionnaire comprises 25 items, of which 5 sub-scales can be derived; Emotional symptoms, Conduct problems, Hyperactivity / inattention, Peer relationships problems, and Prosocial behaviour. To investigate internalising symptoms for the purposes of this study, only the Emotional symptoms sub-scale was used (see Table 3 for sub-scale items).

Table 10

Item	Responses
3. Often complains of headaches, stomach-aches, or sickness	0 – Not true; 1 – Somewhat true; 2 – Certainly true
8. Many worries, often seems worried	
13. Often unhappy, downhearted, or tearful	
16. Nervous or clingy in new situations, easily loses confidence	
24. Many fears, easily scared	

Strengths & Difficulties Questionnaire, Emotional Problems sub-scale

The parent-report SDQ has established reliability and validity (Goodman, 1997) and has been extensively used with autistic children (e.g., Simonoff et al., 2013, Colvert et al., 2022). The Emotional symptoms sub-scale has been found to be a valid measure of internalising symptoms, and comparable to other measures of internalising symptoms (such as the CBCL Internalising sub-scale; Goodman & Scott, 1999). The emotional symptoms sub-scale has been found to have acceptable internal consistency (Cronbach's alpha of 0.78) for the parent-report SDQ in a sample of autistic children (Findon et al., 2016). In the MCS, the parent-report SDQ was completed by the main parent when the child was aged approximately 3-, 5-, 7-, 11-, 14- and 17-years-old.

Peer Victimisation

– Peer victimisation was measured by item 19 of the parent-reported SDQ at each time point (child aged approximately 3-, 5-, 7-, 11-, 14- and 17-years-old). Parents were asked whether their child was "Picked on or bullied by other children", choosing from one of three multiple-choice responses ("Not true", "Somewhat true", "Certainly true").

Parental Mental Health – Parental MH was measured by parent self-report at each sweep, using the Kessler-6 questionnaire (K6). The K6 is a brief screening measure of psychological distress, made up of six items (Table 4):

Table 11

During the last 30 days, how often did…	Responses
… you feel nervous?	0 – None of the time; 1 – A little of the time; 2 – Some of the time; 3 – Most of the time; 4 - All of the time
… you feel hopeless?	
… you feel restless or fidgety?	
… you feel so depressed that nothing could cheer you up?	
… you feel that everything was an effort?	
… you feel worthless?	

Kessler 6 questionnaire items

The K6 has shown good reliability and validity across a variety of socio-demographic subsamples, including for community mental health screening in a variety of countries (including Ethiopia, Vietnam, Australia, China (Tesfaye, Hanlon, Wondimagegn, & Alem, 2010; Kawakami et al., 2020; Kang et al., 2015; Slade, Grove & Burgess, 2010)), as well as excellent internal consistency, with Cronbach's alpha of 0.89 across subsamples (Kessler et al., 2003).

It is shown to reliably discriminate between community cases and non-cases of diagnosable DSM disorders, and to correlate with depression and anxiety (as defined by the DSM-IV), (Kessler et al., 2002).

Covariates

A number of covariates are included in our study in order to control for any potentially confounding relationship these may have with the variables of interest. In line with previous research focusing on autistic children in the MCS (Midouhas et al., 2013; Hosozawa et al., 2020), as well as the systematic review reported earlier in this thesis, the following covariates were considered: child sex, birthweight (kilograms), IQ measured at 7-years-old (computed factor score), parental education (highest NVQ level or equivalent), child ethnicity (white *vs* any non-white ethnicity), and socio-

economic status (SES; banded family income). See Appendix 9 for the measures used to derive these covariates.

As the structural equation models (RI-CLPMs) attempt to segregate between-person variation to be captured by the random-intercepts, leaving only within-person variation in the central regression-path model, I reasoned that it was important not to additionally over-control the model for between-person variation in the form of covariates. For this reason, RI-CLPMs were run first as unadjusted models, and then secondly with a limited set of covariates (sex, ethnicity and IQ), before thirdly with the full set of covariates listed above (see appendices 4 and 5). Sex, Ethnicity and IQ were selected as the covariates to be included in the limited set for a number of reasons. There are documented sex-differences in the presentation of autistic traits and psychological or behavioural difficulties in samples of autistic children (Holtmann et al., 2007; Mandy et al. 2012; Solomon et al., 2012). IQ varies widely in our sample and includes a proportion of children that fall within the range for likely intellectual disability. Prior research has identified impacts of intellectual disability on behavioural and social difficulties (Baker et al., 2020) and peer victimisation (Sentenac et al., 2011). Additionally, in the UK differences have been observed in the prevalence and trajectory of mental health symptoms between children of differing ethnicities (Atzaba-Poria et al., 2004; Midouhas 2017; Terhaag et al., 2021). Whilst these effects are not yet documented within an autistic sample, it is possible that they may generalise to this population as well. These three covariates were therefore considered most important to include as potential confounding variables within the RI-CLPM.

Ethics

Ethical approval for the MCS was granted by the UK National Health Service Research Ethics Committee ahead of each new sweep of data collection, and written consent from the children's parents was also obtained at each sweep.

Consent for the present study was granted under application 19439/001 by the UCL Research Ethics Committee.

Data for the MCS was accessed from the UK Data Service under an End User License (EUL).

Data analytic plan

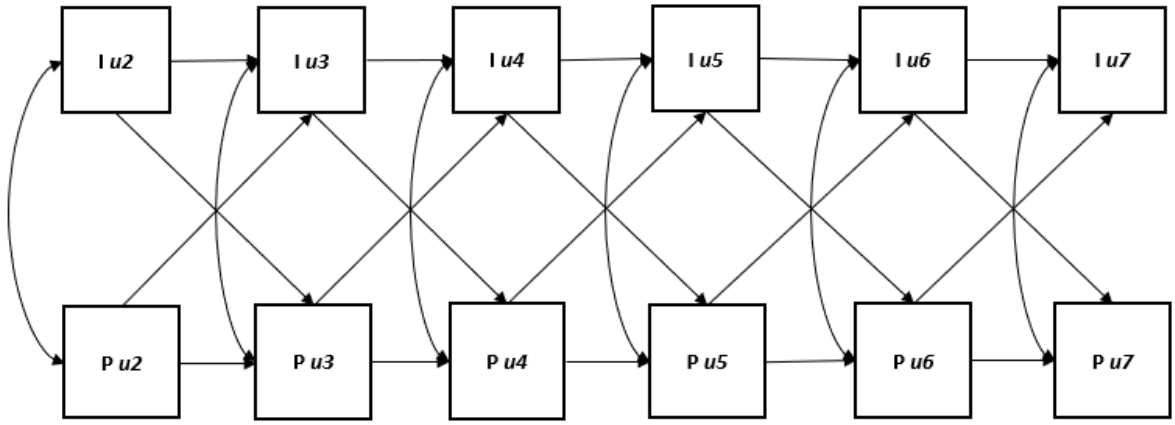
Data cleaning and analysis of descriptive statistics was performed using Stata version 17 (StataCorp, 2021).

MPlus (version 8.7; Muthén & Muthén, 1998–2021) was used to perform Multiple Imputation for missing covariate data (IQ at 7-years-old), and to run random-intercept cross lagged panel models (RI-CLPMs) to investigate the longitudinal stability of, and directional relationships between, the following variables, at the following timepoints:

1) Children's internalising symptoms and peer victimisation at ages 3, 5, 7, 11, 14 and 17.
2) Children's internalising symptoms and parental mental health at ages 3, 5, 7, 11, 14 and 17.

Cross-lagged panel models (CLPMs) are a form of Structural Equation Model used to test relationships in longitudinal data, where each case has observations of several variables recorded over more than one timepoint. This type of analysis can identify and describe directional relationships between variables over time (Sage, 2017), as well as allowing the identification of reciprocal relationships. The models are 'cross-lagged' because they estimate relationships from one variable to another, and vice versa ('cross'), and estimate relationships between variables across subsequent time points ('lagged'). CLPMs therefore estimate the directional influence variables have on each other over a period of time (Sage, 2017). The CLPM also includes auto-regressive paths controlling for the influence that a variable at say, timepoint 2, has on the same variable at timepoint 3. Additionally, covariance between the two variables in the CLPM is specified and controlled for within each timepoint. Figure 4 below illustrates this basic structure of a CLPM.

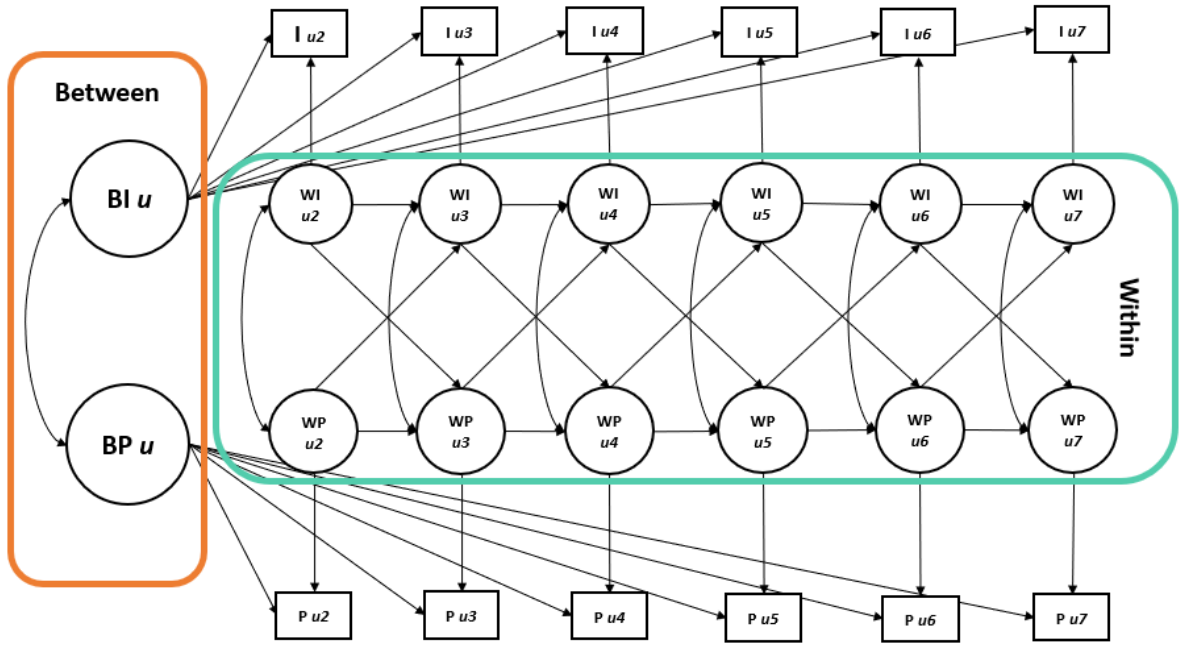
Figure 2



Six-wave Cross-Lagged Panel Model. In this example, Iut denotes the observed internalising symptoms, and Put denotes the parental mental health problems for individual u at timepoint t.

In 2015 Hamaker et al. proposed an extension of this model, in the form of the 'random- intercept' CLPM. This was in response to criticisms of the generic CLPM, in that the model could produce biased results when the concepts being studied included possible trait-like, between-person differences that are not sufficiently accounted for by the auto-regressive paths. The random-intercept CLPM (RI-CLPM) specifically aims to separate out this stable, between-person variation (accounting for long term trait-like differences between individuals) so that the 'lagged' associations in the CLPM represent only the within-person effect. Given that the present study aims to investigate internalising symptoms, parental mental health, and peer-victimisation, all of which may be subject to stable trait-like variation between individuals, RI-CLPMs have been used. The structure of these models is described below and illustrated by Figure 3.

Figure 3



Six-wave Random-Intercept Cross-Lag Panel Model. To illustrate the model, this is based on the relationships between autistic young person internalising symptoms and parental mental health only. Iut denotes the observed internalising symptoms, and Put denotes the parental mental health problems for individual u at timepoint t. W represents latent variables accounting for within-person variation, and B represents the random intercept latent variables accounting for between-person variation.

Two RI-CLPMs were used to investigate the two separate research questions in this study. Each RI-CLPM is constructed based on observed scores, in this case consisting of internalising symptoms and peer victimisation measured by the SDQ, and parental mental health measured by the K6. The observed scores are represented in the model by three components; 1) the grand means (the mean across all individuals at each time point), 2) a stable between-person component, and 3) a variable within-person component. The stable between-person component is represented in the model by random intercepts (BI*u* and BP*u*) – accounting for each person's difference from the grand means, and therefore separating out the effect of stable between-person variation in the data. In the RI-CLPM this is specified by creating a latent variable associated with the observed repeated measures, with all factor loadings fixed at 1. The final component of the observed scores in the RI-CLPM accounts for within-person variation, based on the difference between the observed score and expected score given the grand mean and random intercepts. In the RI-CLPM this is specified by

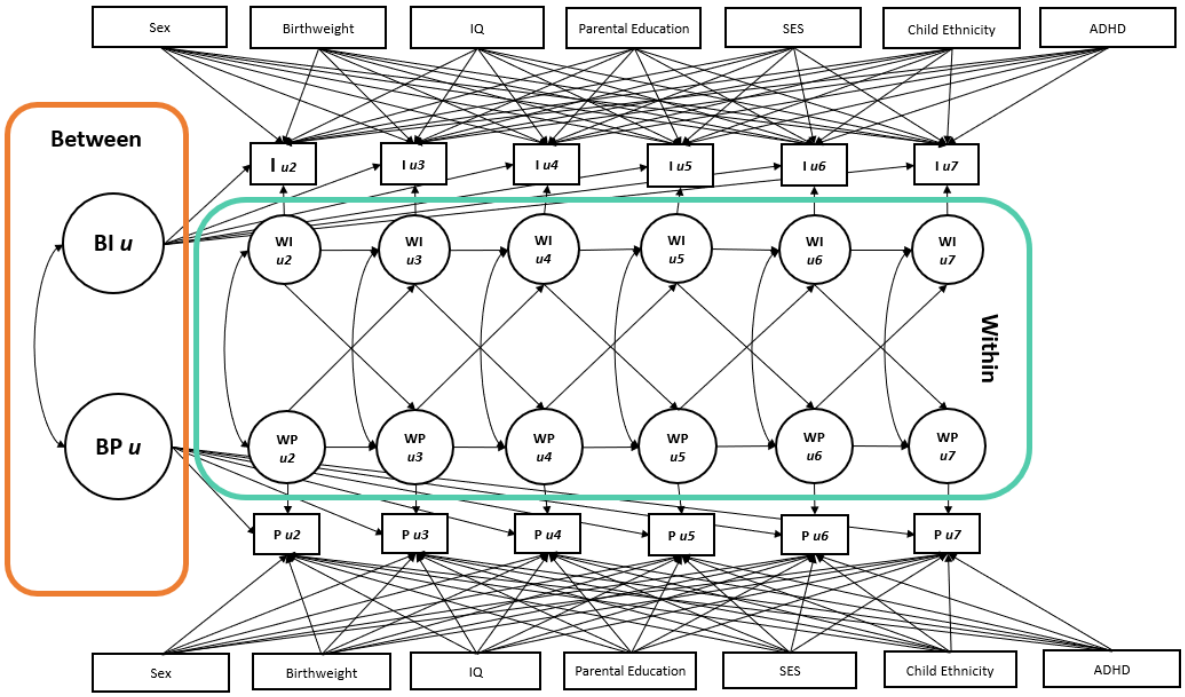
creating latent variables for each observed repeated measure (WI*u2*-*Wlu7* and WP*u2*-*WPu7*), and constraining measurement error variances to 0.

Next, structural relations in the CLPM are specified between the within-person latent variables. The auto-regressive effects (e.g. from WI*u2* to WI*u3*) represent the predictive relationship between, for example, a person's internalising symptoms at timepoint 2, to their internalising symptoms at the timepoint 3. The cross-lagged effects in the model therefore represent the predictive relationship between one variable on another. For example, the predictive effect of parental mental health at timepoint 3, on internalising symptoms at timepoint 4.

Finally, the model also includes the covariances between the within-person latent variables at each timepoint. In this RI-CLPM, the covariance between the random-intercepts (BI*u* and BP*u*) was not constrained, allowing it to be freely estimated. Allowing this to be freely estimated, rather than constrained at 0, is chosen in order to be most representative of the data, where we might expect that the stable, within-person component of internalising symptoms relates to the stable, within-person component of parental mental health.

As mentioned above, the RI-CLPMs were further extended to include time-invariant covariates related to the autistic children and their parents in sensitivity analyses. As shown in Figure 4 these covariates were specified in the CLPM to influence the observed variables directly, as opposed to being associated with the random-intercepts or latent within-person variables (Mulder & Hamaker, 2021). A limited set of covariates (child sex, IQ and ethnicity) were specified for the models presented in the main text, with the full set of covariates being specified in the RI-CLPMs in appendices 4 and 5.

Figure 4



Six-wave Random-Intercept Cross-lag Panel Model, including six covariates specified in the model. Iut denotes the observed internalising symptoms, and Put denotes the parental mental health problems for individual u at timepoint t. W represents latent variables accounting for within-person variation, and B represents the random intercept latent variables accounting for between-person variation

Model fit indices

Model fit indices are used to evaluate how well a structural equation model (in this case the RI-CLPMs) fits the sample data. In this study three indices will be used to report model fit; the Root Mean Square Error of Approximation (RMSEA); the Comparative Fit Index (CFI) and Standardised Root Mean Square Residual (SRMR). The RMSEA and SRMR are 'absolute fit indices', in that they assess how far a hypothesised model is from a 'perfect' model. Therefore, a value of zero would indicate a 'perfect' fit. A value of less than 0.08 is generally considered acceptable for the SRMR (Hu & Bentler, 1999). For the RMSEA, MacCallum et al. (1996) have suggested 0.01, 0.05 and 0.08 to indicate excellent, good and mediocre fit, respectively. The CFI, conversely, is an 'incremental fit index' that compares the fit of a hypothesized model with that of a baseline model (a model with the

worst fit). Values of the CFI range from 0-1, with values over 0.90 (ideally 0.95) indicating a good fit (Hu & Bentler, 1999).

RESULTS

Descriptive statistics

Descriptive statistics are presented in Table 5. The children were predominantly male (77.1%) and white (88.4%). This is in line with the male-to-female ratio in high-quality autism prevalence studies (Loomes et al., 2017), and with the ethnic composition of the UK population at the time of recruitment to the MCS (UK Census, Office of National Statistics, 2001).

Table 5

Demographic variable		N (Percentage)
Sex	Male	432 (77.1%)
	Female	128 (22.9%)
ADHD	Parents indicated a stable diagnosis at age 5yo, 7yo, 11yo or 14yo	169 (30.2%)
	No stable ADHD indication	391 (69.8%)
Ethnicity	White	495 (88.4%)
	Mixed	19 (3.4%)
	Indian	3 (0.5%)
	Pakistani and Bangladeshi	14 (2.5%)
	Black	17 (3.0%)
	Other Ethnic group	8 (1.4%)
	Missing ethnicity	4 (0.7%)
SES (Family income)	£ 0 - £31,199	431 (77.0%)
	£31,200 and greater	87 (15.5%)
	Missing income information	42 (7.5%)
Parental Education	NVQ levels 1-2 (GCSE level)	228 (40.7%)
	NVQ levels 3, 4, 5 (A-level - higher education)	237 (42.3%)
	Missing education information	95 (17.0%)

Demographic variable	(N)	Mean, SD Range
Birthweight (kg)	555	3.35 ± 0.67, 0.62 – 5.73
IQ (age 7)	388	92.5 ± 17.47, 41.18 – 133.91

Descriptive statistics of sample

Table 6 provides means, standard deviations and response rates for peer victimisation, parental mental health and internalising symptoms at each sweep. The table also includes the percentage of individuals scoring in the clinical range for internalising symptoms or parental mental health, and the percentage of parents responding 'Certainly True' to their child being the victim of peer victimisation. Mean scores across all measures show a general upward trend over time, although

there is some stabilisation of internalising symptoms from 11-years-old, whilst mean parental mental health appears to worsen sharply at sweep 7 (child aged 17-years-old). These trends are shown in Figure 7.

Table 6

Variable		Time 2 (3yo)	Time 3 (5yo)	Time 4 (7yo)	Time 5 (11yo)	Time 6 (14yo)	Time 7 (17yo)
Internalising symptoms	*(N)*	*477*	*506*	*480*	*474*	*431*	*346*
	Mean, SD	1.67 ± 1.73	2.27 ± 2.08	3.07 ± 2.42	4.30 ± 2.66	4.29 ± 2.63	4.18 ± 2.79
% scoring within clinical range		6.92	15.61	29.37	46.41	45.94	44.51
Peer Victimisation	*(N)*	*448*	*472*	*442*	*475*	*423*	*296*
	Mean, SD	0.13 ± 0.39	0.39 ± 0.61	0.58 ± 0.68	0.86 ± 0.75	0.82 ± 0.74	0.69 ± 0.72
% reporting peer victimisation		2.01	6.78	11.09	22.11	19.86	14.86
Parental Mental Health	*(N)*	*448*	*498*	*471*	*469*	*422*	*336*
	Mean, SD	4.58 ± 4.81	4.52 ± 4.73	5.03 ± 4.97	6.05 ± 5.35	6.12 ± 4.75	8.87 ± 5.25
% scoring within clinical range		8.04	7.63	9.13	11.51	11.85	21.73

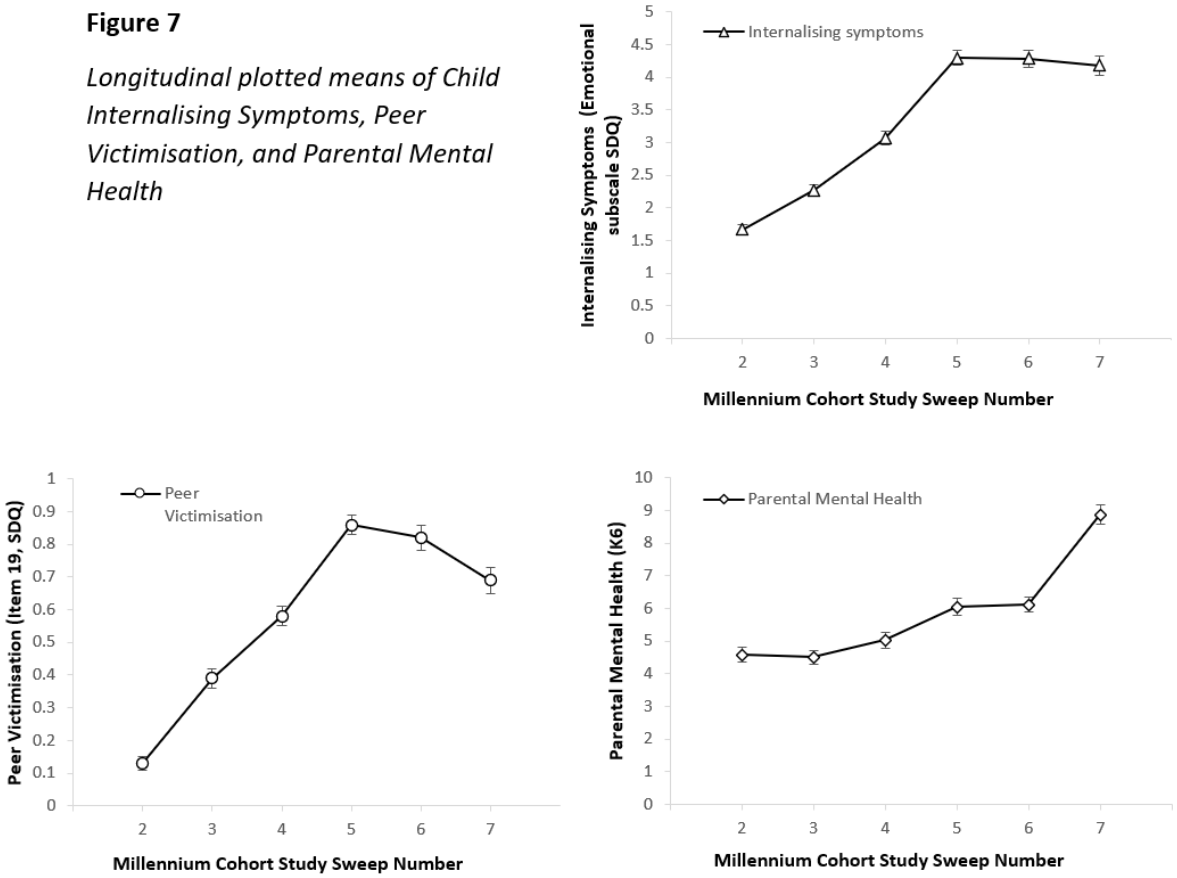
Descriptive statistics for Internalising symptoms, Peer Victimisation and Parental Mental Health over 6 sweeps. Internalising symptom scores are SDQ emotional sub-scale (range 0 - 10), Peer victimisation scores are SDQ item 19 (range 0 - 2), Parental mental health scores are K6 (range 0 – 24). In all cases higher scores indicate greater severity.

As shown in Table 6, the percentage of children with internalising symptoms in the clinical range increases from a small percentage (6.92%, Sweep 2) to its highest level at just under half the sample (46.41%, Sweep 5), stabilising thereafter. The percentage of parents with clinically significant mental health symptoms remains at a small percentage of the sample (approximately 7-11%), until sharply rising to 21.73% by sweep 7. For peer victimisation, the frequency of children reported as having 'certainly' experienced bullying mirrored the trend of internalising symptoms, increasing throughout

childhood until 11-years-old (22.11%, sweep 5), thereafter stabilising and decreasing slightly to 14.86% at sweep 7.

Figure 7

Longitudinal plotted means of Child Internalising Symptoms, Peer Victimisation, and Parental Mental Health



Longitudinal plotted means of parent-reported child Internalising symptoms, Parental Mental Health and Peer Victimisation over 6 sweeps. Internalising symptom scores are SDQ emotional sub-scale (range 0 - 10), Parental Mental Health scores are K6 (range 0-24) and Peer Victimisation scores are item 19, SDQ (range 0-2). In all cases, higher scores indicate greater severity. Standard error bars are given.

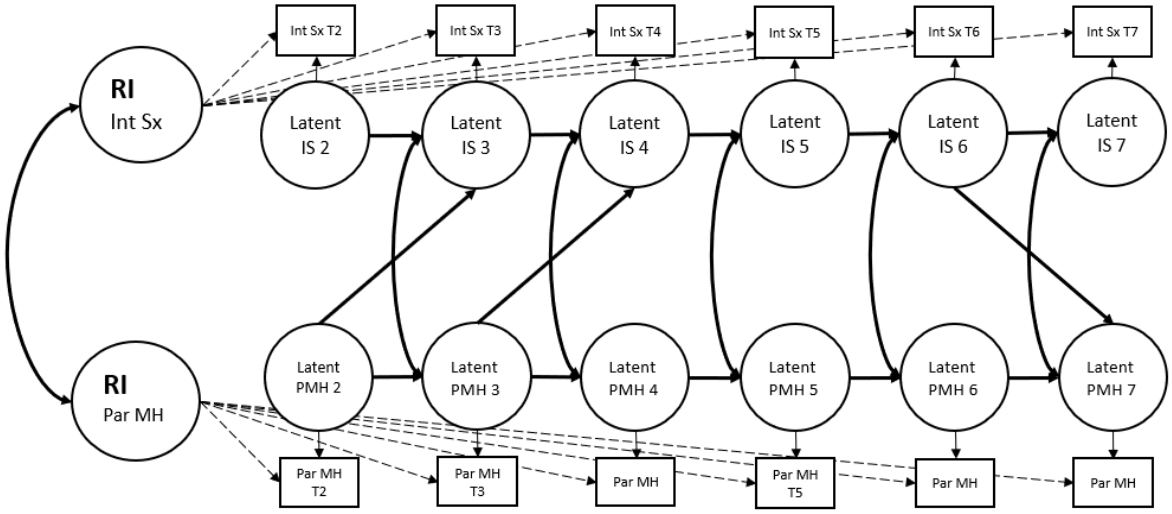
RI-CLPMs are presented below for the longitudinal relationships between internalising symptoms and parental mental health, as well as peer victimisation. In each case these are presented first without covariates, and then with ethnicity, sex and IQ included in the model as covariates. RI-CLPMs with the full set of covariates specified are presented in appendices 4 and 5.

Results of RI-CLPM between Internalising symptoms and Parental Mental Health

A RI-CLPM was constructed to model the longitudinal relationships between child internalising symptoms and parental mental health across sweeps 2 to 7 (Figure 8). All auto-regressive paths in

the model were statistically significant, indicating that internalising symptoms or parental mental health difficulties at any one timepoint had strong predictive power over the individual's internalising symptoms or parental mental health at the next timepoint. Other than at sweep 2, internalising symptoms and parental mental health were significantly related within each sweep. The random intercepts representing trait-like between-person variation in internalising symptoms and parental mental health were significantly related ($p < 0.001$). Additionally, three statistically significant cross-lagged regressive paths were found, with worsened parental mental health at sweeps 2 and 3, contributing to worsened child internalising symptoms at sweeps 3 and 4. Conversely, from sweep 6 to 7, worsened child internalising symptoms were associated with worsened parental mental health. All autoregressive and cross-lagged regression paths are shown in Table 7. Model fit was within acceptable bounds (RMSEA = 0.068, CFI = 0.940, SRMR = 0.078).

Figure 8



Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), without covariates. Statistically significant regressive and covariance paths are shown in bold in the model.

Table 7

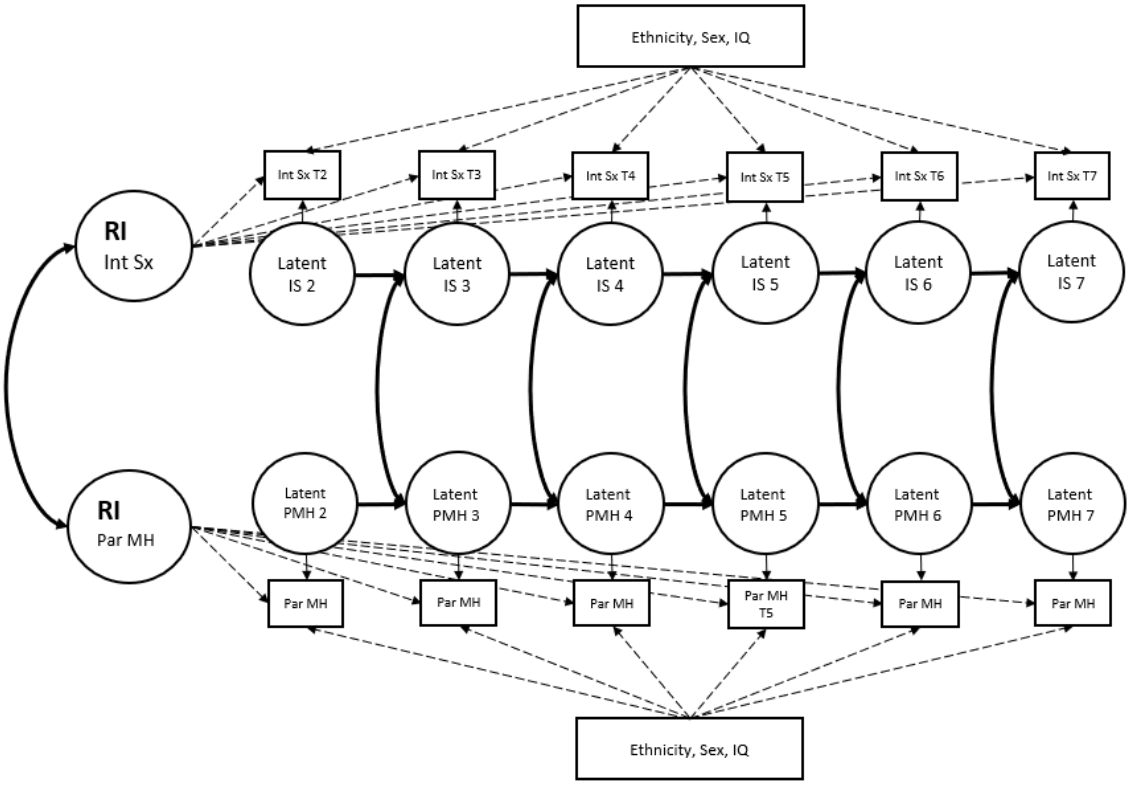
	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.368**	**<0.001**
	Par MH 2	**Par MH 3**	**0.380**	**0.001**
	Int Sx 3	**Int Sx 4**	**0.445**	**<0.001**
	Par MH 3	**Par MH 4**	**0.328**	**0.005**
	Int Sx 4	**Int Sx 5**	**0.486**	**<0.001**
	Par MH 4	**Par MH 5**	**0.451**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.461**	**<0.001**
	Par MH 5	**Par MH 6**	**0.434**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.573**	**<0.001**
	Par MH 6	**Par MH 7**	**-0.247**	**0.032**
Cross-lagged	**Par MH 2**	**Int Sx 3**	**0.076**	**0.041**
	Int Sx 2	Par MH 3	-0.010	0.959
	Par MH 3	**Int Sx 4**	**0.068**	**0.051** see note
	Int Sx 3	Par MH 4	-0.068	0.684
	Par MH 4	Int Sx 5	-0.016	0.676
	Int Sx 4	Par MH 5	0.041	0.766
	Par MH 5	Int Sx 6	0.014	0.601
	Int Sx 5	Par MH 6	0.118	0.159
	Par MH 6	Int Sx 7	-0.031	0.432
	Int Sx 6	**Par MH 7**	**0.355**	**0.011**

Tabulated results of Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6). No covariates included. Statistically significant relationships are highlighted in bold. Note: the association between Par MH 3 and Int Sx 4 is just over the conventional threshold of significance of $p < 0.05$

Results of RI-CLPM between Internalising symptoms and Parental Mental Health with Ethnicity, Sex and IQ as covariates

The RI-CLPM in Figure 8 above was further extended to include the child's ethnicity, sex and IQ as covariates (Figure 9). All auto-regressive paths in the model remained statistically significant, as did the covariance between random-intercepts and covariances between variables at each sweep (excepting for sweep 2). The inclusion of the covariates however has weakened the cross-lagged associations, with no statistically significant cross-lagged associations remaining between internalising symptoms and parental mental health. All autoregressive and cross-lagged regression paths are shown in Table 8. The model fit improved slightly with the addition of covariates (RMSEA = 0.068, CFI = 0.945, SRMR = 0.065).

Figure 9



Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), with all covariates included. Statistically significant regressive and covariance paths are shown in bold in the model. For simplicity, covariates are shown as combined in this model.

Table 8

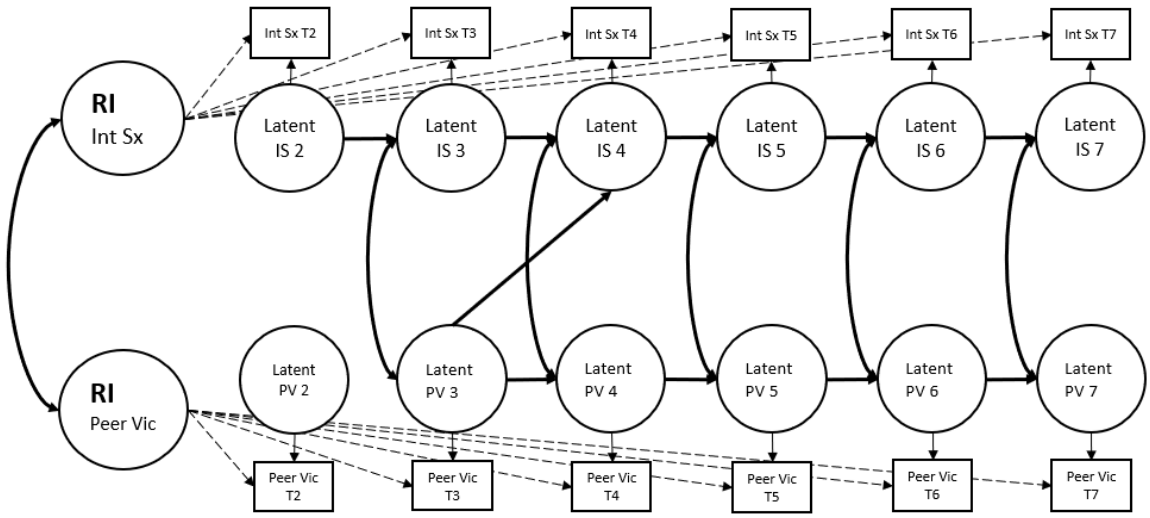
	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.294**	**<0.001**
	Par MH 2	**Par MH 3**	**0.359**	**0.001**
	Int Sx 3	**Int Sx 4**	**0.359**	**<0.001**
	Par MH 3	**Par MH 4**	**0.307**	**0.009**
	Int Sx 4	**Int Sx 5**	**0.447**	**<0.001**
	Par MH 4	**Par MH 5**	**0.402**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.502**	**<0.001**
	Par MH 5	**Par MH 6**	**0.536**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.502**	**<0.001**
	Par MH 6	**Par MH 7**	**-0.191**	**0.017**
Cross-lagged	Par MH 2	Int Sx 3	0.149	0.078
	Int Sx 2	Par MH 3	-0.078	0.659
	Par MH 3	Int Sx 4	0.103	0.100
	Int Sx 3	Par MH 4	-0.096	0.576
	Par MH 4	Int Sx 5	-0.018	0.637
	Int Sx 4	Par MH 5	0.022	0.873
	Par MH 5	Int Sx 6	0.015	0.583
	Int Sx 5	Par MH 6	0.098	0.246
	Par MH 6	Int Sx 7	-0.037	0.348
	Int Sx 6	Par MH 7	0.121	0.067

Tabulated results of Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6). All covariates included. Statistically significant relationships are highlighted in bold.

Results of RI-CLPM between Internalising symptoms and Peer Victimisation

A second RI-CLPM was constructed to model the longitudinal relationships between child internalising symptoms and peer victimisation across sweeps 2 to 7 (Figure 10). Other than peer victimisation between sweeps 2 and 3, all auto-regressive paths in the model were statistically significant, suggesting stable longitudinal trajectories for child internalising symptoms and peer victimisation. Other than at sweep 2, internalising symptoms and peer victimisation were significantly related within each sweep. The random intercepts representing between-person variation in internalising symptoms and peer victimisation were also significantly related ($p < 0.001$). Additionally, one statistically significant cross-lagged regressive path was found, with worsened peer victimisation at sweep 2 contributing to worsened child internalising symptoms at sweep 3. Autoregressive and cross-lagged regression paths are shown in Table 9. Model fit was found to be good (RMSEA = 0.031, CFI = 0.984, SRMR = 0.041).

Figure 10



Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), without covariates. Statistically significant regressive and covariance paths are shown in bold in the model.

Table 9

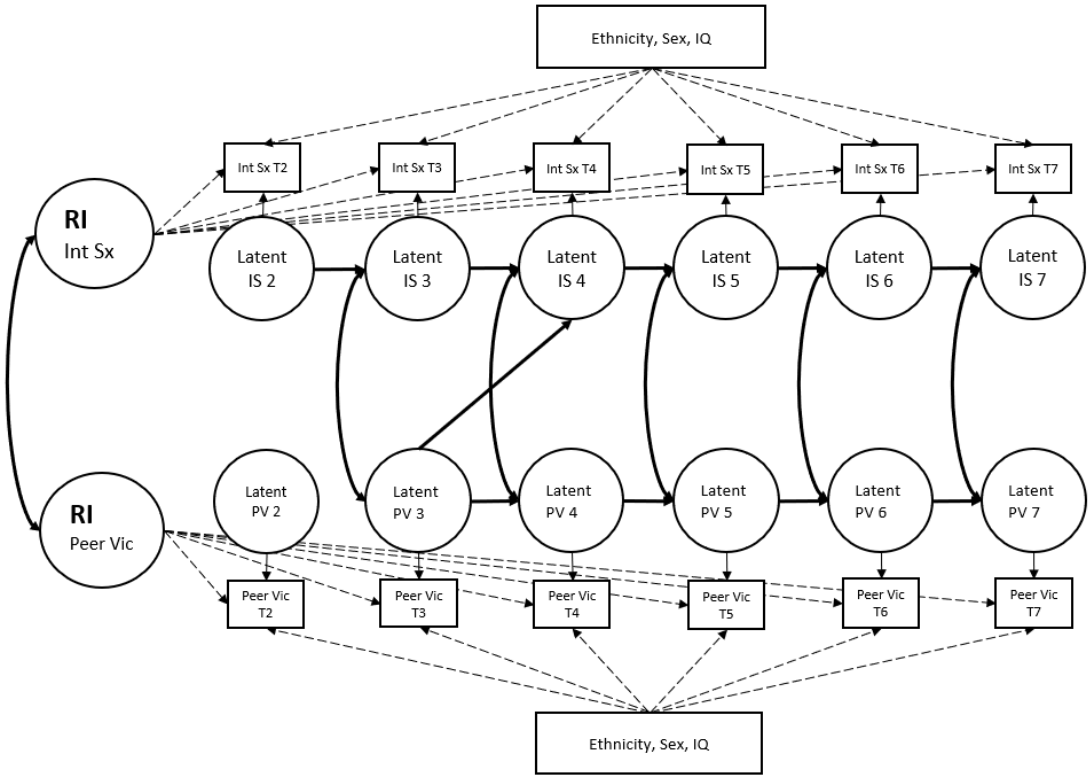
	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.378**	**<0.001**
	Peer Vict 2	Peer Vict 3	0.010	0.938
	Int Sx 3	**Int Sx 4**	**0.439**	**<0.001**
	Peer Vict 3	**Peer Vict 4**	**0.336**	**<0.001**
	Int Sx 4	**Int Sx 5**	**0.468**	**<0.001**
	Peer Vict 4	**Peer Vict 5**	**0.315**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.482**	**<0.001**
	Peer Vict 5	**Peer Vict 6**	**0.419**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.567**	**<0.001**
	Peer Vict 6	**Peer Vict 7**	**0.454**	**<0.001**
Cross-lagged	Peer Vict 2	Int Sx 3	0.166	0.614
	Int Sx 2	Peer Vict 3	0.017	0.607
	Peer Vict 3	**Int Sx 4**	**0.491**	**0.014**
	Int Sx 3	Peer Vict 4	0.010	0.652
	Peer Vict 4	Int Sx 5	0.217	0.242
	Int Sx 4	Peer Vict 5	-0.014	0.487
	Peer Vict 5	Int Sx 6	-0.085	0.635
	Int Sx 5	Peer Vict 6	0.019	0.212
	Peer Vict 6	Int Sx 7	0.038	0.853
	Int Sx 6	Peer Vict 7	0.019	0.292

Tabulated results of Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ), without covariates. Statistically significant relationships are highlighted in bold.

Results of RI-CLPM between Internalising symptoms and Peer Victimisation with Ethnicity, Sex and IQ as covariates

The RI-CLPM in Figure 10 above was further extended to include the child's ethnicity, sex and IQ as covariates (Figure 11). The previous auto-regressive paths in the model remained statistically significant, as did the covariance between random-intercepts and covariances between variables at each sweep (excepting for sweep 2). The cross-lagged regression path between sweep 2 and 3 also remained statistically significant with the inclusion of the covariates. All autoregressive and cross-lagged regression paths are shown in Table 10. The model fit remained largely unchanged (RMSEA = 0.036, CFI = 0.981, SRMR = 0.037).

Figure 11



Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ), with covariates included. Statistically significant regressive and covariance paths are shown in bold in the model. For simplicity, covariates are shown as combined in this model.

Table 10

	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.369**	**<0.001**
	Peer Vict 2	Peer Vict 3	-0.013	0.920
	Int Sx 3	**Int Sx 4**	**0.429**	**<0.001**
	Peer Vict 3	**Peer Vict 4**	**0.339**	**<0.001**
	Int Sx 4	**Int Sx 5**	**0.443**	**<0.001**
	Peer Vict 4	**Peer Vict 5**	**0.304**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.462**	**<0.001**
	Peer Vict 5	**Peer Vict 6**	**0.406**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.540**	**<0.001**
	Peer Vict 6	**Peer Vict 7**	**0.456**	**<0.001**
Cross-lagged	Peer Vict 2	Int Sx 3	0.092	0.787
	Int Sx 2	Peer Vict 3	0.009	0.782
	Peer Vict 3	**Int Sx 4**	**0.461**	**0.021**
	Int Sx 3	Peer Vict 4	0.010	0.660
	Peer Vict 4	Int Sx 5	0.200	0.275
	Int Sx 4	Peer Vict 5	-0.016	0.413
	Peer Vict 5	Int Sx 6	-0.115	0.517
	Int Sx 5	Peer Vict 6	0.016	0.294
	Peer Vict 6	Int Sx 7	0.042	0.838
	Int Sx 6	Peer Vict 7	0.014	0.414

Tabulated results of Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ), including all covariates. Statistically significant relationships are highlighted in bold.

Further results of RI-CLPMs performed with a fuller set of covariates are presented in appendices 4 and 5. The statistically significant covariance and regressive paths are unchanged from the RI-CLPMs with covariates presented in this section.

Additionally, descriptive statistics, means, standard deviations and response rates for each variable, and results of RI-CLPMs are presented for the alternative, more restrictively defined, autism sample in appendices 6, 7 and 8. Descriptive statistics of the sample and trajectory of variables over time (means, response rates) do not differ meaningfully from that of the analytic sample above. Whilst the covariance associations and longitudinal auto-regressive paths within the models are unchanged from the RI-CLPMs of the analytic sample, one cross-lagged regression path was lost from each RI-CLPM.

DISCUSSION

In order to better understand the mechanisms driving internalising symptoms in autistic children, we examined the bidirectional relationships between autistic children's internalising symptoms and parental mental health, and between internalising symptoms and peer victimisation. Children in our sample experienced internalising symptoms that increased with age, peaking around entrance to secondary school (age 11, sweep 5) before stabilising. Parental mental health showed a more slowly increasing trajectory, with a sharp increase as children moved into late adolescence (age 17, sweep 7). Peer victimisation was also reported by parents at increasing rates as children aged, peaking alongside internalising symptoms as children entered secondary school (age 11, sweep 5), with over a fifth of children reporting bullying at this time point.

Whilst the plots of descriptive statistics for the whole sample revealed changeable trajectories over time, all four RI-CLPMs identified within-person rank stability in the form of statistically significant auto-regressive paths. This was true for internalising symptoms and parental mental health (sweeps 2-7), as well as peer victimisation (sweeps 3-7). This suggests that for a given individual, the severity

of their internalising symptoms, parental mental health, or peer victimisation at any age predicted the severity across these concepts at later timepoints. Therefore, a child who was experiencing, for example, greater internalising symptoms at 7-years-old, would continue to be more likely to experience these difficulties at older ages.

We also found statistically significant covariance associations between the random-intercepts in each of the four RI-CLPMs. This suggests that the stable, trait-like components of these constructs (such as internalising symptoms and peer victimisation) are strongly interrelated, in addition to the association of the constructs at each cross-sectional point in time.

Aim: To explore the longitudinal relationship between parental mental health and internalising symptoms in children with autism

Using RI-CLPMs, we identified bidirectional relationships for the within-person component of children's internalising symptoms and parental mental health, with greater parental mental health difficulties when their child was 3- and 5-years-old, contributing to worsened internalising symptoms when the child was aged 5- and 7-years-old. This relationship was reversed in older adolescence, where child internalising symptoms at age 14 were positively associated with their parent's mental health difficulties at age 17. The early prediction of child internalising symptoms from parental mental health is in line with previous research findings in typically-developing samples (Mattejat et al., 2008; Bayer et al., 2011). The later directional change of adolescent internalising symptoms predicting parental mental health could be understood in a number of ways, including in the context of times of transition for the child, increasing independence, or changing needs for parenting support. Despite these hypotheses, this association appears to be a novel finding in the literature for a sample of autistic young people. Interestingly, one recent study of a typically-developing sample within the MCS found a similar bi-directional relationship between child internalising symptoms and maternal distress, including the prediction of maternal distress at age 17 from child internalising

symptoms at age 14 (Speyer et al., 2022). This shared pattern suggests that this relationship may represent a general effect of adolescence or transition rather than an autism-specific effect.

In the subsequent RI-CLPMs extended by the addition of covariates (both limited set, and full set presented in appendix), the cross-lagged associations were no longer identified. It therefore appears that the child's sex, IQ and ethnicity may impact on the relationship between parental mental health and child internalising symptoms over time. Drawing on existing research, this may be due to sex-differences in the expression and prevalence of emotional distress for autistic children (Holtmann et al., 2007; Mandy et al. 2012; Solomon et al., 2012), or the impact of IQ on the interaction between parental mental health and internalising symptoms. IQ (encompassing possible learning disability) may explain some of this relationship due to differences in parental involvement, parental stressors and difficulties of transition throughout the study period (Benderix et al., 2007; Al-Yagon, 2014). There may also be differences in the parent-child transmission of mental health difficulties in relation to ethnicity, with some tentative research findings suggesting that this may reflect cultural differences, differing parenting roles or ratings of distress in BAME groups (Goodman et al., 2008; Turner et al., 2015; Yeh et al., 2005). In addition, parents from BAME groups may be subject to additional stressors such as discrimination or acculturation which may contribute to their ratings of distress (Eisenhower & Blacher, 2006).

Within timepoints, both RI-CLPMs consistently found significant associations between internalising symptoms and parental mental health (sweeps 3-7), suggesting that the severity of these two concepts remains related cross-sectionally, even when longitudinal auto-regressive effects and between-person, stable, trait-like differences are controlled for by the model. The between-person, stable, trait-like components of child internalising symptoms and parental mental health were also found to be significantly, positively associated.

Aim: To explore the longitudinal relationship between peer victimisation and internalising symptoms in children with autism

RI-CLPMs of internalising symptoms and peer-victimisation identified one cross-lagged effect, with peer victimisation at 5-years-old predicting internalising symptoms at 7-years-old. This effect remained statistically significant even when covariates (limited or full set) were controlled for in the model. This effect is perhaps surprising at such a young age, especially as both peer victimisation and internalising symptoms peaked later, at entry to secondary school (11-years-old, sweep 5). This cross-lagged effect appears to be identifying what may be children's first exposure to peer victimisation (on entry to primary school or formal education) at 5-years-old, which subsequently predicts the development of internalising symptoms at the next sweep (7-years-old). Previous research has identified widespread bullying in population-samples of children of a similar age: by the age of 6-years-old ((24.3% reporting 'very frequent' bullying) Wolke et al., 2001) or 7-years-old ((48.6% reporting have ever been bullied, 9% reporting daily – weekly bullying) Campbell et al., 2019). It should be noted that these rates are higher than those identified in the present study, despite expecting higher rates of bullying in an autistic population (Maiano et al., 2016). One factor for this may be that both of the referenced studies utilised child self-report to collect bullying prevalence (individual interviews – Wolke et al., 2001; and self-report SDQ interviews – Campbell et al., 2019), whereas the present study uses parent self-report which may be under-identifying peer victimisation.

The model did not meaningfully differ when covariates (limited or full set) were controlled for, suggesting that a child's sex, IQ, ethnicity (or in full set; ADHD diagnosis, SES, parental education, or low birth weight) did not meaningfully impact upon the relationship between peer victimisation and internalising symptoms for this sample. This is clinically meaningful in that identification of individuals, preventative measures and/or intervention to reduce internalising symptoms in autistic children may not require targeting based on these demographic characteristics.

As with parental mental health, covariance within timepoints was statistically significant (sweeps 3-7), indicating that internalising symptoms and peer victimisation are cross-sectionally related, even when auto-regressive effects and stable, between-person trait-like differences have been controlled for. Between-person, trait-like differences in internalising symptoms and peer victimisation were also found to be significantly, positively related.

Sensitivity analyses

Sensitivity analyses were conducted for a more restrictively-defined autism sample (N = 415), see appendices 7 and 8 for the further RI-CLPMS. Whilst the statistically significant cross-sectional associations and auto-regressive paths remained unchanged, one cross-lagged regression path was lost from each model. In the sensitivity analyses, a predictive path was identified from parental mental health at child age 5-years-old to child internalising symptoms at 7-years-old, and from internalising symptoms at 14-years-old to parental mental health at 17-years-old, however the regression path from parental mental health at 3-years-old to internalising symptoms at 5-years-old no longer met statistical significance. Equally, in the RI-CLPM of internalising symptoms and peer victimisation, the one previous cross-lagged path from peer victimisation at 5-years-old to internalising symptoms at 7-years-old no longer met significance. Nevertheless, the coefficients remain of a similar magnitude and directionality. These results appear to be best explained by the loss of power given that the sample has now been reduce from 560 to 416 children.

Clinical implications

The RI-CLPMs presented in this study report on risk factors known to relate to children's internalising symptoms in typically developing samples (Reijntjes et al., 2010; Bayer et al., 2011; Fitzimons et al., 2017). Within this sample of autistic children, stable within-person rank-stability was identified for peer victimisation, parental mental health and internalising symptom. Persistent cross-sectional relationships were also found at each sweep between child internalising symptoms and parental mental health, or peer victimisation respectively. This indicates the importance of these factors in

understanding the development and stability of internalising symptoms during the childhood and adolescence of autistic children. Using the conceptualisation of the Vulnerability, Scar and Reciprocal Risk model discussed in the introduction (Figure 1), the results from the present study support the 'vulnerability model' as a way of understanding the relationships between peer victimisation and child internalising symptoms, in which the impact of peer victimisation at 5-years-old bestows a vulnerability for emotional difficulties at 7-years-old. Conversely, the relationships between parental mental health and internalising symptoms (in the uncontrolled RI-CLPM) appear to form a 'reciprocal risk' model, with both parental mental health predicting later child internalising difficulties, and vice versa, at differing points in childhood-adolescence.

It is important to note that internalising symptoms are known to be more prevalent in samples of autistic children than typically-developing children (Lai et al., 2019) so it remains to be considered whether these shared risk factors (parental mental health, peer victimisation), have a greater effect on children with autism (such as an increased susceptibility to the impact of these risk factors), or whether there are additional autism-specific factors (either individual or environmental) which explain the greater prevalence of difficulties.

Both of the factors considered in this study are potentially modifiable, and may be amenable to change via a variety of levels of influence. Parental mental health for example, is already prioritised in the perinatal period (NICE, 2014), however in the uncontrolled model in this study we found parental mental health was associated with child internalising symptoms at 3-years-old and 5-years-old, which is well outside of the typically-defined perinatal period (pregnancy to 12-months-old; NICE, 2014). This result therefore suggests that identification of parental mental health difficulties would be a valuable target for intervention within the child's first 5 years of life, as an attempt to reduce the risk of childhood internalising symptoms in mid-childhood. It is possible that this is an autism-specific difference compared with parents of typically-developing children. Interestingly, in later adolescence, the uncontrolled RI-CLPM revealed a potential risk of adolescent internalising

difficulties contributing to parental mental health problems. This raises the consideration of screening for parental mental health in adolescence, pro-actively offering preventative interventions for parents of children with emotional difficulties, and increased awareness of parent / carer wellbeing and coping when young autistic people are referred to psychology services.

In the case of peer victimisation, the relationships revealed by the RI-CLPMs were not altered by the addition of covariates, indicating that stratifying by demographic variables to target individuals for intervention of peer victimisation is unlikely to be helpful for the demographic characteristics considered in this study, including sex, ethnicity or IQ. A bidirectional relationship was not found for peer victimisation and internalising symptoms, with only peer victimisation (at 5-years-old) predicting internalising symptoms (at 7-years-old). This is a relatively young age, within the first year of primary school, and therefore suggests early identification of bullying in home, school and social environments is likely to be very important. Interventions to reduce the risk of bullying should be considered, as well as screening for subsequent emotional difficulties.

Limitations and further research

There are a number of limitations to consider in the interpretation of these results. One limitation is that all measures were reported by parents on behalf of their child with autism. This methodology was chosen to allow inclusions of early sweeps (such as at 3- and 5-years-old), and subsequently to ensure consistency of measures over time. Additionally, it allows for inclusion of children with learning disability and / or communication difficulties, who may not have been able to self-report these measures. It is possible however, that parents did not have insight into all internalising difficulties or peer victimisation, or indeed that they may have over-estimated their child's difficulties (López-Pérez & Wilson, 2015; Van der Meer et al., 2008). Additionally, it is possible that associations identified in the models may have been inflated by the effects of common method variance, given that all three key measures were parent-reported.

The use of the parent-reported SDQ item 19 as a measure of peer victimisation is also a limitation for our study, as this is one-item response is not a validated measure of peer victimisation, and does not explicitly capture bullying in multiple contexts (such as sibling bullying at home, *versus* at school, or cyber-bullying). Additionally, it captures only a general measure of bullying, without providing prompts to cover categories of victimisation such as verbal, physical, theft, amongst others. As a result, rates of peer victimisation may be under-estimated by this single-item measure.

An additional limitation is the drop-out of participants or inconsistent measure-completion within the MCS, which, as shown in Table 6, increases over time (maximum measure completion at 85% of sample at sweep 2, reducing to maximum measure completion of 62% at sweep 7). It is possible that this drop-out or non-completion of measures was non-random, and resulted in a set of remaining children that are not representative of the sample at large.

Finally, an additional limitation of this study, using measures that have been collected within the MCS, is that none of the key measures are autism-specific, that is, enquire about difficulties in internalising symptoms, parental mental health or peer victimisation that may be directly related to the characteristic set of difficulties experienced by people with an autism diagnosis (social communication difficulties, sensory sensitivity, restricted and repetitive behaviours and highly restricted interests (American Psychiatric Association, 2013)). This study aimed to understand the impact of two possible external risk factors and associated bi-directional relationships with internalising symptoms, however it is also possible that there are more important autism-specific risk factors which interact with the autism-related difficulties described above. These may for example include exposure to environments that are a poor fit for autistic people (Lai et al., 2020; Mandy, 2022), sensory sensitivity (Costley et al., 2021; Corbett et al., 2009; Hwang et al., 2019; Neil et al., 2016), or difficulties conforming to norms of social communication (Acker et al., 2018; Costley et al., 2021). Whilst it was not possible to study these effects using the pre-collected measures within the data-rich MCS sample, further research in this area would be very important to

understand how the phenomenology and risk factors of typically-developing and autistic children may differ. In turn, this may open the door to more specific strategies for preventing or alleviating emotional difficulties for children and young people with autism.

CONCLUSION

In conclusion, two potential risk factors were found to have important associations with internalising symptoms experienced by children and young people with autism. This was in the form of a vulnerability model (peer victimisation conferring vulnerability to later internalising symptoms) and a reciprocal risk model (parental mental health predicting early child internalising symptoms, and adolescent internalising symptoms predicting later parental mental health difficulties). These associations indicate a shared aetiology of internalising symptoms in autistic children and typically-developing children based on existing research, however further research is needed to understand whether there are further autism-specific risk factors that explain the increased prevalence of internalising symptoms in this group. One gap in the present research literature is for longitudinal studies using autism-specific measures, in order to assess what impact these may have on the development of internalising symptoms across childhood and adolescence, given there remains a greater prevalence of internalising symptoms in autistic children and young people. Longitudinal analytic models such as RI-CLPMs are well suited to answer these questions.

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Part 3

Critical Appraisal

CRITICAL APPRAISAL

Introduction

The following critical appraisal is a reflection on the process of conducting the research outlined in parts one and two of this thesis. I have chosen to focus my reflections on three key themes. Firstly, I discuss the implications of conducting research focussed on autism spectrum disorder, and the interactions between research, lived experience and advocacy in this area. Secondly, I reflect on the opportunities and limitations afforded by using secondary data, especially in the use of longitudinal cohort studies. Finally, I discuss the use of random-intercept cross-lagged panel models for drawing clinical interpretations.

Being a non-autistic researcher within the field of autism research

During the planning and conducting of research for this thesis, I have moved from a position of relatively 'naive' intellectual curiosity about the field of autism research, to a position acknowledging the complex interaction of stakeholders within the field, who hold a variety of perspectives including lived experience of autism, parenting or caring for an autistic young person, clinicians meeting autistic young people in routine clinical practice, and researchers specialising in the study of the condition. In the earlier stages of research, especially positioned as a 'Trainee Clinical Psychologist', I did not conceptualise myself as a researcher whose work may have wider implications. Having now had the opportunity to immerse myself in the field of autism research, which is developing and re-shaping thanks to the voices of autism-community advocates (den Houting, 2019; Kapp, 2020), I have seen that taking an uncritical stance on autism research has the potential to perpetuate unhelpful, or even harmful, narratives for autistic people and their families. Examples of these research narratives or constructs have been publicised by autism-community advocates and researchers, such as the history of 'categorising' people with autism as 'high' *vs.* 'low' functioning (Alvares et al., 2020), prior narrow definitions of autism which under-recognised female presentations of autism (Wijngaarden-Cremers et al., 2014; Hull & Mandy, 2017), or interventions

focused on supporting autistic people to conform to environments designed for neurotypical people (Milton, 2014; Kirkham, 2017) amongst others.

I have therefore gained a greater awareness of my position as a non-autistic researcher, and the potential power and influence that I may hold given my ability to conduct and disseminate the research presented in this thesis, despite having no personal experience of being autistic. As a result, I felt a responsibility to hold existing hypotheses around medical or disorder-specific models of autism lightly, and consider how my research findings may ultimately be useful to autistic people. This felt especially important given that my research was conducted *via* systematic review and secondary data, so I was not able to meet any of the autistic children, young people or families that I write about in this thesis.

I have attempted to frame my research from the perspective of neurodiversity (Singer, 1998) – where autism is seen as one form of neurodevelopment within a diverse variety of minds. Taking the perspective of neurodiversity, 'disability' is considered as arising when the environment is a poor fit for the physical, cognitive or emotional characteristics of an individual (den Houting, 2018). The individual is therefore considered to be 'disabled by their environment', rather than as a result of being autistic.

Whilst neither the systematic review or empirical paper in this thesis investigated constructs that are autism-specific, internalising symptoms (experienced broadly across the population) are known to be more prevalent amongst autistic children, young people (Lai et al., 2019). Attempting to work from a perspective of neurodiversity informed my decision to focus on the association of environmental factors with internalising symptoms, as opposed to hypothesised 'individual' factors (such as IQ, social skills, alexithymia, or 'autism severity'). This focus on difference or disorder being situated 'within' the individual misses the opportunity to address environmental contexts that are poorly suited for autistic people. By focusing my research explicitly on environmental factors, I

hoped to identify potentially-modifiable factors that could ameliorate the environment for autistic children and young people, thereby aiming for less disabling environments.

The concept of neurodiversity applies not just to autism, but has begun to be applied to down's syndrome, dyslexia, attention-deficit hyperactivity disorder (ADHD), and bi-polar disorder – amongst others (Armstrong, 2015). As a result, researchers within the fields of psychology, medicine or education may increasingly wish to consider the position of their research with regards to prior deficit-focused models of neurocognitive difference, based on the assumptions of individual deficits as opposed to poorly-fitting environments.

Opportunities and Limitations of using secondary data from longitudinal cohort studies

Part two of this thesis (the empirical paper) utilises secondary data from the Millennium Cohort Study (MCS), a longitudinal cohort study that follows the health and development of children within 19,231 families. The study began at the children's birth (between September 2000 and January 2002), and continues today, visiting children for follow-up data sweeps every few years. The UK has a strong history of longitudinal cohort studies (including 4 studies held by the Centre for Longitudinal Studies, https://cls.ucl.ac.uk/cls-studies/; the Avon Longitudinal Study of Parents and Children, http://www.bristol.ac.uk/alspac/; Growing Up in Scotland, https://growingupinscotland.org.uk/; Understanding Society, https://www.understandingsociety.ac.uk/ – amongst others).

To date the MCS has been following children for 22 years, with 6 sweeps of data published so far. Having read Helen Pearson's 2016 book, The Life Project, which documents the creation and subsequent research contributions of several UK cohort studies, including the MCS, I was already aware of the significant investment of time and resources required to set up and sustain these cohort studies, and the generosity of the participating families in giving their time and personal information to these projects. I felt that it was a considerable privilege to have this wealth of data at my fingertips via the UK Data Service (https://ukdataservice.ac.uk/).

Opportunities

Birth cohort studies such as the MCS offer a wealth of opportunities for answering questions about psychopathology and risk factors, particularly across early development, when these constructs are likely to be changeable. Equally, they offer the possibility of examining bidirectional effects between factors over time. Whereas a cross-sectional, observational study may offer a single snapshot of an individual's trajectory, data from a birth cohort study can reveal a far richer story.

In this thesis, the MCS allowed me to investigate questions regarding the occurrence of internalising symptoms in a group of children with autism, and the associations with potential environmental risk factors. Autism has an approximately 1% prevalence in the UK population (NHS, 2012) and therefore the large sample size of the MCS cohort enabled a sufficient sample size of autistic children to be identified. The use of the prospective birth cohort design may also have reduced selection bias at the recruitment stage, as all eligible families were proactively approached by study researchers. Whilst there may be subsequent bias in the loss of participants to follow up, it is likely that this design resulted in a more naturalistic sample of autistic children than if recruitment had taken place in a clinical setting. The MCS, like other birth cohort studies, was also very broad in measurement of variables, making it very well suited to answer questions about risk factors, as a wide variety of measures can be investigated, and as these are typically repeated at each sweep. Equally, this rich availability of measured variables may also allow the inclusion of a robust set of covariates in analysis, and perhaps offer a wider array of possibilities for handling missing data, for example by using multiple imputation with auxiliary variables (Romaniuk et al., 2014).

Limitations

However, despite the large sample size, richness of data, and opportunities afforded by long-term follow-up, I also encountered a number of limitations in my use of the MCS study data. In my case, many of these limitations occurred particularly in the context of asking research questions about autistic children and young people using a population-representative sample. A key limitation for

example, was in how children with autism should be identified to create the analytic sample. The MCS was not explicitly intended to answer questions about autism, and so there is no explicit assessment of autism to confer a diagnosis, and instead parents were asked at each sweep from the age of 5 whether "a doctor or other health professional ever told you that your child had Autism, Asperger's Syndrome or other autistic spectrum disorder?" (alongside questions about other developmental conditions). Naturally, as this question was posed to both parents at each sweep, and repeated over time, variability was introduced into the data for a given participant. This poses questions about which responses should be used to qualify an autism diagnosis, and naturally there is uncertainty about the origin and validity of these diagnoses. Additionally, as mentioned above, the original MCS researchers were not explicitly considering questions about autism in the design of their study, and so questionnaire measures and interview schedules do not include any autism-specific measures, for example parental adaptation or stimulation of the environment in the context of autism (Maljaars et al., 2014).

Further, non-autism-specific, limitations of using secondary data from cohort studies include a general lack of control of measure selection and change over time – for example, the MCS included measures of cyber-bullying in some sweeps but not others, meaning that this couldn't be used as a variable in the longitudinal analyses. As with all research, birth cohort studies are shaped by the social, political and research context of the time in which they were set up, and whilst additional measures can be introduced in later sweeps, it is likely that the research questions which motivated the design of the study and selection of measures at on the onset of the study (grounded in the social, political and research context of the time) would be different than research questions of interest 20 years further on. An example of this might be 'screen time', or 'social media use', which may be measured in more recent sweeps of data, but would not have felt relevant to include in early sweeps of the study.

A further limitation for the research as a whole, already alluded to in section one of this critical appraisal, is that using secondary data from a longitudinal cohort study such as this distances the researcher from the participants taking part. This has been considered to be more important when using qualitative analytic processes (Irwin, 2013; Ruggiano & Perry, 2019), however should also be considered in quantitative analysis. Secondary data reduces the participating children and families to anonymous data, which while rich and informative, removes the researcher's connection with the hopes of the participants for the research, the participants' lived experiences, and results in a loss of the researcher's ability to learn from and be shaped by encounters with participants.

Using Random-Intercept Cross-Lagged panel models for drawing clinical interpretations

In order to make the most of the rich longitudinal data made available by birth cohort studies, specialist analytic techniques are required. One example of this are cross-lagged regression models, which are now commonly used to investigate the time-lagged effects of one construct on another over a series of timepoints (Orth et al., 2021). In this thesis I use Random-Intercept Cross-Lagged Panel Models (RI-CLPMs; Hamaker et al., 2015), which in contrast to the basic Cross-Lagged Panel Model (CLPM), separates variance into its 'between-unit' and 'within-unit' components of variance. In the case of the study presented in part two of this thesis, the 'unit' of analysis is the autistic child or young person. Taking the example of internalising symptoms, the 'between-unit' variance would consist of the variability in stable, trait-level internalising symptoms experienced between different children in the sample. The 'within-unit' variance would consist of the differing levels of internalising symptoms experienced by a given child at one point in time, compared to their 'trait-level' internalising symptoms.

The RI-CLPM is generally considered to be superior to the standard CLPM (Hamaker et al., 2015; Orth et al., 2021) and has been widely used to investigate the longitudinal associations between psychological constructs in samples of children and young people (Fredrik et al., 2021; Kojima et al.,

2021; Neville et al., 2021; Zondervan-Zwijnenburg et al., 2022). However, I would like to consider here the implications of model choice for the results and subsequent clinical interpretations. Using a standard CLPM, where no distinction is made between within-person and between-person variance, we might, for example, conclude from a model: "When children with autism experience greater peer victimisation (than other children), they will experience a subsequent rank-order increase in internalising symptoms, compared to children experiencing less peer victimisation". Conversely, using the RI-CLPM, which separates between-person (trait-like) variance, from within-person variance, we might conclude from a model of the same constructs: "when children with autism experience more peer victimisation than usual (for them), they will experience a subsequent increase in internalising symptoms".

The interpretations of the models are therefore subtly different, and the model choice therefore has implications for what can be concluded from the research. In the RI-CLPMs presented in section two of this thesis, statistically significant cross-lagged associations were found for parental mental health and child internalising symptoms, and peer victimisation and child internalising symptoms. As these results were found with the use of RI-CLPMs, what can be concluded from these models tells us more about the consequences of variability over time for an individual child, rather than the consequences of variation in these constructs between children. As a result, there is a less precise estimate of identifying *which* children should be targeted by measures intended to reduce internalising symptoms, and perhaps a greater clinical implication for the preventative impact of interventions on an individual child's trajectory of internalising symptoms.

As described by Orth et al. (2021), not all research questions about the prospective effects of one construct on another are suited to an approach based solely on within-person variance, and it is worth careful reflection on whether it is between-person or within-person variance that a research question seeks to investigate. Additionally, this may be an important learning point for clinicians and policy-makers who wish to interpret research findings to draw conclusions from this.

Conclusion

Quantitative researchers are rarely required to reflect explicitly on their own position within their research, or on the process of conducting the research (Wren, 2004). I have however found this to be very important during the process of this thesis, particularly in my role as an 'outsider' to the field of autism (Wigginton & Setchell, 2016), and having conducted the research at a distance from the research participants, by systematic review and secondary data analysis.

Taking the opportunity for personal reflexivity in the writing of this critical appraisal has enabled me to consider where my research fits in amongst the wider stakeholders within the autism and research community, and how it has been shaped by the methodology of the cohort study and longitudinal analytic techniques. I hope that, whilst this thesis reports specifically on the experiences of autistic children and young people, these reflections may be helpful to others working as an 'outsider-researcher' in other contexts.

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APPENDICES

Appendix 1: JBI Quality Appraisal Tool

JBI Critical Appraisal Checklist for analytical cross sectional Studies

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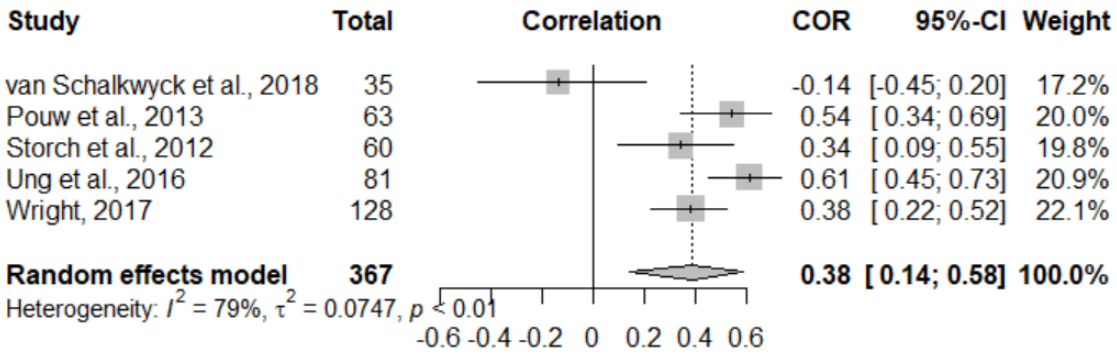
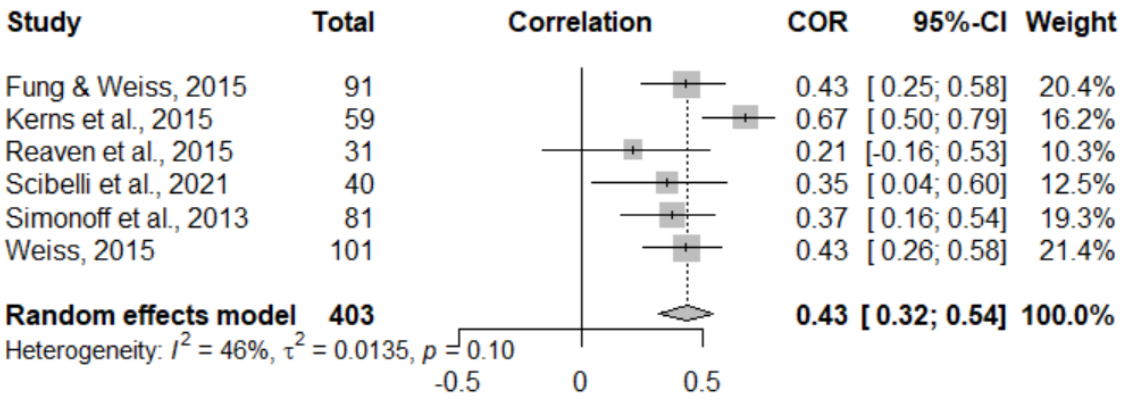
Appendix 2: Sensitivity meta-analysis of the associations between self-reported autistic adolescent internalising symptoms, and peer victimisation



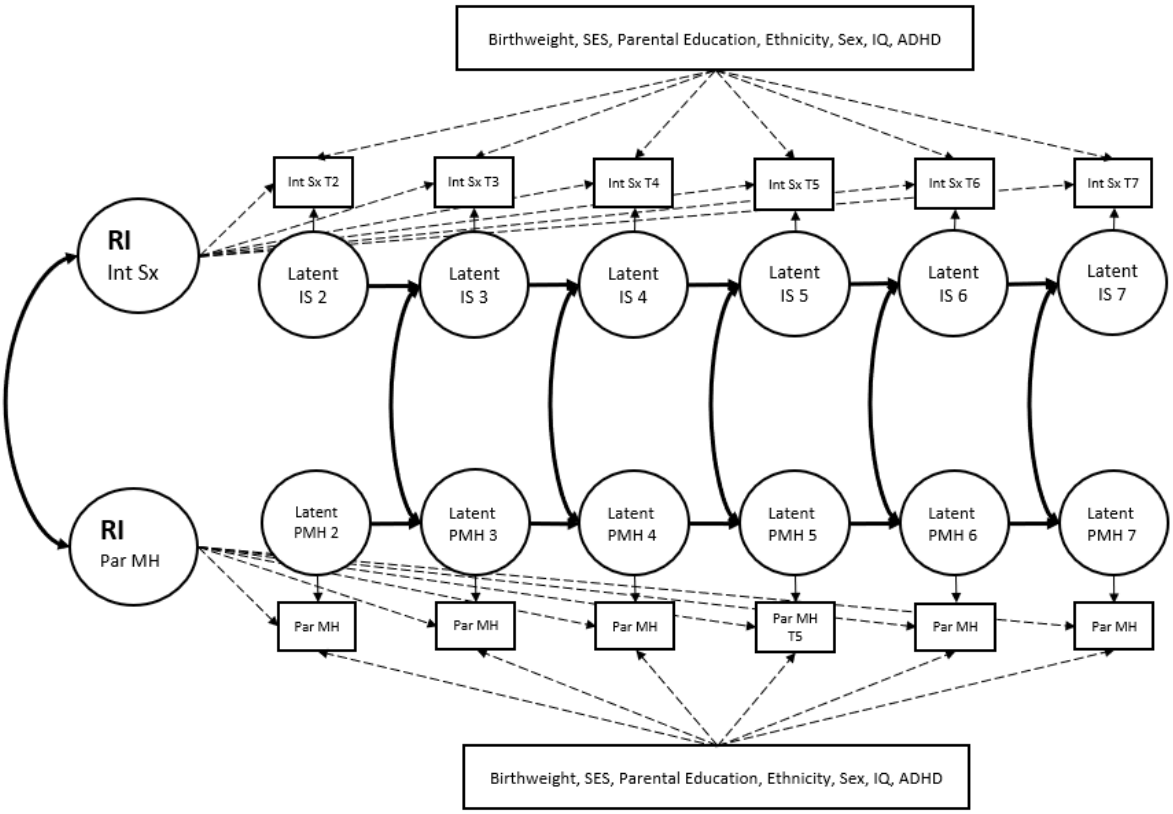
Sensitivity meta-analysis of the associations between self-reported autistic adolescent internalising symptoms, and peer victimisation.

Appendix 3: Sensitivity meta-analysis of the associations between autistic adolescent internalising symptoms, and parental MH or stress



Sensitivity meta-analysis of the associations between autistic adolescent internalising symptoms, and parental MH or stress

Appendix 4: Results of RI-CLPM between Internalising symptoms and Parental Mental Health with Birthweight, SES, Parental Education, Ethnicity, Sex, IQ and ADHD included as covariates



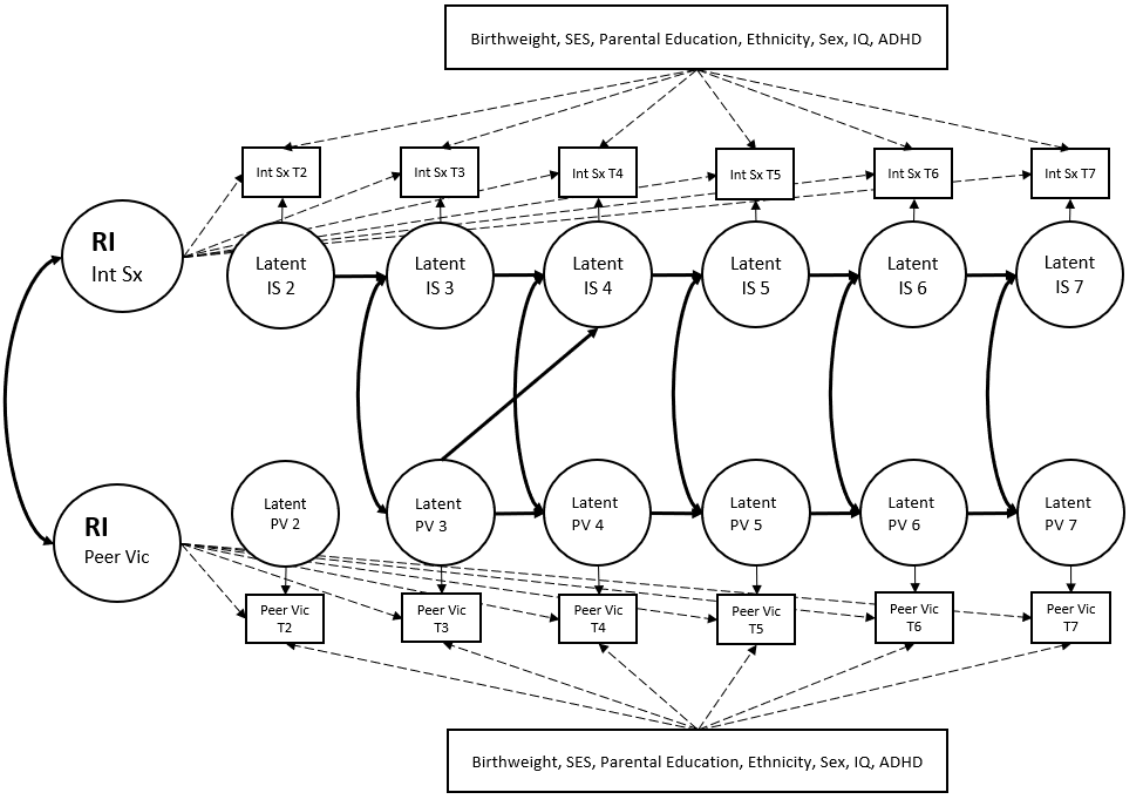
Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), with all covariates included. Statistically significant regressive and covariance paths are shown in bold in the model. For simplicity, covariates are shown as combined in this model.

The model fit was acceptable (RMSEA = 0.070, CFI = 0.946, SRMR = 0.054).

	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.300**	**<0.001**
	Par MH 2	**Par MH 3**	**0.373**	**0.001**
	Int Sx 3	**Int Sx 4**	**0.372**	**<0.001**
	Par MH 3	**Par MH 4**	**0.309**	**0.009**
	Int Sx 4	**Int Sx 5**	**0.410**	**<0.001**
	Par MH 4	**Par MH 5**	**0.396**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.447**	**<0.001**
	Par MH 5	**Par MH 6**	**0.528**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.490**	**<0.001**
	Par MH 6	**Par MH 7**	**-0.188**	**0.017**
Cross-lagged	Par MH 2	Int Sx 3	0.156	0.063
	Int Sx 2	Par MH 3	-0.032	0.621
	Par MH 3	Int Sx 4	0.101	0.107
	Int Sx 3	Par MH 4	-0.033	0.652
	Par MH 4	Int Sx 5	-0.031	0.627
	Int Sx 4	Par MH 5	0.004	0.949
	Par MH 5	Int Sx 6	0.011	0.834
	Int Sx 5	Par MH 6	0.057	0.307
	Par MH 6	Int Sx 7	-0.063	0.276
	Int Sx 6	Par MH 7	0.118	0.077

Tabulated results of Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6). All covariates included. Statistically significant relationships are highlighted in bold.

Appendix 5: Results of RI-CLPM between Internalising symptoms and Peer Victimisation with Birthweight, SES, Parental Education, Ethnicity, Sex, IQ and ADHD included as covariates



Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ), with all covariates included. Statistically significant regressive and covariance paths are shown in bold in the model. For simplicity, covariates are shown as combined in this model.

The model fit was good (RMSEA = 0.036, CFI = 0.981, SRMR = 0.029).

	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.410**	**<0.001**
	Peer Vict 2	Peer Vict 3	-0.007	0.955
	Int Sx 3	**Int Sx 4**	**0.458**	**<0.001**
	Peer Vict 3	**Peer Vict 4**	**0.347**	**<0.001**
	Int Sx 4	**Int Sx 5**	**0.447**	**<0.001**
	Peer Vict 4	**Peer Vict 5**	**0.301**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.465**	**<0.001**
	Peer Vict 5	**Peer Vict 6**	**0.405**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.532**	**<0.001**
	Peer Vict 6	**Peer Vict 7**	**0.444**	**<0.001**
Cross-lagged	Peer Vict 2	Int Sx 3	0.128	0.700
	Int Sx 2	Peer Vict 3	0.018	0.575
	Peer Vict 3	**Int Sx 4**	**0.451**	**0.025**
	Int Sx 3	Peer Vict 4	0.012	0.574
	Peer Vict 4	Int Sx 5	0.193	0.296
	Int Sx 4	Peer Vict 5	-0.017	0.405
	Peer Vict 5	Int Sx 6	-0.149	0.399
	Int Sx 5	Peer Vict 6	0.015	0.319
	Peer Vict 6	Int Sx 7	0.044	0.826
	Int Sx 6	Peer Vict 7	0.020	0.268

Tabulated results of Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ), including all covariates. Statistically significant relationships are highlighted in bold.

Appendix 6: Sensitivity analysis – descriptive statistics of conservative autism sample

Demographic variable		N (Percentage)
Sex	Male	316 (76.1%)
	Female	99 (23.9%)
ADHD	Parents indicated a stable diagnosis at age 5yo, 7yo, 11yo or 14yo	121 (29.2%)
	No stable ADHD indication	294 (70.8%)
Ethnicity	White	370 (89.2%)
	BAME Ethnicity	45 (10.8%)
SES (Family income)	£ 0 - £31,199	314 (75.7%)
	£31,200 and greater	70 (16.9%)
	Missing income	31 (7.5%)
Parental Education	NVQ levels 1-2 (GCSE level)	166 (40.0%)
	NVQ levels 3, 4, 5 (A-level - higher education)	186 (44.8%)
	Missing education	63 (15.2%)
Demographic variable	(N)	Mean, SD Range
Birthweight (kg)	555	3.36 ± 0.68, 0.62 – 5.73
IQ (age 7)	388	93.0 ± 17.5 41.2 – 133.9

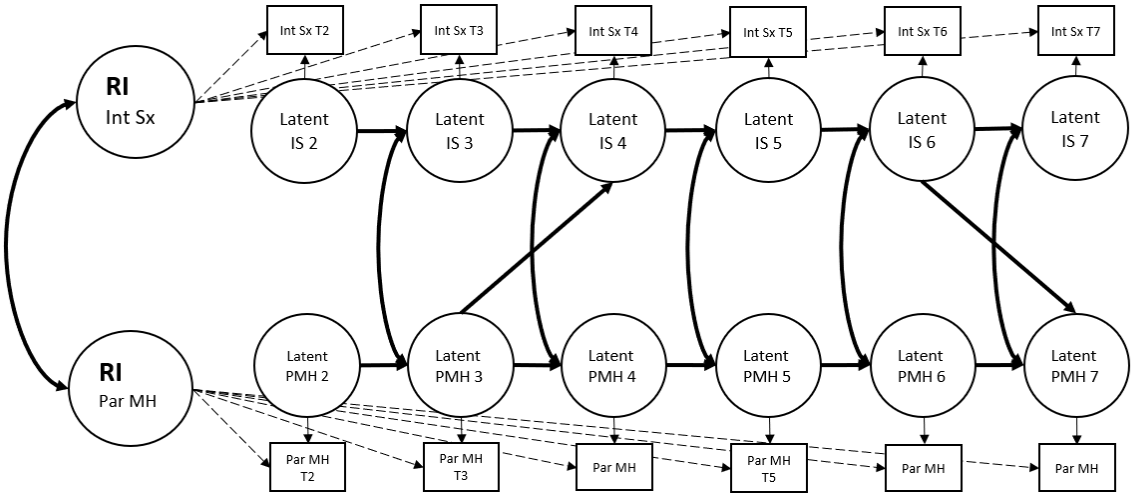
Sensitivity analysis – descriptive statistics of conservative autism sample (n = 415)

Variable		Time 2 (3yo)	Time 3 (5yo)	Time 4 (7yo)	Time 5 (11yo)	Time 6 (14yo)	Time 7 (17yo)
Internalising symptoms	*(N)*	*374*	*399*	*401*	*388*	*382*	*307*
	Mean, SD	1.65 ± 1.78	2.21 ± 2.02	2.96 ± 2.42	4.17 ± 2.70	4.34 ± 2.64	4.12 ± 2.83
% within clinical range		6.42	15.54	28.43	43.81	47.12	43.97
Peer Victimisation	*(N)*	*355*	*378*	*372*	*386*	*376*	*266*
	Mean, SD	0.12 ± 0.36	0.38 ± 0.60	0.55 ± 0.67	0.85 ± 0.75	0.81 ± 0.74	0.67 ± 0.71
% reporting peer victimisation		1.41	6.35	9.68	21.24	19.41	13.91
Parental Mental Health	*(N)*	*350*	*394*	*394*	*383*	*375*	*292*
	Mean, SD	4.44 ± 4.74	4.50 ± 4.81	4.87 ± 5.01	5.93 ± 5.36	6.05 ± 4.83	8.67 ± 5.14
% within clinical range		8.00	7.36	8.88	10.97	12.27	19.86

Sensitivity analysis – descriptive statistics of conservative autism sample (n = 415), of Internalising symptoms, Peer Victimisation and Parental Mental Health over 6 sweeps. Internalising symptom scores are SDQ emotional sub-scale (range 0 - 10), Peer victimisation scores are SDQ item 19 (range 0 - 2), Parental mental health scores are K6 (range 0 – 24). In all cases higher scores indicate greater severity.

Appendix 7: Sensitivity analysis. RI-CLPM between Internalising symptoms and Parental Mental Health – conservative autism sample

This RI-CLPM was performed using a more conservative sample of children with autism (N = 415). This sensitivity analysis was performed in order to compare the RI-CLPM to those based on the general sample of autistic children.



Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), without covariates. Statistically significant regressive and covariance paths are shown in bold in the model.

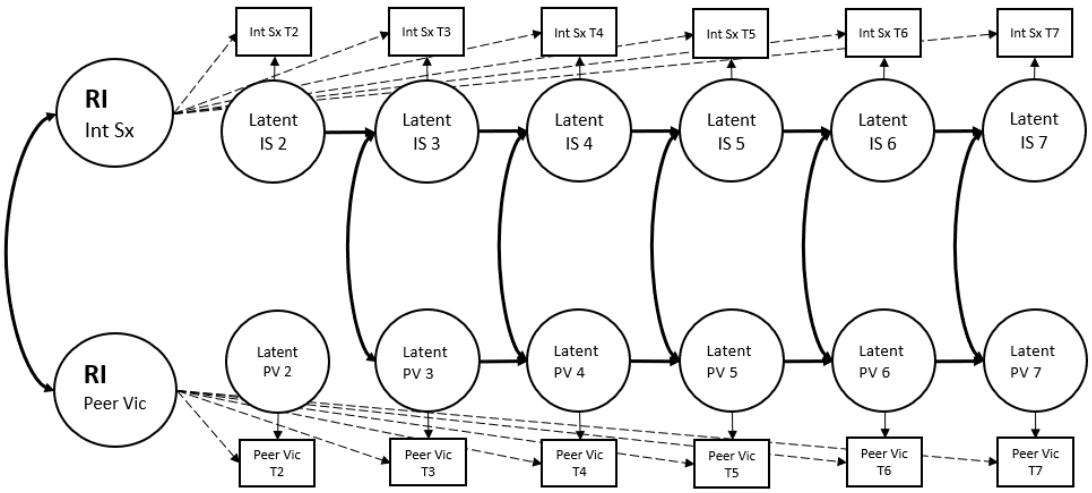
The model fit was acceptable (RMSEA = 0.077, CFI = 0.937, SRMR = 0.085).

	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.299**	**0.002**
	Par MH 2	**Par MH 3**	**0.353**	**0.009**
	Int Sx 3	**Int Sx 4**	**0.462**	**<0.001**
	Par MH 3	**Par MH 4**	**0.336**	**0.010**
	Int Sx 4	**Int Sx 5**	**0.486**	**<0.001**
	Par MH 4	**Par MH 5**	**0.439**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.454**	**<0.001**
	Par MH 5	**Par MH 6**	**0.442**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.562**	**<0.001**
	Par MH 6	**Par MH 7**	**-0.267**	**0.042**
Cross-lagged	Par MH 2	Int Sx 3	0.068	0.127
	Int Sx 2	Par MH 3	-0.029	0.889
	Par MH 3	**Int Sx 4**	**0.077**	**0.043**
	Int Sx 3	Par MH 4	-0.126	0.514
	Par MH 4	Int Sx 5	-0.016	0.711
	Int Sx 4	Par MH 5	0.121	0.407
	Par MH 5	Int Sx 6	0.022	0.450
	Int Sx 5	Par MH 6	0.110	0.215
	Par MH 6	Int Sx 7	-0.025	0.573
	Int Sx 6	**Par MH 7**	**0.304**	**0.042**

Tabulated results of Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6). Covariates not included. Statistically significant relationships are highlighted in bold.

Appendix 8: Sensitivity analysis. RI-CLPM between Internalising symptoms and Peer Victimisation – conservative autism sample

This RI-CLPM was performed using a more conservative sample of children with autism (N = 415). This sensitivity analysis was performed in order to compare the RI-CLPM to those based on the general sample of autistic children.



Random-Intercept Cross-Lag Panel Model between autistic children's internalising symptoms (Emotional sub-scale SDQ) and parental mental health (K6), without covariates. Statistically significant regressive and covariance paths are shown in bold in the model.

The model fit was good (RMSEA = 0.032, CFI = 0.986, SRMR = 0.042).

	Predictor	Outcome	Standardized coefficient	P
Autoregressive	**Int Sx 2**	**Int Sx 3**	**0.293**	**0.003**
	Peer Vic 2	Peer Vic 3	-0.071	0.609
	Int Sx 3	**Int Sx 4**	**0.464**	**<0.001**
	Peer Vic 3	**Peer Vic 4**	**0.310**	**<0.001**
	Int Sx 4	**Int Sx 5**	**0.484**	**<0.001**
	Peer Vic 4	**Peer Vic 5**	**0.258**	**<0.001**
	Int Sx 5	**Int Sx 6**	**0.471**	**<0.001**
	Peer Vic 5	**Peer Vic 6**	**0.406**	**<0.001**
	Int Sx 6	**Int Sx 7**	**0.549**	**<0.001**
	Peer Vic 6	**Peer Vic 7**	**0.447**	**<0.001**
Cross-lagged	Peer Vic 2	Int Sx 3	0.223	0.563
	Int Sx 2	Peer Vic 3	0.006	0.869
	Peer Vic 3	Int Sx 4	0.372	0.089
	Int Sx 3	Peer Vic 4	0.010	0.683
	Peer Vic 4	Int Sx 5	0.054	0.790
	Int Sx 4	Peer Vic 5	-0.013	0.553
	Peer Vic 5	Int Sx 6	-0.055	0.770
	Int Sx 5	Peer Vic 6	0.015	0.334
	Peer Vic 6	Int Sx 7	0.106	0.636
	Int Sx 6	Peer Vic 7	0.018	0.329

Tabulated results of Random-Intercept Cross-Lag Panel Model between Autistic children's internalising symptoms (Emotional sub-scale SDQ) and peer victimisation (item 19, SDQ). Covariates not included. Statistically significant relationships are highlighted in bold.

Appendix 9: Methodological details of covariates

Ethnicity

The child's ethnicity, as indicated by the main parent respondent at child age of 9-months-old. For the purpose of statistical analyses, this was categorised into white *vs* any non-white ethnicity. Whilst the distribution of ethnicities was representative of the UK population at the time of sampling, ethnic diversity was still low, so the decision was made to split into two categories only for statistical analysis.

Sex

The child's sex, as indicated by the main parent respondent at child age of 9-months-old.

Child IQ

In line with previous MCS research, general intellectual ability was indexed with a factor score derived from principal components analysis of age-adjusted scores from three ability assessment tests: BAS Pattern Construction, BAS Word Reading (measuring educational knowledge of reading) and the National Foundation for Educational Research Progress in Maths, all measured at 7-years-old. See Flouri et al., 2018 and Hanscombe et al., 2012 for further details.

Parental education

Highest level of education achieved by main parent respondent, as measured by NVQ level or NVQ equivalent. Data collected at first MCS sweep (9-months-old), except in the case of 27 children who were born within the study timeframe, but did not join the study until sweep 2. Parental education data was therefore collected at sweep 2 for these 27 children.

Family SES

Measured by banded family income (single parent income, or combined if there are two resident parents) at the first MCS sweep (9-months-old). In the case of the 27 children who were born within

the study timeframe, but did not join the study until sweep 2, family SES data was collected at sweep 2.

ADHD diagnosis

This was chosen as a covariate given the increased prevalence of co-occurring ADHD amongst autistic individuals (Reiersen et al., 2007; Simonoff et al., 2008; Steinhausen et al., 2006). In our autistic sample, approximately 30% were reported to have a diagnosis of ADHD. Diagnosis was based on the main parent's answer to the question, "Has a doctor or health professional ever told you that [Cohort child's name] had ADHD?" at any sweep between the ages of 5 – 14.

Child's birthweight

The child's birthweight, measured in kilograms, as indicated by the main parent respondent at child age of 9-months-old.