EXPLORING THE OCCURRENCE OF COMMON MENTAL HEALTH DIFFICULTIES ACROSS THE AUTISTIC SPECTRUM

by

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THESIS OVERVIEW

This thesis contains a single volume composed of five chapters and is submitted to the University of Birmingham to satisfy requirements for the degree of Doctorate of Clinical Psychology. This thesis includes a literature review, an empirical research paper and two corresponding press releases providing brief summaries of each piece of work in language suitable for a lay audience.

Literature Review

The literature review section features a meta-analysis of anxiety and depressive disorder prevalence rates among young autistic people. The impact of moderators is explored and sources of heterogeneity are discussed.

Empirical Research Paper

The empirical research paper describes a study conducted to explore the relationship between depressive symptoms, autistic traits and sleep difficulties. Theoretical and clinical implications are discussed.

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Chapter One

Literature Review

A Meta-Analysis Estimating the Prevalence of Anxiety and Unipolar Depressive Disorders

Among Autistic Youth

Word count: 11, 258

Abstract

Background: The co-occurrence of both anxiety and unipolar depression with autism is welldocumented. Less known is the prevalence of specific anxiety disorders and prevalence of specific unipolar depressive disorders, and how prevalence varies based on informant. **Aims:** This meta-analysis aimed to calculate prevalence estimates for specific anxiety and unipolar depressive disorders among young autistic people (ages 5-25); whilst considering the impact of moderators, particularly informant type.

Method: A meta-analytic review of the literature was conducted according the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines. Studies were screened and 56 articles met criteria. Pooled prevalence estimates were calculated and sources of heterogeneity were explored.

Results: The following prevalence rates were recorded; unspecified anxiety (40%), specific phobia (23%), generalised anxiety (15%), social anxiety (15%), separation anxiety (11%), agoraphobia (6%), panic disorder (3%), unspecified depressive disorder (24%), major depressive disorder (8%), dysthymia (5%). Prevalence estimates varied according to rating type. Unspecified anxiety and depressive disorder estimates exceeded the respective prevalence rates for neurotypical young people.

Conclusions: Anxiety and unipolar depressive disorders are highly prevalent among young autistic people, highlighting the need for development of more effective adapted interventions. Much improvement to current assessment methods is needed, along with greater inclusion of people with intellectual disabilities and systematic reporting of prevalence rates according to sex.

Introduction

Autistic Spectrum Disorder (ASD) is a neurodevelopmental condition comprising variable impairment to social interaction and communication, in addition to preoccupation with restricted interests and repetitive patterns of behaviour (American Psychiatric Association, 2013).

Autism is associated with increased risk of co-morbid mood disorders such as anxiety and depression (Gotham, Brunwasser & Lord, 2015). There are many reasons why autistic people might experience anxiety or depression; particularly when considering the challenge of existing within a social environment without the necessary awareness, skill or motivation to thrive. ASD characteristics themselves may increase vulnerability to mood difficulties. For instance, hyper-sensitivity may lead to development of phobia and subsequent avoidance of objects, people or places (Uljarevic, Nuske, & Vivanti, 2016); whilst, social cognition deficits could lead to increased anxiety in social situations (Pearcey et al., 2021). Avoidance in either scenario could then disrupt relationships or reduce access to meaningful activities, leading to unmet needs or feelings of loneliness. It is of note that loneliness has been reported in samples of young autistic people, and loneliness is associated with increased rates of unipolar depression (Bauminger, Shulman & Agam, 2003; Lempinen, Junttila & Sourander, 2018; White & Roberson-Nay, 2009). Additionally, experiences of bullying are associated with development of anxiety and unipolar depression, and young people with ASD are 3-4 times more likely to be bullied than typically developing peers (Forrest, Kroeger & Stroope, 2020; Hoover & Kaufman, 2018; Rodriguez, Drastal & Hartley, 2021).

It is important to highlight the scale and impact of comorbidities in the autistic population for several reasons. Firstly, widescale epidemiological studies that incorporate the prevalence and potential harm of a particular condition to determine its burden, report that anxiety and depression are the most disabling mental disorders globally, irrespective of age or sex; secondly, both disorders are associated with increased risk of chronic disease and suicide (Clarke & Currie, 2009; Moitra et al., 2021; Santomauro et al., 2020; Vos et al., 2019). Third, mental disorders among autistic people are associated with worse outcomes than for neurotypical people (Joshi et al., 2013; Kraper et al., 2017), and comorbidities are associated with greater severity of ASD symptomology and reduced quality of life (Bellini, 2004; Ben-Sasson et al., 2008; van Steensel, Bögels, & Dirksen, 2012; Wood & Gadow, 2010).

Whilst several studies have reported on prevalence of anxiety in young autistic people; results have varied according to whether experience of anxiety in general is reported or disorder-level anxiety, as determined by a standardised measure. Previous reviews have reported greater levels of anxiety compared to the general population (MacNeil, Lopes & Minnes, 2009; Van Steensel & Heeman, 2017), with prevalence estimates between 11% and 84% (White, Oswald, Ollendick & Scahill, 2009). A meta-analysis by Lai et al. (2019) reported estimated anxiety disorder prevalence of 20%; however, this was not specific to young people. Only one previous meta-analysis has investigated disorder-level prevalence of anxiety among young autistic people specifically. Van Steensel, Bogels and Perrin (2011) reported that 39.6% of autistic people < 18 years had a co-morbid anxiety disorder; among these, specific phobia (29.8%) and social anxiety disorder (16.6%) were the most common.

Synthesis of the literature reporting on prevalence of unipolar depression among young people with ASD is scant. To date, only one published meta-analysis exists on this topic. Hudson, Hall and Harkness (2018) reported cumulative prevalence rates of 10.6% across 18 studies reporting on depression among autistic people <18 years. However, prevalence rates relating to specific unipolar depressive disorders as classified in DSM-5, such as major depressive disorder and persistent depressive disorder (referred to hereon as

dysthymia) were not reported. Arguably, it is important to ascertain clarity regarding prevalence of depressive disorder subtypes, in order to determine duration and severity of distress, and ensure adequate provision of support from health services.

Further, since the COVID-19 pandemic, evidence indicates a 25.6% increase in global anxiety prevalence and a 27.6% increase in global major depressive disorder prevalence, with greater increases being observed for younger people (COVID-19 Mental Disorders Collaborators, 2021). The full extent to which the pandemic has impacted the mental health of young autistic people is not yet known; however, preliminary evidence indicates that mental distress has increased substantially within this group (Vasa et al., 2021). Such findings are understandable considering the differences experienced by autistic people; for instance, difficulty tolerating uncertainty and reliance on routines and external support. Therefore, there is reason to believe that previous prevalence estimates are out-dated.

The current meta-analysis aimed to conduct a timely review of the relevant literature and update prevalence estimates for specific anxiety and unipolar depressive disorders. For the purpose of this meta-analysis, the term 'young people' refers to people between 5 and 25 years old. Mental health difficulties in youth typically onset around age five; therefore, the lower age limit of five years was chosen. Additionally, it is suggested that 75% of mental health conditions onset between childhood and young adulthood (Kessler et al., 2005; NHS Digital, 2018). Therefore, the upper age limit of 25 was chosen to allow for prevalence estimates that are more inclusive of autistic youth (18-25 years), instead of primarily focusing on children (<18 years) as has been reported previously.

It is known that anxiety and unipolar depression prevalence estimates vary according to a number of factors, with significant heterogeneity being reported (Hudson et al., 2019; van Steensel et al., 2011, 2017). A further aim of this meta-analysis was to explore the extent to which study factors (design, recruitment location, measurement method) and participant characteristics (age, sex and intellectual ability) moderate prevalence estimates.

One well-established source of variance in anxiety and unipolar depression prevalence estimates is whether data is derived from self-report, parent-report or professional assessment. For instance, for anxiety, a meta-analysis by van Steensel et al. (2011) reported a relationship between use of interview-based measures in which professionals were rating anxiety and higher prevalence of disorders; whilst the same relationship was found between questionnaires-based methods using self or parent report for social, separation and generalised anxiety disorders. Similarly, a later meta-analysis by van Steensel et al. (2017) reported that studies using parent-report demonstrated larger differences between autistic and neurotypical children in relation to anxiety severity. For depression, a meta-analysis of lifetime unipolar depression prevalence reported that self-report was associated with higher prevalence rates than caregiver report (Hudson et al., 2019). Given this variance, van Steensel et al. (2011) proposed that the best estimate of disorder prevalence among young autistic people may be achieved by studies that use a combination of measures and multiple informants. Therefore, to most accurately estimate prevalence and present sources of variance, this meta-analysis reports pooled prevalence estimates in addition to a breakdown of estimates according to informant or 'rater' type (self, parent, professional).

Method

Identifying Primary Studies

Search of Electronic Databases

A systematic search was conducted on 15th July 2021, using online databases with key word search (PsycINFO, OVID MEDLINE (R), Embase). The anxiety search terms are outlined in Table 1 and were developed from Van Steensel et al. (2011) and Mingins, Tarver,

Waite, Jones & Surtees (2021). Obsessive Compulsive Disorder (OCD) was not included in the anxiety search terms because OCD is not classified as an anxiety disorder in DSM-5 or ICD-11, but instead both manuals have included OCD in a new category titled 'Obsessive Compulsive and Related Disorders'; therefore, OCD prevalence is not included in this metaanalysis.

Previous meta-analyses had not reported depression prevalence at the disorder level; therefore, the depression search terms outlined in Table 1 were designed to capture unipolar depressive disorder classifications in ICD-11 and DSM-5. In DSM-5, chronic major depressive disorder and dysthymia have been combined to form the new category of persistent depressive disorder. As dysthymia has been renamed but the underlying phenomenology of the disorder remains the same (which was not the case for OCD as it was recategorised), search terms included 'dysthymia' to capture data that was collected under the former diagnostic term. The aim of the search was to obtain a comprehensive overview of literature pertaining to prevalence of anxiety and unipolar depressive disorders among young autistic people (5-25 years).

Table 1

Search Criteria

Construct	Free Text Search Terms	Method of Search	Limits
Autism Spectrum Disorder	Autis* asperger* PDD (Pervasive developmental adj1 disorder)	All search terms combined with <i>OR</i>	Human English language 1994- July 2021
Anxiety	Anxiety Disorders/ (Panic adj3 Disorder) Mutism Agoraphobi* Phobia* (Disorder* adj3 Social* adj3 Function*) (anxiety or anxious*)		Age 2 – 29 years
Depression	Depressive* Depression Dysthymi*		
Disorders to exclude from search	Obsessive Compulsive Disorder, Attention Deficit Disorder with Hyperactivity, exp Dementia, Trichotillomania, Posttraumatic Stress Disorder, Complex PTSD, Schizophrenia, Bipolar Disorder, Cyclothymic Disorder, Psychosis, Alcohol Abuse, Substance Use Disorder, Drug Abuse, Alcoholism, Tics, Tourette Syndrome, Dissociative Disorders, Depersonalisation, Bulimia, Eating disorders, Anorexia Nervosa, Binge Eating, Purging, Oppositional Defiant Disorder, Conduct Disorder	All search terms combined with <i>OR</i> and then combined with main search using <i>NOT</i>	

Paper Selection

The search strategy was guided by Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guide-lines (Page et al., 2020), and is presented in Figure 1. Search terms were combined and limited to articles published in English, including human participants only. Age limits were used to include articles reporting on prevalence for the target age range of 5-25 years. Age limit functions differed across each database but aimed to be inclusive of studies covering preschool to young adulthood, whilst effectively limiting search results. Combined database search results totalled 5344 articles. Following removal of duplicates 3717 articles were screened by study title and abstract using the exclusion criteria. The three most common reasons for exclusion were: not being specifically related to ASD (n=1243), not being focused on anxiety or depression (n=783), not being focused on young people between the ages of 5 and 25 (n=365). Following exclusion of a further 3234 articles, the full texts of the remaining 483 articles were then reviewed in more detail against the exclusion criteria (Table 2). Subsequently, 56 articles met the full inclusion/exclusion criteria for this meta-analysis.

Figure 1

PRISMA Flow Diagram Depicting the Results of the systematic search and application of inclusion criteria



Table 2

Inclusion and Exclusion Criteria

Inclusion Criteria	Justification
Disorders of Interest	
Studies that reported on young people with an ASD diagnosis.	The target population for this meta-analysis.
Studies that included assessment of anxiety and/or depression. Studies had to report the number/percentage of subjects with a DSM-5 anxiety/depressive disorder and/or the number of subjects falling above clinical cut-off for anxiety/depression on a standardised measure.	To enhance validity of prevalence rates only studies that included proper assessment of anxiety/depression were included. This meta-analysis aimed to estimate anxiety/depressive disorder prevalence and not presence of any level of anxiety/depression. Previous meta- analyses did not include prevalence rates for anxiety and depression, or collect data regarding prevalence of anxiety/depressive disorders within this age range.
Participant focus	
Studies including young people between the ages of 5 and 25. Studies collecting data from parents about their child were also included provided that their child's age fell within the specified range.	A previous review conducted by van Steensel, Bogels and Perrin (2011) included young people <18. This meta- analysis aimed to update prevalence rates with a wider age range, stretching from school age to young adulthood. Presentation of depression/anxiety in children <5 is likely to be highly variable, particularly when compared to older children, and it is difficult to decipher whether levels of anxiety/depression are clinically significant (Whalen, Sylvester & Luby, 2017). The inclusion of young adults (age 18 to 25) aimed to capture data that would typically be missed when focusing on young people under 18.
Outcome data	
Studies had to be data-based and not reviews of the literature. The studies were required to report either the number or percentage of participants meeting criteria for an anxiety or depressive disorder.	To ensure that prevalence rates could be calculated.
Studies had to report current prevalence rates, not lifetime prevalence.	To ensure that prevalence rates could be estimated for people age 5-25. Studies that reported lifetime prevalence included participants older than 25 and collected data at multiple time points, making it difficult to determine which data to disaggregate. If lifetime prevalence and current prevalence were reported, the current prevalence estimate was included.

Table 2

Inclusion and Exclusion Criteria

Inclusion Criteria	Justification
Date of Article	
Only articles published from 1994 onwards were included.	The DSM-IV was published in 1994 and Autism was considered as spectrum of conditions hereafter. Additionally, there was an increase in the number of conditions classified as mood disorders. Although it is likely that studies published in 1994 collected data using earlier criteria, 1994 was considered a reasonable cut-off point for studies included in this meta-analysis.
Exclusion Criteria	Justification
Type of article	
Article types excluded: meta-analyses, theoretical papers, reviews, commentaries, clinical guidance, conference abstracts, qualitative studies, case reports and treatment, experimental or intervention studies.	These articles did not provide the outcome data needed for this meta-analysis.
Outcome Data and study design (N<20, single-case designs, Case series)	
Studies that did not clearly report prevalence data or if it was impossible to disaggregate information to determine prevalence (e.g., only group mean for clinical cut-off reported).	To ensure that effect size could be calculated.
Studies that did not include an assessment of anxiety or depression and simply reported rates based on self or parent report/survey/medical records were not included.	To ensure that the proper assessment of anxiety and depression had been undertaken. Bouts of anxiety and low mood can be common in young people. If no assessment was documented then it is unclear whether a proper assessment was ever completed or whether anxiety/depression was clinically significant or not, which could have artificially increased prevalence rates.
Participant Characteristics	
Young people with ASD traits or studies that selected participants based on any criteria other than ASD (e.g., Fragile X syndrome, existing diagnoses anxiety and/or depression) were not included.	The focus of this meta-analysis was ASD not autistic traits or other syndromes/conditions. Prevalence rates for anxiety and depression may vary across these groups. Exclusion aimed to reduce heterogeneity in the target population and the corresponding impact on prevalence rates. Excluding studies that selected participants on the basis of existing anxiety or depression reduced the risk of selection bias and increased likelihood of obtaining a representative sample to enable generalisations.

Data Extraction

The author extracted the following data from each paper; demographics, sample source and size, whether people with an intellectual disability were included, diagnostic method and measures used, type of anxiety or depression measured, and whether data was gathered by self, parent or professional report. A summary of study characteristics can be found in Table 3. Event rates were calculated by extracting the number of participants with and without the condition of interest. When possible, prevalence estimates were calculated separately for data derived from self, parent and professional ratings to highlight any variation in prevalence due to rating source.

Anxiety and depression data were extracted separately for each specific disorder (generalised anxiety disorder, agoraphobia, specific phobia, social anxiety disorder, panic disorder, separation anxiety disorder or major depressive disorder, dysthymia/persistent depressive disorder). If the study used a measure that provided data on total anxiety or depression but did not specify type, then data was extracted to calculate prevalence estimates for unspecified disorder-level anxiety or depression (referred to hereon in as 'unspecified anxiety disorder' or 'unspecified depressive disorder'). For one study, more than one measure was used for the same disorder (Kaat & Lecavalier, 2015), the measure with the most desirable reliability statistics was chosen. For longitudinal studies that reported more than one time-point, the most recent data was extracted.

Table 3Characteristics of Studies Included in the Meta-analysis and the Results Reported by Each

FIRST AUTHOR	N	N Mean % Male		Method N	Measure/ Rater(s)	Rater(s)	Anxiety Disorder (%) ^b							Depressive				
(YEAR)		age			Interview			D							visorder (%) ^b			
		(Years)			name ^a	le ^a 1	UA	G	So	Se	Sp	Р	Α	UD	Μ	D		
AMR (2012)	60	8.36	61.7	Interview	SCICA	Professional	58	1	-	8	4	-	-	-	13	-		
BELLINI (2004)	41	14.2	85.4	Questionnaire	SAS-A	Self	-	-	48	-	-	-	-	-	-	-		
BEN-ITZCHAK (2020)	61	13.8	Not reported	Questionnaire	SCARED	Parent	56	18	28	39	-	66	-	-	-	-		
BITSIKA (2015A)	33	10.96	100	Questionnaire	CASI-D	Self	-	-	-	-	-	-	-	47	-	-		
BITSIKA (2015B)	128	11.25	100	Interview	Kid-SCID	Professional	-	32	-	-	-	-	-	-	-	-		
BITSIKA (2016)	150	11.2	100	Questionnaire	CASI-GAD-	Parent	43	-	-	-	-	-	-	8	-	-		
					D	Self	31	-	-	-	-	-	-	9	-	-		
BRADLEY (2011)	36			Interview	SAPPA	Professional	-	6	-	-	-	-	-	-	-	-		
BRIOT (2020)	79	11.51	89.97	Questionnaire	LSAS	Parent	72	-	19	-	-	1	14	19	-	-		
DE BRUIN (2007)	94	8.5	88.3	Interview	DISC-IV-P	Professional	-	5	12	9	38	1	7	-	11	2		
CAI (2019)	56	18.5	70	Questionnaire	DSM-5- GAD-D	Self	41	-	-	-	-	-	-	-	36	-		
COSKUN (2020)	60	12.66	86.9	Interview	K-SADS-PL- T	Professional	-	20	8	25	23	2	-	-	25	-		
DECKERS (2017)	73	11.22	84.93	Interview	Not reported	Professional	12	-	-	-	-	-	-	-	-	-		
DEMIRKAYA (2016)	55	13.56	Not reported	Interview	K-SADS-PL	Professional	44	5	6	2	7	-	-	18	-	-		
DEN HOUTING (2018)	100	11.4	78	Questionnaire	ASC-AD	Parent	63	-	-	-	-	-	-	-	-	-		
DEN HOUTING (2019)	30	11.2	83	Questionnaire	SCAS-PV	Parent Self	80 43	-	-	-	-	-	-	-	-	-		
GADOW (2005)	284	8.3	85	Questionnaire	CSI-4	Parent	-	24	-	7	-	-	-	-	6	11		

						Professional	-	24	13	-	-	-	-	-	2	10
GJEVIK (2011)	71	11.8	82	Interview	K-SADS-PL	Professional	42	0	7	0	31	-	-	7	1	1
HEBRON (2014)	22	14	86.3	Questionnaire	BYI	Self	59	-	-	-	-	-	-	36	-	-
HERRINGTON (2017)	38	12.76	68.4	Interview	ADIS	Professional	63	-	-	-	-	-	-	-	-	-
HESSL (2021)	48	11.77	75	Interview	ADIS	Professional	58	23	8	8	33	-	-	-	-	-
JOHNSTON (2017)	67	9.82	85	Questionnaire	BASC-2	Parent	34	-	-	-	-	-	-	48	-	-
KAAT (2015)	46	12.4	Not reported	Questionnaire	RCADS	Parent Self	37 7	28 0	37 7	33 13	-	30 13	-	48 44	-	-
KAAT (2013)	115	8.5	86	Questionnaire	CASI-4	Parent Professional	32 21	18 14	23 10	3	-	-	-	11	3	11
KERNS (2014)	50	10.48	78	Interview	ADIS	Professional	17	3	3	2	5	-	-	-	-	-
KUUSIKKO (2008)	54	11.2	77.7	Questionnaire	SPAI-C	Self	-	-	39	-	-	-	-	-	-	-
LEYFER (2006)	109	9.2	94.29	Interview	ACI-PL	Professional	-	2	7	12	44	0	-	3	10	-
LOPATA (2010)	40	9.75	80	Questionnaire	BASC-2	Parent	8	-	-	-	-	-	-	40	-	-
MAGIATI (2014)	38	12.83	76.3	Questionnaire	SCAS	Self Parent	29 45	16 29	11 21	29 32	-	-	-	-	-	-
MAGIATI (2016)	241	10.33	81.7	Questionnaire	SCAS-PV	Parent	26	19	9	20	-	-	-	-	-	-
MAYES (2011)	350	8.3	91	Questionnaire	PBS	Parent	75	-	-	-	-	-	-	50	-	-
MONTAZERI (2020)	118	11	84.7	Questionnaire	RCADS	Self Parent	:	2	:	-	-	-	-	29 24	2	-
MUKADDES (2010)	60	10.65	100	Interview	K-SADS-PL- T	Professional	42	2	7	8	30	2	-	2	5	2
MUSCATELLO (2020)	41	11.49	73.18	Questionnaire	CBCL	Parent	-	-	-	-	-	-	-	39	-	-
OOI (2011)	71	10.24	78.7	Questionnaire	CBCL	Parent	34	-	-	-	-	-	-	31	-	-
ORINSTEIN (2015)	42	13.9	90.47	Interview	K-SADS-PL	Professional	-	5	5	0	14	0	-	-	7	-
OZSIVADJIAN (2014)	30	13	100	Questionnaire	SCAS-PV	Parent	57	-	-	-	-	-	-	-	-	-
PANDOLFI (2014)	76	12	86.84	Interview	K-SADS-PL	Professional	46	-	-	-	-	-	-	20	-	-

PATEL (2017)	25	15	96	Interview	ACI-PL	Professional	28	-	-	-	-	-	-	8	-	-
PFEIFFER (2005)	50	9.8	84	Questionnaire	RCMAS	Parent	10	-	-	-	-	-	-	12	-	-
QUEK (2012)	60	15.47	73	Questionnaire	SCAS-CV	Self	27	-	-	-	-	-	-	11	-	-
RICHDALE (2014)	27	15.05	82	Questionnaire	DASS-21	Self	19	-	-	-	-	-	-	37	-	-
RZEPECKA (2011)	167	Not reported	82	Questionnaire	SCAS-PV	Parent	46	-	-	-	-	-	-	-	-	-
SCHWARTMAN (2021)	125	11.4	74.4	Questionnaire	CDI	Self	-	-	-	-	-	-	-	40	-	-
SIMONOFF (2008)	112	11.5	87.5	Interview	CAPA	Professional	42	13	29	0	9	10	8	2	1	0
SNOW (2011)	51	5.4	82	Questionnaire	CBCL	Parent	24	-	-	-	-	-	-	16	-	-
SOH (2020)	91	14	86.8	Interview	MINI-KID	Professional	23	11	10	2	3	3	3	-	-	-
SOLOMON (2012)	40	12.22	50	Questionnaire	CDI	Professional	-	-	-	-	-	-	-	25	-	-
STRANG (2012)	95	11.67	86	Questionnaire	CBCL	Parent	56	-	-	-	-	-	-	45	-	-
TAYLOR (2016)	36	18.7	85.3	Interview	K-SADS-PL	Professional	14	6	-	-	8	-	-	-	14	6
ULJAREVIC (2017)	71	18.71	69	Interview	DSM-5- ADDS	Professional	41	-	-	-	-	-	-	-	-	-
VASA (2018)	57	10.94	82.5	Interview	DISC-IV	Professional	35	9	19	2	-	-	-	-	-	-
VERHEIJ (2015)	74	16	88	Interview	DISC-IV	Professional	31	4	1	4	26	0	3	-	11	0
VICKERSTAFF (2007)	22	11.86	86.3	Questionnaire	CDI	Parent	-	-	-	-	-	-	-	27	-	-
WHITEHOUSE (2009)	35	14.2	80	Questionnaire	CES-D	Self	-	-	-	-	-	-	-	66	-	-
WILLIAMS (2015)	109	9.74	73	Questionnaire	CBCL	Parent	75	-	-	-	-	-	-	-	-	-
ZACHOR (2020)	57	11	Not reported	Questionnaire	SCARED	Parent	61	-	-	-	-	-	-	-	-	-

^a Measure Abbreviations: SCICA, Semi-structured clinical interview for children and adolescents; SAS-A, Social anxiety scale for adolescents; SCARED, Screen for Child Anxiety-related Emotional Disorders; SAPPA, Schedule for Assessment of Psychiatric Problems Associated with Autism; LSAS, Liebowitz Social Anxiety Scale; DSIC-IV-P, Diagnostic Interview Schedule for Children – Parent version; DSM-5 GAD-D, The Diagnostic and Statistical Manual of Mental Disorders-5 Generalized Anxiety Disorder Dimensional Scale; K-SADS-PL-T, Schedule for Affective Disorders and Schizophrenia for School Age Children-Present and Lifetime Version-Turkish Version; K-SADS-PL, Schedule for Affective Disorders and Schizophrenia for School Age Children-Present and Lifetime Version; SCAC-PV, Spence Children's Anxiety Scale - Parent Version; CSI-4, Child Symptom Inventory-4; BYI, Beck Youth Inventory; ADIS, Anxiety Disorders Interview Schedule; CASI-4, Child and Adolescent Symptom Inventory; SPAI-C, Social Phobia and Anxiety Inventory; ACI-PL, Autism Comorbidity Interview - Present and Lifetime; SCAC-CV, Spence Children's Anxiety Scale - Child Version; PBS, Paediatric Behaviour Scale; CBCL, Child Behaviour Checklist; RCMAS, Revised Children's Manifest Anxiety Scale; DASS-21, Depression, Anxiety and Stress Scale; CAPA, Child and Adolescent Psychiatric Assessment; MINI-KID, Mini-International Neuropsychiatric Interview for Children and Adolescents; DSM-5-ADDS, Diagnostic and Statistical Manual of Mental Disorders-5 Anxiety Disorder Dimensional Scale (DSM-5-ADDS); DISC-IV, Diagnostic Interview Schedule for Children; BASC-2, Behaviour Assessment System for Children Second Edition; RCADS, Revised Child Anxiety and Depression Scale; ACI-PL, Autism Comorbidity Interview - Present and Lifetime; CDI, Child Depression inventory (CDI) - Youth Report; CES-D, Centre for Epidemiological Studies Depression scale; CASI-GAD-D, Child and Adolescent Symptom Inventory Generalised Anxiety Subscale and Depressive Disorder Subscale; Kid-SCID, Structured Clinical Interview for DSM-IV Diagnoses .^bDisorder Abbreviations: UA (unspecified anxiety), G (generalised anxiety), So (social anxiety), Sp (specific phobia), P (panic), A (Agoraphobia), UD (unspecified depression), M (major depressive disorder), D (Dysthymia).

Defining Problematic Variance

A degree of variance across study findings is expected in a meta-analysis, owing to methodological differences, measurement error or uncontrolled individual difference factors (Higgins, Thomspon, Deeks & Altman, 2003). However, it is important to assess the extent of the variance and how it effects the meta-analysis, as it can hinder generalisability of conclusions. Greater difference indicates variation in effect that cannot be attributed to true variation in prevalence in the general population. Thus, to distinguish genuine difference (heterogeneity) from that due to chance, Higgins I² was calculated. Higgins I² value greater than 75% with an associated probability value of p < 0.01 was used as a marker of problematic heterogeneity (Higgins et al., 2022). Where problematic heterogeneity was observed (I² value > 75%), further analysis sought to identify sources of heterogeneity; this included "leave-one-out" analysis and reviewing Baujat plots (Baujat, Pignon, & Hill, 2002) to assess the impact of discrepant and disproportionately influential studies. Identified studies were then re-examined with a view to removal from the meta-analysis if substantive reason could be identified. Subsequent change in weighted average effect size (i.e., influence) and change in heterogeneity (i.e., discrepancy) was recorded.

Study Design Hierarchy and Risk of Bias Assessment

Each study received a score ranging from 10 to 40 for study design with the highest score (40) being given to prospective case cohort studies, as this design is best suited to a meta-analysis of prevalence rates. Designs, descriptions and corresponding scores are defined in Table 4.

A set of quality criteria were developed to assess any risk of bias within the literature. The quality criteria were adapted from existing risk of bias frameworks, including the Cochrane Collaboration Risk of Bias Tool (Higgins et al., 2011) and the Risk of Bias Assessment Tool for Nonrandomised Studies (Kim et al., 2013). The current framework assesses risk of bias in six domains: selection bias, performance bias, detection bias, statistical bias, reporting bias and generalisation. The risk of bias in the six domains and the criteria for low, unclear or high risk are described in Table 4. Risk ratings were equally weighted and associated with scores of zero to two (high risk= 0, unclear risk= 1, low risk = 2). An overall risk of bias score was achieved by adding the scores across the six domains together. Then, a study quality score was calculated for each study by adding the study design score to the total risk of bias score. The maximum possible score was 52, scores were then converted into percentages by dividing the overall quality score by 52 and multiplying by 100. The same quality assessment method was conducted for each study, no separate assessment was conducted for studies including more than one rating type (parent, self, professional). No extra points were awarded in the risk of bias assessment for including more than one rating type in the study. The application of the criteria is reported in Table 5.

The reliability of the risk of bias was checked using an approximately 10% random sample. These 6 studies were cross-validated by a second rater. Initial inter-rater agreement was 83.3%. There were only minor disagreements in ratings and a consensus was reached following discussion. The risk of bias criteria were updated to reflect the consensus position.

Table 4

Study Design	Quality Score	Description
Prospective Case	40	Cohort Study (prospective) is a study of a group of individuals, some of whom are exposed to a
Conort Study		variable of interest (e.g., ASD), in which participants are followed up over time to determine who
		develops the outcome of interest (anxiety/depression) and whether the outcome is associated with
		the exposure. For example, following a group of people with ASD to see if they develop
Determinetion	20	anxiety/depression)
Retrospective	30	Conort Study (retrospective) is when data is gathered for a conort that was formed sometime in the
Case Conort		you are studying the risk factor and see if you can associate a disease to it. Individuals split by
Study		You are studying the fisk factor and see if you can associate a disease to it. Individuals split by
		exposure. For example, people, with ASD answering questions about mood to see if they have
Case Control	20	Case Control Study is a study in which nation who already have a specific condition or outcome
Study	20	(e.g. ASD) are compared with people who do not (neurotypical). Researchers look back in time
Study		(retrospective) to identify possible exposures (e.g., anxiety/depression). They often rely on medical
		records and patient recall for data collection.
Cross-sectional	10	Cross-Sectional Study is the observation of a defined population at a single point in time or during
Study		a specific time interval to examine associations between the outcomes and exposure to
		interventions. Exposure and outcome are determined simultaneously. Often rely on data originally
		collected for other purposes. E.g., selecting a group from the population and looking at prevalence
		of anxiety/depression and seeing whether there is co-morbid ASD or vice-versa.
Domain	Details	Risk of Bias
Selection Bias	Selection bias in	High Risk- Convenience sampling used. The characteristics of the study population are not
	epidemiological	reported. Recruited from one clinical setting. Sample described as 'clinical' or 'treatment seeking'
	studies occurs	with no further information.
	when there is a	
	systematic	Unclear Risk-An attempt was made to recruit a representative sample. Participant characteristics
	difference between	are reported but not clearly or in sufficient detail e.g., alludes to medication use in the sample but
	the characteristics	does not report existing conditions.
	of those selected	
	for the study and	Low Risk-The characteristics of the study population are clearly described and without evidence of
	those who are not.	bias. The source population is well described (ideally non-clinical), and the study reports the

Study Design Hierarchy and Domains of Risk of Bias and the Criteria for Ratings of Low, Unclear or High Risk

	Randomisation cannot be applied to observational studies or within- subject intervention designs and the effects of selection bias in these studies should be considered and, potentially, penalised.	characteristics of the sample e.g., learning disability, co-morbid diagnoses. The recruitment method is clearly reported and well defined. Attempts have been made to recruit a representative sample e.g., <40% of participants have an existing psychiatric diagnosis.
Performance Bias	Performance bias refers to systematic differences between/within groups in the participants motivation to complete the study.	 High Risk- Participants were rewarded for their participation in the study. Participants were told which questionnaires they were completing and why and any proposed hypotheses. Unclear Risk- There is some indication that information was given to participants that could have affected their responses but this is not definitively clear/adequately described. Low Risk-Information and procedures are provided in a way that does not differentially motivate participants, or exact procedures are not fully described but no obvious attempts to influence participant responses are apparent.
Detection Bias	Detection bias refers to whether the design of the study is optimised to detect the effect in question. The research may be poorly designed	High Risk - The outcome measures were implemented differently across participants. The outcome measures used had poor reliability and validity e.g., Cronbach's Alpha < 0.6. and/or test/retest reliability < 0.6. States that the measure has been translated but does not detail how this was conducted or clear problems in translation.Unclear Risk- Information regarding the outcome measures are either not reported or not clearly reported e.g., definition, validity, reliability, cut-off. Cronbach's Alpha for outcome measures is between 0.6 and 0.7. Test-retest reliability for outcome measures is between .6 and .7. It is not

	and question may be unclear.	clear if the measure was implemented consistently across all participants. The research question is unclear. The measure is translated and does not adequately report psychometric properties.
	Detection bias in the context of this meta-analysis refers to systematic differences between participants in how outcomes are determined and the power of outcome measures to detect an effect. For instance, lack of information about psychometrics, use of translated measures that have	Low Risk- The outcome measures are clearly defined, valid and reliable, and are implemented consistently across all participants.
Statistical Bias	Bias resulting from the (inappropriate) statistical treatment of the data e.g., multiple comparisons made without making adjustments to the alpha level.	 High Risk- Statistics were not reported or the wrong statistical test was used and it was not appropriate for the study design. Unclear Risk – Unclear what statistical test was used. Confidence intervals or exact p-values for effect estimates were not reported and could not be calculated. Low Risk – Appropriate statistical testing was used. Confidence intervals or exact p-values for effect estimates were given or possible to calculate.
Reporting Bias	Reporting bias refers to	High Risk – Has not reported full outcome measures that are stated in the method section/ reported only a subsample of results/only significant results/ not reported the measure as it should be.

	systematic differences between reported and unreported findings. Within a published report those analyses with statistically significant differences between intervention groups are more likely to be reported than non- significant	Unclear Risk – Not all descriptive and/or summary statistics are presented. There is a description (narrative) in the results but it does not record statistics. Reported more than one correlation. Low Risk – Reported all results of measures as outlined in the method.
Generalisation	differences. Generalisability describes the extent to which research findings can be applied to settings other than that in which they were originally tested. Any differences between the study participants and those persons to whom the review is applicable.	 High Risk- Small sample with or without idiosyncratic features. High percentage (over 85%) of sample is represented by one gender and people with/without LD excluded. The sample size is not adequate to detect an effect. Unclear Risk- Sufficient sample for generalisation but with some idiosyncratic features. A sample size justification, estimate and power analysis were not provided Low Risk- Sufficient sample for generalisation and representative of target population. A sample size justification, estimate and power analysis were provided. The sample size is adequate to detect an effect

Table 5

Study Name	Year	Study Design ^a	Selection Bias	Performance Bias	Detection Bias	Statistical Bias	Reporting Bias	Generalisability Bias	Overall Quality Rating (%)
Amr et al.	2012	RCC	Unclear risk	Low risk	Low risk	Low risk	Low risk	Low risk	79
Bellini et al.	2004	RCC	Low risk	High risk	High risk	High risk	Low risk	High risk	65
Ben-Itzchak et al.	2020	PCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	High risk	94
Bitsika & Sharpley	2015	CC	Low risk	Unclear risk	Unclear risk	Low risk	Low risk	High risk	54
Bitsika et al.	2015b	RCC	Low risk	Low risk	Low risk	Low risk	Low risk	High risk	77
Bitsika et al.	2016	RCC	Low risk	Low risk	Unclear risk	Low risk	Unclear risk	High risk	73
Bradley et al.	2011	CC	Unclear risk	Low risk	Unclear risk	Low risk	Low risk	High risk	54
Briot et al.	2020	CC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Low risk	60
Cai et al.	2019	CS	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	38
Coskun et al.	2020	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	77
De Bruin et al.	2007	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	77
Deckers et al.	2017	CC	Unclear risk	Unclear risk	High risk	Low risk	Low risk	Unclear risk	52
Demirkaya et al.	2016	PCC	Low risk	Low risk	Low risk	Low risk	Low risk	High risk	96
Den Houting et al.	2019	RCC	Low risk	High risk	High risk	Low risk	Low risk	High risk	69
Gadow et al.	2005	CC	Unclear risk	Low risk	Unclear risk	Unclear risk	Unclear risk	High risk	50
Gjevik et al.	2011	RCC	Low risk	Low risk	Low risk	Unclear risk	Low risk	High risk	75
Hebron et al.	2014	RCC	Low risk	Low risk	Low risk	Low risk	Low risk	Unclear risk	79
Herrington et al.	2017	RCC	Unclear risk	Low risk	Low risk	Low risk	Low risk	Low risk	79
Hessl et al.	2021	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Low risk	79
Kaat et al.	2013	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	77
Kerns et al.	2014	RCC	Low risk	High risk	Low risk	Low risk	Low risk	Low risk	77
Kuusikko et al.	2008	CC	Low risk	Low risk	High risk	Low risk	Low risk	High risk	54
Leyfer et al.	2006	RCC	Low risk	Low risk	High risk	Low risk	Low risk	High risk	73
Lopata et al.	2010	CC	Low risk	Low risk	Low risk	Low risk	Low risk	High risk	58
Magiati et al.	2014	RCC	Low risk	High risk	Unclear risk	Unclear risk	Unclear risk	High risk	67
Mayes et al.	2011	CC	Unclear risk	Low risk	Low risk	Low risk	Low risk	Unclear risk	58
Magiati et al.	2016	RCC	Low risk	High risk	Unclear risk	Low risk	Low risk	Low risk	75
Montazeri et al.	2020	CC	Low risk	Low risk	Low risk	Low risk	Low risk	Unclear risk	60
Mukaddes et al.	2010	RCC	Unclear risk	Low risk	Unclear risk	Low risk	Low risk	High risk	73
Muscatello et al.	2020	CC	Low risk	Low risk	Low risk	Unclear risk	Unclear risk	Unclear risk	56
Ooi et al.	2011	RCC	Unclear risk	Low risk	High risk	Unclear risk	Low risk	High risk	69

Ratings of Risk of Bias with Red Indicating High risk of bias, Amber Indicating Unclear Risk and Green Indicating Low Risk

Orinstein et al.	2015	RCC	Low risk	Low risk	Low risk	High risk	Low risk	Unclear risk	75
Ozsivadjian et al.	2014	CC	Unclear risk	Unclear risk	High risk	Low risk	Unclear risk	High risk	48
Pandolfi et al.	2014	RCC	Unclear risk	Low risk	Unclear risk	Low risk	Low risk	High risk	73
Pfeiffer et al.	2005	RCC	High risk	Low risk	Low risk	Low risk	Low risk	High risk	73
Quek et al.	2012	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	77
Richdale et al.	2014	CC	Low risk	Unclear risk	Low risk	Low risk	Low risk	High risk	56
Schwartman et al.	2021	CC	Unclear risk	Low risk	High risk	Low risk	Low risk	High risk	52
Simonoff et al.	2008	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	77
Snow et al.	2011	RCC	Low risk	High risk	77				
Soh et al.	2020	RCC	High risk	Low risk	Low risk	Low risk	Low risk	Unclear risk	75
Solomon et al.	2012	CC	Unclear risk	Low risk	High risk	Unclear risk	Low risk	Unclear risk	52
Taylor et al.	2016	RCC	Low risk	Unclear risk	79				
Uljarevic et al.	2017	RCC	Unclear risk	Low risk	Low risk	Low risk	Low risk	Unclear risk	77
Vasa et al.	2018	CC	Low risk	High risk	58				
Verheij et al.	2015	PCC	Low risk	High risk	96				
Vickerstaff et al.	2007	RCC	Unclear risk	Low risk	Low risk	High risk	Low risk	High risk	71
Whitehouse et al.	2009	CC	Low risk	Low risk	High risk	Low risk	Low risk	High risk	54
Zachor et al.	2020	PCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	Unclear risk	96
den Houting et al.	2018	RCC	Unclear risk	Low risk	Unclear risk	Low risk	Unclear risk	Unclear risk	73
Johnston et al.	2017	CC	Low risk	High risk	Low risk	Low risk	Low risk	Unclear risk	56
Kaat	2015	RCC	Unclear risk	High risk	Unclear risk	Unclear risk	Low risk	Unclear risk	69
Patel et al.	2017	CC	Low risk	Low risk	Low risk	Low risk	Unclear risk	High risk	56
Rzepecka et al.	2011	RCC	Unclear risk	Low risk	High risk	Low risk	Low risk	Unclear risk	73
Strang et al.	2012	RCC	Low risk	Low risk	Unclear risk	Low risk	Low risk	High risk	75
Williams et al.	2015	RCC	Low risk	Low risk	Unclear risk	Unclear risk	Low risk	Low risk	69

^aAbbreviations Retrospective case cohort study (RCC), prospective case cohort study (PCC), case control study (CC), cross-sectional study (CS)

Selection Bias

Risk of selection bias was low across studies. Seventeen studies were rated as unclear risk of bias, two with high risk of bias and 37 rated as low risk of bias. Low-risk studies described participant characteristics sufficiently and attempted to recruit a representative sample. If participants were on medication or existing diagnoses, these were well-described. Unclear-risk studies often used convenience sampling, recruiting from a clinical setting. High-risk studies included large numbers of participants with existing diagnoses of anxiety or depression and participant characteristics were not clearly described (Pfeiffer et al., 2005; Soh et al., 2020).

Performance Bias

Performance bias was mostly low across studies. Four studies were rated as unclearrisk, seven were rated as high-risk and 45 were rated as low-risk. Unclear-risk studies were associated with unclear reporting about the information provided to participants about study hypotheses and aims; therefore, it is not known how this could have affected performance (Bitsika & Sharpley, 2015; Deckers et al., 2017; Ozsivadjian et al., 2014; Richdale et al., 2014). In the studies rated as high-risk it was clear that participants were rewarded for participation or the information provided likely affected responses (Bellini, 2004; den Houting et al., 2019; Johnston & Iarocci, 2017; Kaat & Lecavalier, 2015; Kerns et al., 2014; Magiati et al., 2014).

Detection Bias

A roughly equal number of studies were rated as unclear-risk and low-risk of detection bias (22 studies and 23 studies respectively). Unclear-risk was associated with not reporting psychometric properties or cut-offs for the measures used. Eleven studies were rated as highrisk. High-risk studies used outcome measures that had poor reliability and/or validity (Cronbach's Alpha < 0.6, test/retest reliability < 0.6) or that had been translated without validation. Use of such measures threatens reliability and validity of prevalence rates.

Statistical Bias

Most studies were rated as low risk of statistical bias. Eight studies were rated as unclear-risk due to lack of detail about statistical analysis. Three studies were rated as highrisk due to obvious inappropriate treatment of data; e.g., not making adjustments for multiple comparisons (Bellini, 2004; Orinstein et al., 2015; Vickerstaff et al., 2007).

Reporting Bias

No studies were rated as high-risk and only seven were rated as unclear-risk. Unclear risk studies failed to report full results (Bitsika et al., 2016; den Houting et al., 2018; Gadow et al., 2005; Magiati et al., 2014; Muscatello et al., 2020; Ozsivadjian et al., 2014; Patel et al., 2017).

Generalisability Bias

The most common risk of bias related to generalisability. Only seven studies obtained a low-risk rating. Most studies (n = 28) were rated high-risk and 21 studies were rated unclear-risk. All samples were predominantly male. Many recruited from clinical settings or one recruitment site, some had small samples and excluded participants with lower intellectual ability, making it difficult to generalise prevalence rates to females, people with intellectual difficulties and non-clinical populations.

Summary

Overall, bias was varied across studies and no study was rated low-risk in every area. Generalisability and detection bias were most prevalent across studies. Unclear or high-risk studies were not excluded from the meta-analysis. However, if influential or discrepant, risk of bias was considered again with a view to exclusion if sufficient reason was found.

Results

There were two stages of analysis. The first stage calculated the mean effect sizes (prevalence rates) for clinically significant anxiety and depression of unspecified type, and each of the DSM-5 anxiety and depressive disorders (generalised anxiety disorder, agoraphobia, specific phobia, social anxiety disorder, panic disorder, separation anxiety disorder, major depressive disorder, dysthymia). At this stage of analysis, the impact of influential studies was assessed to determine whether there were any outliers and whether these studies should be excluded from further analysis.

The second stage of analysis explored whether prevalence rates were affected by study characteristics (i.e., design, measurement method, recruitment location) or participant characteristics (i.e., age, sex, participants with intellectual disability included or excluded). Analyses were conducted separately for each type of disorder. It was expected that prevalence rates would differ according to who was rating anxiety or depression (self, parent or professional); therefore, subgroup analysis of rating type was conducted independently of evidence of heterogeneity and the results are presented alongside pooled prevalence estimates. All other subgroup analyses were conducted based on high levels of heterogeneity and are presented in the subsequent sections.

Selection of the Meta-Analytic Model

The distribution of primary study effects is shown in Figure 2. The between studies variance (tau²) was estimated using the random effects model calculated using the restricted maximum-likelihood estimator. There was no evidence of marked non-normality in the distribution of anxiety or depression prevalence within each of the different types of anxiety and depression. This indicated that the use of the random effects model calculated using the restricted maximum-likelihood estimator was appropriate for this meta-analysis.


QQ Plot of the Distribution of Prevalence of the Different Types of Anxiety and Depression within the Primary Studies

Results of Meta-Analysis: Anxiety

Results are displayed in Table 6. There was a statistically significant difference between the different types of anxiety disorder, $X^2(6, k = 49) = 166.95, p < .0001$; with substantial heterogeneity ($I^2 = 96\%, t^2 = .0325, p < .0001$). The 40 studies reporting the presence of unspecified anxiety disorder had a weighted average prevalence rate of 39.61% (k= 40, 95% CI 33.74%, 45.48%), marked heterogeneity between the primary studies was evident ($I^2 = 95\%, t^2 = .0368, p < .01$). The highest prevalence for a specific anxiety disorder was seen in specific phobia, at 23.36% (k = 14, 95% CI 15.89%, 30.82%). Again, significant heterogeneity was observed ($I^2 = 91\%, t^2 = .0171, p < .01$). Heterogeneity above 75% was also noted among studies that reported on generalised ($I^2 = 93\%, t^2 = .011, p < .01$), social (I^2 = 85%, $t^2 = .0099, p < .01$) and separation anxiety disorders ($I^2 = 89\%, t^2 = .010, p < .01$). Heterogeneity below the threshold of 75% was reported for panic disorder ($I^2 = 72\%, t^2 = .0009, p < .01$) and agoraphobia ($I^2 = 57\%, t^2 = .0008, p < .01$).

Effect of Rating Type

As shown in Table 6, the variation between prevalence estimates of generalised, social and separation anxiety according to rating type was statistically significant. For generalised anxiety disorder (Figure 3), the parent-rated prevalence estimate was highest, whilst professional-rated prevalence was lowest and self-rated prevalence was in the mid-range, $X^2(2, k = 24) = 13.34, p < .001$. For social anxiety disorder (Figure 4) the self-rated prevalence estimate was highest, followed by parent, then professional ratings, $X^2(2, k = 23) =$ 9.45, p < .001. For separation anxiety disorder (Figure 5), parent and self-rated prevalence estimates were highest, whilst the professional-rated prevalence estimate was much lower, $X^2(2, k = 20) = 9.52, p < .001$). No significant differences regarding rating type were identified for specific phobia, agoraphobia or panic disorder; however, these disorders had fewer data points contributing to prevalence estimates.

Irrespective of who rated anxiety, prevalence of unspecified anxiety, generalised anxiety, social anxiety and specific phobia, considerably exceeded the global prevalence of anxiety for young people, estimated to be 6.5%; represented by the central line on Figures 3, 4 and 5 (Polanczyk, Salum, Sugaya, Caye & Rohde, 2015).

Table 6

Summary of Anxiety Disorder Overall Prevalence and Prevalence According to Rating Type

	Studies ^a	Data Points ^a	PR	%	Lower 95%	Upper 95%	tau ²	I ²	<i>X</i> ²
					CI	CI			
UNSPECIFIED ANXIETY									
DISORDER	40	45	.3961	39.61	.0271	.4548	.0368	95%*	
Rated by parent	19	19	.4667	46.67	.3669	.5665	.0461	97%*	
Rated by professional	17	19	.3537	35.37	.2778	.4296	.0251	91%*	
Rated by self	7	7	.3069	30.69	.1798	.4339	.0241	87%*	
Subgroup differences							.0368	95%*	4.63
OPE OFFICE PROPERTY			2226	22.24	4 500	2002		0.40/.4	
SPECIFIC PHOBIA	14	14	.2336	23.36	.1589	.3082	.0171	91%*	
Rated by parent									
Rated by professional	14	14	.2336	23.36	.1589	.3082	.0171	91%*	
Rated by self									
GENERALISED ANXIETY	24	29	.1450	14.5	.1040	.1860	.0110	93%*	
Rated by parent	7	7	.2527	25.27	.1821	.3233	.0070	81%*	
Rated by professional	19	19	.1026	10.26	.0636	.1417	.0061	89%*	
Rated by self	3	3	.1582	15.82	0190	.3353	.0229	97%*	
Subgroup differences							.0110	93%*	13.34*
SOCIAL ANXIETY	23	25	.1488	14.88	.1061	.1915	.0099	85%*	
Rated by parent	6	6	.2148	21.48	.1388	.2907	.0066	83%*	
Rated by professional	15	15	.0978	9.78	.0652	.1303	.0029	78%*	
Rated by self	4	4	.2544	25.44	.0512	.4575	.0395	92%*	
Subgroup differences							.0099	85%*	9.45*
								0.00/ 4	
SEPARATION ANXIETY	20	22	.1076	10.76	.0627	.1526	.0100	89%*	
Rated by parent	6	6	.2094	20.94	.0877	.3311	.0208	94%*	
Rated by professional	14	14	.0499	4.99	.0234	.0763	.0018	74%*	
Rated by self	2	2	.2007	20.07	.0459	.3555	.0087	69%	
Subgroup differences							.0100	89%*	9.52*
AGORAPHOBIA	5	5	.0594	5.94	.3374	.0917	.0008	57%	
Rated by parent	1	1	.1392	13.92	.0629	.2156			
Rated by professional	4	4	.0458	4.58	.0226	.0690	<.000 1	21%	
Rated by self	0	0							
Subgroup differences							.0008	57%	5.27
BANKS BISSBEE		4.0						=00//f	
PANIC DISORDER	11	12	.0318	3.18	.0112	.0525	.0009	72%*	
Kated by parent	3	3	.1168	11.68	0492	.2829	.0198	90%*	
Kated by professional	8	8	.0113	1.13	.0032	.0194	<.000 1	44%	
Rated by self	1	1	.1304	13.04	.0331	.2278			
Subgroup differences							.0009	72%*	7.24

^aFigures in the column titled 'data points' represent how many data points there were. This is different to number of 'studies' because some studies included more than one rater (e.g., self, parent, professional).

*Significant at *p* <.01 level

**Significant at p<.0001 level

Forest Plot Showing Significant Difference in Generalised Anxiety Disorder Prevalence among autistic young people according to Rater

Study	те	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
Brent			LC 3				
Parent Bop Itzebak et al. 2020	0.19	0.0402		0.19	10.08.0.281	1 09/	2 20/
Bitsika et al. 2016	0.43	0.0405		0.43	[0.35: 0.51]	1.5%	3.5%
Gadow Devincent Pomerov & Azizian 2005	0.24	0.0253		0.40	[0.19: 0.29]	3.9%	3.8%
Kaat Kenneth Gadow & Lecavalier 2013	0.18	0.0360		0.18	[0.11:0.25]	1.9%	3.6%
Magiati et al. 2014	0.29	0.0736		0.29	[0.15: 0.43]	0.5%	2 7%
Magiati et al. 2016	0.19	0.0251		0.19	[0.14:0.24]	3.9%	3.8%
Kaat et al. 2015	0.28	0.0664		0.28	[0.15: 0.41]	0.6%	2.8%
Fixed effect model	0.20	0.0001		0.24	[0.21: 0.26]	13.3%	
Random effects model				0.25	0.18: 0.321		23.3%
Prediction interval				0.20	0.02: 0.491		
Heterogeneity: $I^2 = 81\%$, $\tau^2 = 0.0070$, $p < 0.01$							
Brafasalanal							
Amr. et al. 2012	0.10	0.0297		0.10	10.02.0.191	4 70/	2 59/
Amretal. 2012 Bitaika at al. 2015	0.10	0.0367		0.10	[0.02; 0.18]	1.770	3.5%
Bitsika et al. 2015 Bradley et al. 2014	0.32	0.0412		0.32	[0.24, 0.40]	1.5%	3.470
Coskup Heidigi Alaek & Karayaamurlu 2020	0.00	0.0362		0.06	[-0.02; 0.13]	0.0%	3.5%
Coskun, Hajdini, Alnak & Karayagmurlu 2020	0.20	0.0516		0.20	[0.10; 0.30]	0.9%	3.2%
Demideava Tutkuskerdes 8 Mukaddas 2016	0.05	0.0231		0.05	[0.01; 0.10]	4.0%	3.0%
Gadow, Devincent, Remercy & Azizian 2005	0.05	0.0306		0.05	[-0.01; 0.11]	2.0%	3.7%
Gadow, Devincent, Fomeroy & Azizian 2005	0.24	0.0252		0.24	[0.15, 0.29]	25 19/	3.0%
Hosel et al. 2021	0.01	0.0099		0.01	[-0.01, 0.03]	25.1%	3.9%
Kaat Kannath Gadow & Lacavaliar 2013	0.14	0.0323		0.23	[0.08:0.20]	2 4%	3.6%
Kerns et al. 2014	0.20	0.1265		0.14	[-0.05: 0.45]	0.2%	1.6%
Levfer et al. 2006	0.02	0.0241		0.02	[-0.02: 0.07]	4 3%	3.8%
Mukaddes 2010	0.02	0.0165		0.02	[-0.02: 0.05]	9.1%	3.9%
Orinstein 2015	0.05	0.0329		0.02	[-0.02: 0.11]	2 3%	3.6%
Simonoff 2008	0.13	0.0322		0.13	[0.07: 0.20]	2.4%	3.6%
Soh 2020	0.11	0.0328		0.11	[0.05: 0.17]	2.3%	3.6%
Taylor 2016	0.06	0.0382		0.06	[-0.02: 0.13]	1.7%	3.5%
Vasa 2018	0.09	0.0375		0.09	[0.01:0.16]	1.8%	3.5%
Verheii 2015	0.04	0.0229		0.04	[0.00: 0.09]	4.7%	3.8%
Fixed effect model	0.0.1	010220	0	0.06	[0.05: 0.07]	73.7%	
Random effects model				0.10	[0.06: 0.14]		66.3%
Prediction interval					[-0.07: 0.27]		
Heterogeneity: $I^2 = 89\%$, $\tau^2 = 0.0061$, $p < 0.01$							
Self							
Bitsika et al. 2016	0.31	0.0379		0.31	[0.24:0.39]	1.7%	3.5%
Magiati et al. 2014	0.16	0.0592		0.16	[0.04: 0.27]	0.7%	3.0%
Kaat et al. 2015	0.01	0.0153		0.01	[-0.02: 0.04]	10.6%	3.9%
Fixed effect model	0.0.			0.06	[0.03: 0.09]	13.0%	
Random effects model				0.16	[-0.02: 0.34]		10.4%
Prediction interval					[-2.08; 2.40]		
Heterogeneity: $I^2 = 97\%$, $\tau^2 = 0.0229$, $p < 0.01$							
Fixed effect model			•	0.08	[0.07; 0.09]	100.0%	100.000
Random effects model				0.15	[0.10; 0.19]		100.0%
Prediction Interval					[-0.07; 0.36]		
Heterogeneity: $T = 93\%$, $\tau^{*} = 0.0110$, $p < 0.01$ Test for overall effect (random effects): $\tau = 6.94$ ($p < 0.01$)		0.2	0 02 01	0.6			
Test for substance differences (readers effects): $z = 0.94$ ($p < 0.01$)		-0.2	0 0.2 0.4	0.6			
restroi subgroup differences (random effects): $\chi_2 = 13.34$, df = 2 (p < 0.0							

Note. The central line represents the global anxiety disorder prevalence among typically developing young people (Polancyzk et al., 2015)

Forest Plot Showing Significant Difference in Social Anxiety Disorder Prevalence Among Young Autistic People according to Rater

Study	те	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
Parent Ben-Itzchak et al. 2020 Briot et al. 2020 Kaat, Kenneth, Gadow & Lecavalier 2013 Magiati et al. 2014 Magiati et al. 2015 Kaat et al. 2015 Fixed effect model Random effects model Prediction interval Heterogeneity: $l^2 = 83\%$, $\tau^2 = 0.0066$, $p < 0.01$	0.28 0.19 0.23 0.21 0.09 0.37	0.0574 0.0441 0.0390 0.0661 0.0186 0.0712		0.28 0.19 0.23 0.21 0.09 0.37 0.15 0.21	[0.17; 0.39] [0.10; 0.28] [0.15; 0.30] [0.08; 0.34] [0.05; 0.13] [0.23; 0.51] [0.12; 0.18] [0.14; 0.29] [0.03; 0.46]	1.2% 2.0% 2.6% 0.9% 11.4% 0.8% 18.9%	3.6% 4.0% 4.2% 3.3% 4.7% 3.2% 23.0%
ProfessionalCoskun, Hajdini, Alnak & Karayagmurlu 2020Bruin, Ferdinand, Meester & Verheij 2007Demirkaya, Tutkunkardaş & Mukaddes 2016Gadow, Devincent, Pomeroy & Azizian, 2005Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011Hessl et al. 2021Kaat, Kenneth, Gadow & Lecavalier 2013Kerns et al. 2014Leyfer et al. 2006Mukaddes 2010Orinstein 2015Simonoff 2008Soh 2020Vasa 2018Verheij 2015Fixed effect modelRandom effects modelPrediction intervalHeterogeneity: $l^2 = 78\%$, $r^2 = 0.0029, p < 0.01$	0.08 0.12 0.05 0.13 0.07 0.08 0.10 0.07 0.07 0.05 0.29 0.10 0.19 0.01	0.0357 0.0332 0.0306 0.0197 0.0304 0.0399 0.0285 0.0271 0.0322 0.0329 0.0329 0.0431 0.0323 0.0313		0.08 0.12 0.05 0.13 0.07 0.08 0.10 0.07 0.07 0.05 0.29 0.10 0.19 0.01 0.08 0.10	[0.01; 0.15] [0.05; 0.18] [-0.01; 0.11] [0.09; 0.17] [0.01; 0.16] [0.05; 0.16] [-0.05; 0.45] [0.02; 0.13] [0.02; 0.13] [0.02; 0.11] [0.21; 0.38] [0.04; 0.16] [0.09; 0.30] [-0.01; 0.04] [0.06; 0.09] [0.07; 0.13] [-0.02; 0.22]	3.1% 3.6% 4.2% 10.1% 4.3% 2.5% 4.8% 5.4% 3.6% 2.1% 4.0% 1.4% 21.8% 75.0%	4.3% 4.4% 4.6% 4.4% 4.1% 4.5% 4.5% 4.5% 4.4% 4.3% 4.1% 4.4% 3.8% 4.7%
Self Bellini et al. 2004 Kuusikko et al. 2008 Magiati et al. 2014 Kaat et al. 2015 Fixed effect model Prediction interval Heterogeneity: $l^2 = 92\%$, $\tau^2 = 0.0395$, $p < 0.01$ Fixed effect model Random effects model Prediction interval Heterogeneity: $l^2 = 85\%$, $\tau^2 = 0.0099$, $p < 0.01$ Test for overall effect (random effects): $z = 6.82$ ($p < 0.01$) Test for subgroup differences (random effects): $z_a^2 = 9.45$, df = 2 ($p < 0.01$)	0.49 0.39 0.11 0.07	0.0781 0.0663 0.0498 0.0364		0.49 0.39 0.11 0.07 0.17 0.25 0.10 0.15	[0.33; 0.64] [0.26; 0.52] [0.01; 0.20] [0.01; 0.14] [0.12; 0.22] [0.05; 0.46] [0.05; 0.46] [0.07; 1.22] [0.08; 0.11] [0.11; 0.19] [-0.06; 0.36]	0.6% 0.9% 1.6% 3.0% 6.1% 100.0%	3.0% 3.3% 4.2%

Note. The central line represents the global anxiety disorder prevalence among typically developing young people (Polancyzk et al., 2015)

Forest Plot Showing Significant Difference in Separation Anxiety Disorder Prevalence Among Young Autistic People according to Rater

Study	TE	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
						(,	()
Parent	0.00	0.0005		0.00	10.07:0.501	0.40/	2.0%
Ben-Itzchak et al. 2020	0.39	0.0625		0.39	[0.27; 0.52]	0.4%	3.8%
Gadow, Devincent, Pomeroy & Azizian 2005	0.07	0.0146		0.07	[0.04; 0.09]	6.9%	5.2%
Kaat, Kenneth, Gadow & Lecavaller 2013 Magiati at al. 2014	0.03	0.0149		0.03	[0.00; 0.06]	0.0%	D.∠%
Magiati et al. 2014 Magiati et al. 2016	0.32	0.0754		0.32	[0.17, 0.46]	2.2%	J. 9%
Magiali et al. 2010 Kaat et al. 2015	0.20	0.0257		0.20	[0.15, 0.25]	0.3%	4.9%
Fixed effect model	0.55	0.0091		0.00	[0.13, 0.40]	16.6%	5.0 %
Random effects model				0.00	[0.09:0.33]	10.0 /0	26.0%
Prediction interval				0.21	[-0.23: 0.65]		20.070
Heterogeneity: $I^2 = 94\%$, $\tau^2 = 0.0208$, $p < 0.01$					[0.20, 0.00]		
Professional							
Amr et al. 2012	0.08	0.0357		0.08	[0.01:0.15]	1 2%	4 7%
Coskun Haidini Alnak & Karayagmurlu 2020	0.00	0.0559		0.00	[0.14:0.36]	0.5%	4.7%
Bruin Ferdinand Meester & Verheii 2007	0.20	0.0000		0.20	[0.03:0.14]	1.8%	4.0%
Demirkava Tutkunkardas & Mukaddes 2016	0.02	0.0180	_	0.02	[-0.02:0.05]	4.5%	5.1%
Gievik, Eldevik Eiæran-Granumi & Sponheim 2011	0.01	0.0099	-	0.01	[-0.01: 0.03]	14.9%	5.2%
Hessl et al. 2021	0.08	0.0399		0.08	[0.01: 0.16]	0.9%	4.6%
Kerns et al. 2014	0.10	0.0949		0.10	[-0.09; 0.29]	0.2%	2.8%
Leyfer et al. 2006	0.12	0.0322	i + i+	0.12	[0.06; 0.18]	1.4%	4.8%
Mukaddes 2010	0.08	0.0357		0.08	[0.01; 0.15]	1.2%	4.7%
Orinstein 2015	0.01	0.0167		0.01	[-0.02; 0.04]	5.2%	5.1%
Simonoff 2008	0.00	0.0063		0.00	[-0.01; 0.02]	37.0%	5.3%
Soh 2020	0.02	0.0154		0.02	[-0.01; 0.05]	6.2%	5.2%
Vasa 2018	0.02	0.0174		0.02	[-0.02; 0.05]	4.8%	5.1%
Verheij 2015	0.04	0.0229		0.04	[0.00; 0.09]	2.8%	5.0%
Fixed effect model			♦1	0.02	[0.01; 0.03]	82.5%	
Random effects model				0.05	[0.02; 0.08]		66.4%
					[-0.05; 0.15]		
Heterogeneity: $I^{-} = 14\%$, $\tau^{-} = 0.0018$, $p < 0.01$							
Self							
Magiati et al. 2014	0.29	0.0736		0.29	[0.15; 0.43]	0.3%	3.4%
Kaat et al. 2015	0.13	0.0497		0.13	[0.03; 0.23]	0.6%	4.2%
Fixed effect model				0.18	[0.10; 0.26]	0.9%	
Random effects model				0.20	[0.05; 0.36]		7.7%
Prediction interval							
Heterogeneity: $r = 69\%$, $\tau = 0.0087$, $p = 0.07$							
Fixed effect model			<u> ا</u>	0.03	[0.02; 0.04]	100.0%	
Random effects model			\diamond	0.11	[0.06; 0.15]		100.0%
Prediction interval		_			[-0.11; 0.32]		
Heterogeneity: $l^2 = 89\%$, $\tau^2 = 0.0100$, $p < 0.01$							
Test for overall effect (random effects): $z = 4.69$ ($p < 0.01$)		-0.2	0 0.2 0.4 0.6				
Test for subgroup differences (random effects): $\chi_2^{\circ} = 9.52$, df = 2 ($p < 0.01$)						

Note. The central line represents the global anxiety disorder prevalence among typically developing young people (Polancyzk et al., 2015

The Impact of Influential Primary Studies

"Leave-one-out" analysis was conducted for anxiety disorders with problematic heterogeneity (defined by Higgins $I^2 > 75\%$ and p < .01), and Baujat plots that indicated discrepant and disproportionately influential studies (Baujat et al., 2002). Disorders that met these criteria included all anxiety disorders with the exception of agoraphobia and panic disorder.

The random effects model was calculated for each disorder separately, removing each primary study in turn. These studies were then re-examined with a view to removal from the meta-analysis if substantive reason for discrepancy could be identified. Change in weighted average effect size (i.e., influence) and change in heterogeneity (i.e., discrepancy) are shown in Table 7. Baujat plots for each disorder type can be found in Appendix 6.

Anxiety disorders with discrepant and influential studies included unspecified anxiety disorder (k = 3), specific phobia (k = 2), generalised (k = 1), social (k = 1) and separation anxiety (k = 1). Methodological quality among these studies did not vary considerably from others included within this meta-analysis. Further, impact on prevalence and heterogeneity was minimal once removed; therefore, excluding from further analysis on the basis of methodological quality was not deemed appropriate. Instead change to prevalence estimates and heterogeneity are reported (Table 7).

Ta	bl	e	7
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Study	Uncorrected Prevalence	Corrected Prevalence	Difference	95% CI	Corrected I ²	I ² diff- erence
UNSPECIFIE D ANXIETY						
Den Houting (2019)	0.3961	0.3871	009	0.3297; 0.4445	95.2%	1%
Mayes (2011)	0.3961	0.3871	009	0.3295; 0.4447	93.4%	- 1.9%
Williams (2011)	0.3961	0.3874	0087	0.3297; 0.4451	94.9%	- 0.4%
SPECIFIC PHOBIA						
Leyfer (2006)	0.2336	0.2157	0179	0.1433; 0.2881	89.6%	- 1.9%
Soh (2020)	0.2336	0.2509	+.02	0.1781; 0.3238	87.6%	3.9%
GENERALISE D ANXIETY						
Bitsika (2016)	0.1450	0.1332	0118	0.0964; 0.170	91%	- 1.9%
SOCIAL ANXIETY						
Bellini (2004)	0.1488	0.1356	0132	0.0983; 0.1729	83.1%	-4%
GEDADATION						
ANXIETY Ben-Itzchak (2020)	0.1076	0.0923	02	0.0538; 0.1307	87.2%	- 1.8%

Results of the "Leave-One-Out" Analysis

Results of Meta-Analysis: Depression

Results are displayed in Table 8. There was a statistically significant difference between the different types of depressive disorder, $X^2(2, k = 36) = 31.68, p < .0001$; large heterogeneity was also evident ($I^2 = 94\%$, $t^2 = .0225$, p < .01). The 28 studies reporting the presence of unspecified depressive disorder had a weighted average prevalence rate of 23.73% (k = 28, 95% CI 11.70%, 29.76%); although, significant heterogeneity between the primary studies was observed ($I^2 = 95\%$, $t^2 = .0275$, p < .01). The highest prevalence for a specific depressive disorder was seen in major depressive disorder, with a prevalence of 8.32% (k = 13, 95% CI 4.36%, 12.28%). For dysthymia the estimated prevalence was 4.67% (k = 8, 95% CI .0190; .0745).

Effect of Rating Type

As shown in Table 8, the variation between prevalence estimates of unspecified depressive disorder, major depressive disorder and dysthymia according to rating type was statistically significant. For unspecified depressive disorder (Figure 6), self and parent-rated prevalence estimates were similar and professional estimates were the lowest, $X^2(2, k = 28) = 30.95, p < .0001$; with significant heterogeneity noted ($I^2 = 95\%, t^2 = .0275, p < .01$). For major depressive disorder (Figure 7), the self-rated prevalence estimate was highest, whilst parent and professional ratings were similar; however self-rated prevalence was based on only one study; $X^2(2, k = 13) = 22.38, p < .0001$ and heterogeneity was significant ($I^2 = 83\%, t^2 = .0051, p < .01$). For dysthymia (Figure 8), the parent-rated prevalence estimate was highest, followed by professional-rated prevalence; no studies used self-rated measures for dysthymia and fewer studies used parent-rated measures as opposed to professional-rated measures, $X^2(1, k = 8) = 17.3, p < .0001$; again, significant heterogeneity was observed, $I^2 = 86\%, t^2 = .0016, p < .01$

Across all depressive disorders, irrespective of who was rating, estimates exceeded global prevalence for depression among typically developing young people worldwide, reported to be 2.6% (Polanczyk et al., 2015); represented by the central line on Figures 6, 7 and 8. Unspecified depression prevalence estimates determined by parent and self-ratings were approximately ten times the global prevalence estimate.

Table 8

	Studies a	Data Points ^a	PR	%	Lower 95%-	Upper 95%-	tau ²	I^2	Q
					CI	CI			
UNSPECIFIED									
DEPRESSIVE									
DISORDER	28	32	.2373	23.73	.1170	.2976	.0275	95%*	
Rated by parent	15	15	.2901	29.01	.2140	.3662	.0195	93%*	
Rated by	8	8	.0687	6.87	.0255	.1120	.0029	74%*	
professional									
Rated by self	9	9	.3009	30.09	.1680	.4338	.0377	94%*	
Subgroup differences							.0275	95%*	30.95* *
MAJOR DEPRESSIVE DISORDER	13	15	.0832	8.32	.0436	.1228	.0051	83%*	
Rated by parent	2	2	.0418	4.18	.0121	.0714	.0003	55%	
Rated by professional	12	12	.0711	7.11	.0365	.1056	.0028	78%*	
Rated by self	1	1	.3571	35.71	.2316	.4826			
Subgroup differences							.0051	83%*	22.38* *
DYSTHYMIA	8	10	.0467	4.67	.0190	.0745	.0016	86%*	
Rated by parent	2	2	.1128	11.28	.0817	.1438	0	0%	
Rated by professional	8	8	.0306	3.06	.0073	.0539	.0008	78%*	
Rated by self	0	0							
Subgroup differences							.0016	86%*	17.23* *

Summary of Depressive Disorder Prevalence

^aFigures in the column titled 'data points' represent how many data points there were. This is different to number of 'studies' because some studies included more than one rater (e.g., self, parent, professional).

*Significant at *p* <.01 level

**Significant at p<.0001 level

Forest Plot Showing Unspecified Depressive Disorder Prevalence Among Young Autistic People According to Rater

Study	те	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
ParentBitsika et al. 2016Briot et al. 2020Kaat, Kenneth, Gadow & Lecavalier 2013Lopata et al. 2010Mayes 2011Montazeri 2020Ooi 2011Pfeiffer 2005Snow 2011Solomon 2012Vickerstaff 2007Johnston 2017Kaat et al. 2015Strang et al. 2012Fixed effect modelRandom effects modelPrediction intervalHeterogeneity: $J^2 = 93\%$, $\tau^2 = 0.0195$, $p < 0.01$	$\begin{array}{c} 0.09\\ 0.19\\ 0.11\\ 0.40\\ 0.50\\ 0.24\\ 0.12\\ 0.16\\ 0.25\\ 0.27\\ 0.48\\ 0.48\\ 0.44\\ \end{array}$	0.0234 0.0441 0.0295 0.0775 0.0392 0.0762 0.0549 0.0460 0.0509 0.0685 0.0950 0.0610 0.0610 0.0737 0.0510		0.09 0.19 0.11 0.40 0.50 0.24 0.31 0.12 0.16 0.25 0.27 0.48 0.48 0.48 0.44 0.25 0.29	[0.04; 0.14] [0.10; 0.28] [0.06; 0.17] [0.25; 0.55] [0.16; 0.31] [0.24; 0.54] [0.20; 0.42] [0.02; 0.42] [0.06; 0.26] [0.12; 0.38] [0.09; 0.46] [0.36; 0.60] [0.33; 0.62] [0.34; 0.54] [0.23; 0.27] [0.21; 0.37] [-0.02; 0.60]	5.7% 1.6% 3.6% 4.4% 2.0% 1.5% 1.2% 0.5% 1.2% 0.8% 0.8% 0.8% 0.8% 25.6%	3.4% 3.2% 3.3% 2.8% 3.4% 3.1% 3.1% 3.1% 2.9% 2.6% 3.0% 2.9% 3.1% 2.9% 46.2%
Professional Demirkaya, Tutkunkardaş & Mukaddes 2016 Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011 Kaat, Kenneth, Gadow & Lecavalier 2013 Leyfer et al. 2006 Mukaddes 2010 Pandolfi 2014 Simonff 2008 Patel et al. 2017 Fixed effect model Random effects model Prediction interval Heterogeneity: $I^2 = 74\%$, $\tau^2 = 0.0029$, $p < 0.01$	0.18 0.07 0.06 0.03 0.02 0.20 0.20 0.02	0.0520 0.0304 0.0223 0.0157 0.0165 0.0457 0.0125 0.0543		0.18 0.07 0.06 0.03 0.02 0.20 0.02 0.02 0.08 0.04 0.07	[0.08; 0.28] [0.01; 0.13] [0.02; 0.10] [0.00; 0.06] [0.01; 0.05] [0.01; 0.04] [-0.03; 0.19] [0.02; 0.05] [0.03; 0.11] [-0.07; 0.21]	1.2% 3.4% 6.3% 12.7% 11.4% 1.5% 19.9% 1.1% 57.3%	3.1% 3.3% 3.4% 3.4% 3.2% 3.4% 3.1% 26.4%
Self Bitsika & Sharpley 2015 Bitsika et al. 2016 Hebron & Humphrey 2014 Montazeri 2020 Quek 2012 Richdale 2014 Schwartman 2021 Whitehouse 2009 Kaat et al. 2015 Fixed effect model Random effects model Prediction interval Heterogeneity: $I^2 = 94\%$, $\tau^2 = 0.0377$, $p < 0.01$ Fixed effect model Random effects model Random effects model Random effects model	0.47 0.08 0.29 0.11 0.37 0.40 0.66 0.04	0.0597 0.0222 0.1026 0.0417 0.0408 0.0929 0.0438 0.0802 0.0802		0.47 0.08 0.29 0.11 0.37 0.40 0.66 0.04 0.14 0.30	[0.35; 0.59] [0.04; 0.12] [0.16; 0.56] [0.21; 0.37] [0.19; 0.55] [0.31; 0.49] [0.50; 0.81] [-0.02; 0.10] [0.17; 0.43] [-0.19; 0.79] [0.10; 0.13] [0.18; 0.30] [0.14; 0.62]	0.9% 6.3% 0.3% 1.9% 0.4% 1.6% 3.4% 17.1% 	3.0% 3.4% 2.5% 3.2% 2.6% 3.2% 2.8% 3.3%
Prediction interval Heterogeneity: $l^2 = 95\%$, $\tau^2 = 0.0275$, $p < 0.01$ Test for overall effect (random effects): $z = 7.72$ ($p < 0.01$) Test for subgroup differences (random effects): $\chi_2^2 = 30.95$, df = 2 ($p < 0$.01)	-1	0.4 -0.2 0 0.2 0.4 0.6 0.8	1	[-0.11; 0.58]		

Note. The central line represents the global Depressive disorder prevalence among typically developing young people (Polancyzk et al., 2015)

Forest Plot Showing Major Depressive Disorder Prevalence Among Young Autistic People According to Rater

Study	TE	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
Parent Gadow, Devincent, Pomeroy & Azizian 2005 Kaat, Kenneth, Gadow & Lecavalier 2013 Fixed effect model Random effects model Prediction interval Heterogeneity: $l^2 = 55\%$, $\tau^2 = 0.0003$, $\rho = 0.13$	0.06 0.03	0.0137 0.0149		0.06 0.03 0.04 0.04	[0.03; 0.08] [0.00; 0.06] [0.02; 0.06] [0.01; 0.07]	10.2% 8.6% 18.8% 	7.8% 7.7% 15.5%
ProfessionalAmr et al. 2012Coskun, Hajdini, Alnak & Karayagmurlu 2020Bruin, Ferdinand, Meester & Verheij 2007Gadow, Devincent, Pomeroy & Azizian 2005Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011Kaat, Kenneth, Gadow & Lecavalier 2013Leyfer et al. 2006Mukaddes 2010Orinstein 2015Simonff 2008Taylor 2016Verheij 2015Fixed effect modelRandom effects modelPrediction intervalHeterogeneity: $l^2 = 78\%$, $r^2 = 0.0028$, $p < 0.01$	0.13 0.25 0.11 0.02 0.01 0.02 0.05 0.07 0.01 0.14 0.11	0.0439 0.0559 0.0318 0.0092 0.0140 0.0122 0.0281 0.0281 0.0281 0.0397 0.0089 0.0576 0.0361		- 0.13 0.25 0.11 0.02 0.10 0.10 0.05 0.07 0.01 0.14 0.11 0.03 0.07	[0.05; 0.22] [0.14; 0.36] [0.04; 0.17] [0.01; 0.04] [-0.01; 0.04] [-0.01; 0.04] [-0.01; 0.15] [-0.01; 0.15] [-0.01; 0.015] [0.03; 0.25] [0.04; 0.18] [0.02; 0.04] [0.04; 0.11] [-0.05; 0.19]	1.0% 0.6% 1.9% 22.5% 9.8% 12.8% 2.3% 2.3% 2.4% 1.2% 80.7% 80.7%	5.8% 5.0% 6.7% 7.9% 7.7% 7.8% 6.9% 7.0% 6.1% 4.9% 6.4%
Self Cai, Richdale, Dissanayake, Trollor & Uljarević 2019 Fixed effect model Random effects model Prediction interval Heterogeneity: not applicable	0.36	0.0640		0.36	[0.23; 0.48] [0.23; 0.48] [0.23; 0.48]	0.5% 0.5% 	4.4% 4.4%
Fixed effect model Random effects model Prediction interval Heterogeneity: $l^2 = 83\%$, $\tau^2 = 0.0051$, $p < 0.01$ Test for overall effect (random effects): $z = 4.12$ ($p < 0.01$) Test for subgroup differences (random effects): $\chi_2^2 = 23.28$, df = 2 ($p < 0.1$)	01)	-0.1	0 0.1 0.2 0.3	0.03 0.08 0.4 0.5	[0.02; 0.04] [0.04; 0.12] [-0.08; 0.24]	100.0% 	 100.0%

Note. The central line represents the global depressive disorder prevalence among typically developing young people (Polancyzk et al., 2015)

Forest Plot Showing Dysthymia Prevalence Among Young Autistic People According to Rater

									Weight	Weight
Study	TE	seTE			PR		PR	95%-CI	(fixed)	(random)
Parent Gadow, Devincent, Pomeroy & Azizian 2005 Kaat, Kenneth, Gadow & Lecavalier 2013 Fixed effect model Random effects model Prediction interval Heterogeneity: $J^2 = 0\%$, $\tau^2 = 0$, $p = 0.99$	0.11 0.11	0.0188 0.0295					0.11 0.11 0.11 0.11	[0.08; 0.15] [0.06; 0.17] [0.08; 0.14] [0.08; 0.14]	4.8% 1.9% 6.7% 	10.1% 8.0% 18.1%
ProfessionalBruin, Ferdinand, Meester & Verheij 2007Gadow, Devincent, Pomeroy & Azizian 2005Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011Kaat, Kenneth, Gadow & Lecavalier 2013Mukaddes 2010Simonff 2008Taylor 2016Verheij 2015Fixed effect modelRandom effects modelPrediction intervalHeterogeneity: $J^2 = 78\%$, $\tau^2 = 0.0008$, $p < 0.01$	0.02 0.10 0.01 0.06 0.02 0.00 0.06 0.01	0.0149 0.0177 0.0140 0.0223 0.0165 0.0063 0.0382 0.0095				- 	0.02 0.10 0.01 0.06 0.02 0.00 0.06 0.01 0.02 0.03	[-0.01; 0.05] [0.06; 0.13] [-0.01; 0.04] [0.02; 0.10] [-0.02; 0.05] [-0.01; 0.02] [-0.01; 0.03] [0.01; 0.02] [0.01; 0.05] [-0.05; 0.11]	7.6% 5.4% 8.6% 3.4% 6.2% 42.4% 1.2% 18.6% 93.3%	10.8% 10.3% 10.9% 9.4% 10.5% 12.0% 6.5% 11.6%
Fixed effect model Random effects model Prediction interval Heterogeneity: $l^2 = 86\%$, $\tau^2 = 0.0016$, $p < 0.01$ Test for overall effect (random effects): $z = 3.30$ ($p < 0.01$) Test for subgroup differences (random effects): $\chi_1^2 = 17.23$, df = 1 ($p < 0.01$)	-0.1	-0.05	0	0.05 0	.1 0.15	0.02 0.05	[0.01; 0.03] [0.02; 0.07] [-0.05; 0.15]	100.0% 	 100.0%

Note. The central line represents the global Depressive disorder prevalence among typically developing young people (Polancyzk et al., 2015).

The Impact of Influential Primary Studies

The same process stated above for anxiety disorders was completed for unipolar depressive disorders. Depressive disorders with discrepant and influential studies included; unspecified depressive disorder (k = 1), major depressive disorder (k = 1), dysthymia (k = 1). As stated above, due to the minimal impact on prevalence and heterogeneity once removed and no clear differences in methodological quality compared to other studies, these studies were not excluded from further analysis. Change to prevalence estimates and heterogeneity are reported below (Table 9).

Study	Uncorrected Prevalence	Corrected Prevalence	Difference	95% CI	Corrected I ²	I ² difference
UNSPECIFIED DEPRESSION Whitehouse (2009)	0.2373	0.2242	01	0.1672; 0.2811	95.2%	2%
MAJOR DEPRESSIVE DISORDER Cai (2019)	0.0832	0.0632	02	0.0354; 0.0910	76.2%	-6.4%
DYSTHYMIA Gadow (2005)	0.0467	0.0383	0084	0.0126; 0.0640	80.9%	-5.5%

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Results of the "Leave-One-Out" Analysis

Moderator Analyses

Risk of Bias

To assess the impact of risk of bias upon heterogeneity, subgroup analyses were conducted on the prevalence rates of each anxiety and depressive disorder with ratings of "low-risk" and "any-risk" (i.e., unclear risk and high risk of bias combined) for each of the six types of bias. Results for unspecified anxiety and depressive disorders were non-significant across all types of bias. See Appendix 7 (Tables 7.1 - 7.10) for results tables for each disorder. For brevity, significant results are summarised here. Disorders that evidenced statistically significant differences in estimates of prevalence according to risk of bias included specific phobia (generalisability bias), generalised anxiety disorder (detection and reporting bias), social anxiety disorder (detection bias), separation anxiety (performance and detection bias), panic disorder (performance bias), agoraphobia (detection bias), major depressive disorder (statistical bias), and dysthymia (detection and reporting bias). However, for agoraphobia and dysthymia, the few studies included in the analysis for detection and reporting bias respectively, may have impacted statistical analysis.

Table 10, summarises the significant results and the impact on prevalence rates and heterogeneity. Lower levels of detection bias were associated with lower prevalence estimates for generalised anxiety disorder, social anxiety disorder, separation anxiety, agoraphobia and dysthymia. Similarly, lower levels of reporting bias were associated with lower prevalence estimates for generalised anxiety disorder and dysthymia, and lower levels of performance bias were associated with lower prevalence estimates for separation anxiety and panic disorder. This indicates that inclusion of studies at risk of detection, reporting or performance bias respectively may have contributed to heterogeneity and increased prevalence estimates for the aforementioned disorders. For specific phobia, lower levels of generalisability bias were associated with higher prevalence estimates and a similar pattern was reported for major depressive disorder and levels of statistical bias. This means that inclusion of studies at risk of generalisability (specific phobia) or statistical bias (major depressive disorder) may have contributed to heterogeneity and decreased prevalence estimates for the relevant disorders.

Table 10

Table Presenting the Impact of Risk of Bias on Disorder Prevalence Estimates

Detection Bias	Lov	v Risk		Any R	isk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	<i>X</i> ²	р
Generalised Anxiety	.0918	.0361; .1475	10	.1716	.1197; .2236	19	4.21	.0401
Social Anxiety	.0712	.0299; .1125	7	.1750	.1216; .2285	18	9.06	.0026
Separation Anxiety	.0175	.0056; .0294	8	.1560	.0936; .2185	14	18.24	< .0001
Agoraphobia	.0300	.0040; .0561	2	.0838	.0517; .1159	3	6.51	.0107
Dysthymia	.0110	0042; .0261	3	.0580	.0244; .0935	7	5.68	.0171
Reporting Bias	Low Risk			Any Risk				
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Generalised Anxiety	.1076	.0721; 1431	26	.2797	.2062;	6	17.05	< .0001
Dysthymia	.0269	.0066;	8	.1052	.0800;	2	22.57	<.0001
Performance Bias	Low Risk			Any Risk				
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Separation Anxiety	.0692	.0304; .1080	16	.2219	.1532; .2905	6	14.4	.0001
Panic	.0127	.0047; .0206	10	.2112	.0412; .3813	2	5.23	.0222
Generalisability Bias	Low Risk			Any Risk				
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Specific Phobia	.3627	.2762; .4492	3	.2087	.1259; .2915	11	6.35	.0117
Statistical Bias	Low Risk			Any Risk				
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Major Depressive	.1054	.0500; .1607	11	.0339	.0118; .0561	4	5.51	.0189

The Effect of Study Design

The studies reporting on anxiety and depressive disorders were categorised by their overall study design into either (1) case control studies, (2) Prospective case cohort studies and (3) retrospective case cohort studies.

Anxiety Disorders. No significant differences between the weighted average prevalence of case control studies, prospective case cohort studies and retrospective case cohort studies was found for any of the anxiety disorders (see Table 11).

Table 11

Results .	Relating	to the Ef	fect of	^r Study	Design of	n Anxiety .	Prevalence .	Rates
-----------	----------	-----------	---------	--------------------	-----------	-------------	--------------	-------

Disorder	X^2	р
Unspecified Anxiety	1.38	.709
Generalised Anxiety Disorder	2.46	.292
Specific Phobia	0.72	.397
Social Anxiety Disorder	1.85	.397
Agoraphobia	6.81	.033
Separation Anxiety Disorder	4.25	.119
Panic Disorder	2.29	.3184

Depressive Disorders. For unspecified depressive disorder (Figure 9), there was a significant difference between the weighted average prevalence of case control studies, prospective case cohort studies and retrospective case cohort studies. X^2 (2, k = 28) = 15.73, p < 0.001). Case control studies reported higher prevalence of 35.84% (k = 13, 95% CI 27.57%, 24.11%) and retrospective case cohort studies reported the lowest at 15.19% (k = 18, 95% CI 8.84%, 21.53%). There was only one prospective case cohort study. Inclusion of either case

control or retrospective case cohort studies may have contributed to heterogeneity and increased or decreased (respectively) prevalence of unspecified depressive disorder.

Figure 9

The Impact of Study Design for Unspecified Depressive Disorder

Study	TE	seTE	PR	PR	95%-CI	Weight (fixed)	Weight (random)
Case control study	0.47	0.0507		0.47	[0.25, 0.50]	0.0%	2.0%
Brist at al. 2020	0.47	0.0597		0.47	[0.35; 0.59]	1.6%	3.0%
briot et al. 2020	0.19	0.0441		0.19	[0.10; 0.26]	0.5%	3.2%
Mayes 2011	0.50	0.0267		0.40	[0.45: 0.55]	4 4%	3.4%
Montazeri 2020	0.29	0.0417		0.29	[0.21: 0.37]	1.8%	3.2%
Montazeri 2020	0.24	0.0392		0.24	[0.16; 0.31]	2.0%	3.3%
Muscatello 2020	0.39	0.0762		0.39	[0.24; 0.54]	0.5%	2.8%
Richdale 2014	0.37	0.0929		0.37	[0.19; 0.55]	0.4%	2.6%
Schwartman 2021	0.40	0.0438		0.40	[0.31; 0.49]	1.6%	3.2%
Solomon 2012	0.25	0.0685		0.25	[0.12; 0.38]	0.7%	2.9%
Whitehouse 2009	0.66	0.0802		- 0.66	[0.50; 0.81]	0.5%	2.8%
Johnston 2017	0.48	0.0610		0.48	[0.36; 0.60]	0.8%	3.0%
Fatel et al. 2017	0.08	0.0543		0.08	[-0.03; 0.19]	16 7%	3.1%
Random effects model				0.36	[0.28:0.44]	10.7 %	39.5%
Prediction interval				0.00	[0.04: 0.68]		
Heterogeneity: I^2 = 89%, τ^2 = 0.0196, $p < 0.01$					[,]		
Prospective case cohort study							
Demirkaya, Tutkunkardaş & Mukaddes 2016	0.18	0.0520		0.18	[0.08; 0.28]	1.2%	3.1%
Fixed effect model				0.18	[0.08; 0.28]	1.2%	2 4 9/
Random effects model				0.16	[0.06; 0.26]		3.1%
Heterogeneity: not applicable							
Retrospective case cohort study							
Bitsika et al. 2016	0.08	0.0222		0.08	[0.04; 0.12]	6.3%	3.4%
Bitsika et al. 2016	0.09	0.0234		0.09	[0.04; 0.14]	5.7%	3.4%
Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011	0.07	0.0304		0.07	[0.01; 0.13]	3.4%	3.3%
Kaat Kenneth Gadow & Lecavalier 2013	0.30	0.1026		0.30	[0.16; 0.56]	3.6%	2.5%
Kaat, Kenneth, Gadow & Lecavalier 2013	0.06	0.0223		0.06	[0.02: 0.10]	6.3%	3.4%
Leyfer et al. 2006	0.03	0.0157	+	0.03	[0.00; 0.06]	12.7%	3.4%
Mukaddes 2010	0.02	0.0165	+	0.02	[-0.02; 0.05]	11.4%	3.4%
Ooi 2011	0.31	0.0549		0.31	[0.20; 0.42]	1.0%	3.1%
Pandolfi 2014	0.20	0.0457		0.20	[0.11; 0.29]	1.5%	3.2%
Pfeiffer 2005	0.12	0.0460		0.12	[0.03; 0.21]	1.5%	3.2%
Quek 2012	0.11	0.0408		0.11	[0.03; 0.19]	1.9%	3.2%
Simonii 2006 Snow 2011	0.02	0.0125	₹	0.02	[-0.01; 0.04]	1 2%	3.4%
Vickerstaff 2007	0.27	0.0950		0.27	[0.09: 0.46]	0.3%	2.6%
Kaat et al. 2015	0.48	0.0737		0.48	[0.33; 0.62]	0.6%	2.9%
Kaat et al. 2015	0.04	0.0301		0.04	[-0.02; 0.10]	3.4%	3.3%
Strang et al. 2012	0.44	0.0510		0.44	[0.34; 0.54]	1.2%	3.1%
Fixed effect model				0.06	[0.05; 0.08]	82.1%	
Random effects model				0.15	[0.09; 0.22]		57.4%
Prediction interval Heterogeneity: $l^2 = 90\%$, $\tau^2 = 0.0168$, $\rho < 0.01$					[-0.13; 0.43]		
Fixed effect model			♦	0.12	[0.10; 0.13]	100.0%	
Random effects model				0.24	[0.18; 0.30]		100.0%
Heterogeneity: $l^2 = 95\%$, $r^2 = 0.0275$, $n < 0.01$				7	[-0.11; 0.58]		
Test for overall effect (random effects): $z = 7.72$ ($p < 0.01$)		-0.2	0 0.2 0.4 0.6	0.8			
Test for subgroup differences (random effects): χ_2^2 = 15.73, df = 2 ($p < 0.0$	1)						

Major Depressive Disorder. As shown in Figure 10, for major depressive disorder, there was a significant difference between weighted average prevalence according to study design, X^2 (3, k = 13) = 25.02, p = <.0001). The majority of studies were retrospective case cohort studies, yielding a higher prevalence of 6.89% (k = 11, 95% CI 3.22%; 10.56%). There were only two case control studies with average weighted prevalence of 3.89% (k = 2, 95%

CI .80%; 6.98%). There was only one prospective case cohort study and one cross-sectional study.

Figure 10

The Impact of Study Design for Major Depressive Disorder

Case control study Gadow, Devincent, Pomeroy & Azizian 2005 Gadow, Devincent, Pomeroy & Azizian 2005 Fixed effect model Random effects model Prediction interval Heterogeneity: $I^2 = 73\%$, $\tau^2 = 0.0004$, $p = 0.05$	0.06 0.0137 0.02 0.0092	0.06 0.02 0.03 0.04	[0.03; 0.08] [0.01; 0.04] [0.02; 0.05] [0.01; 0.07]	10.2% 22.5% 32.7% 	7.8% 7.9% 15.7%
Cross-sectional study Cai, Richdale, Dissanayake, Trollor & Uljarević 2019 Fixed effect model Random effects model Prediction interval Heterogeneity: not applicable	0.36 0.0640	0.36 0.36 0.36	[0.23; 0.48] [0.23; 0.48] [0.23; 0.48]	0.5% 0.5% 	4.4% 4.4%
Prospective case cohort study Verheij 2015 Fixed effect model Random effects model Prediction interval Heterogeneity: not applicable	0.11 0.0361	0.11 0.11 0.11	[0.04; 0.18] [0.04; 0.18] [0.04; 0.18]	1.5% 1.5% 	6.4% 6.4%
Retrospective case cohort study Amr et al. 2012 Coskun, Hajdini, Alnak & Karayagmurlu 2020 Bruin, Ferdinand, Meester & Verheij 2007 Gjevik, Eldevik, Fjæran-Granumi & Sponheim 2011 Kaat, Kenneth, Gadow & Lecavalier 2013 Kaat, Kenneth, Gadow & Lecavalier 2013 Leyfer et al. 2006 Mukaddes 2010 Orinstein 2015 Simonff 2008 Taylor 2016 Fixed effect model Prediction interval Heterogeneity: I ² = 78%, r ² = 0.0029, p < 0.01	0.13 0.0439 0.25 0.0559 0.11 0.0318 0.01 0.0140 0.02 0.0142 0.02 0.0122 0.10 0.0289 0.05 0.0281 0.07 0.0397 0.01 0.0089 0.14 0.0576	0.13 0.25 0.11 0.01 0.03 0.02 0.10 0.05 0.07 0.01 0.14 0.03 0.07	[0.05; 0.22] [0.14; 0.36] [0.04; 0.17] [-0.01; 0.04] [0.00; 0.06] [-0.01; 0.04] [0.04; 0.16] [-0.01; 0.15] [-0.01; 0.03] [0.03; 0.25] [0.02; 0.04] [0.03; 0.11] [-0.06; 0.20]	1.0% 0.6% 1.9% 9.8% 8.6% 12.8% 2.3% 2.4% 1.2% 0.6% 65.3%	5.8% 5.0% 6.7% 7.7% 7.8% 6.9% 7.0% 6.1% 7.9% 4.9%
Fixed effect model Random effects model Prediction interval Heterogeneily: I^2 = 83%, τ^2 = 0.0051, p < 0.01	F	0.03 0.08	[0.02; 0.04] [0.04; 0.12] [-0.08; 0.24]	100.0% 	 100.0%

Dysthymia. For dysthymia (Figure 11), there was a significant difference between weighted average prevalence according to study design, X^2 (2, k = 8) = 38.21, p = <.0001). Case control studies had a higher prevalence of 10.52% (k = 2, 95% CI 8%, 13.04%), than retrospective case cohort studies which yielded prevalence of 3.24% (k = 7, 95% CI .79%, 5.69%). The difference between these study designs is the comparison to neurotypical participants in case control studies; however, in the study design hierarchy, retrospective case cohort studies were given a higher rating as they enable recruitment of larger samples of autistic people. There was only one prospective case cohort study. These findings suggest that inclusion of different study types may contribute to variance in prevalence rates. In the studies included in this meta-analysis, in most cases, case control studies yielded higher prevalence rates for depressive disorders than retrospective case cohort studies. There were too few prospective case cohort or cross-sectional studies to assess the impact of this design on variability in prevalence rates.

Figure 11



The Impact of Study Design for Dysthymia

The Effect of Recruitment Location

Participants recruited from clinical settings were compared to those recruited from non-clinical settings (i.e., community or special school). No significant difference between recruitment locations for any of the anxiety or depressive disorders was observed. Full results can be found in Appendix 7, Table 7.11.

The Effect of Measurement Method

Studies were categorised by whether they had used interviews or questionnaires to measure anxiety or depression. Full results can be found in Table 10 below.

Anxiety. Statistically significant differences were observed between studies that had used interviews or questionnaires to measure generalised, social, and separation anxiety disorders. For generalised anxiety disorder, questionnaires yielded higher weighted prevalence (21.69%) than interviews (9.06%), X^2 (1, k = 24) = 11.33, p = <.001). Similarly, for social anxiety disorder, weighted prevalence was higher for questionnaires (20.8%) than interviews (9.56%), X^2 (1, k = 22) = 38.21, p = <.01). Studies reporting on separation anxiety also yielded higher weight prevalence for questionnaires (20.59%) as opposed to interviews (4.99%), X^2 (1, k = 20) = 9.61, p = <.01). As shown in Table 12, large heterogeneity was observed for each of these disorders.

Table 12

Results Relating to the Effect of Measurement Method on Anxiety Disorder Prevalence Rates

Anxiety Disorder	K	Prevalence	95% CI	Tau ²	I^2	Q
UNSPECIFIED ANXIETY	40			.0368	95%*	.91
Interview	17	.3625	.2838; 4411	.0255	91%*	
Questionnaire	23	.4181	.3355; .5007	.0443	96%*	
GENERALISED ANXIETY DISORDER	24			.0110	93%*	11.3**
Interview	17	.0906	.0516; .1297	.0053	84%*	
Questionnaire	7	.2169	.1546; .2792	.0104	94%*	
SPECIFIC PHOBIA	14					
Interview	14	.2336	.1589; .3082	.0171	91%*	
Questionnaire	0					
SOCIAL ANXIETY DISORDER	22			.0099	85%*	7.3*
Interview	13	.0956	.0570; .1342	.0036	78%*	
litterview	9	.2080	.1361; .2798	.0137	85%*	
Questionnaire						
AGORAPHOBIA	5			.0008	57%	5.27
Interview	4	.0458	.0226; .0690	<.0001	21%	
Questionnaire	1	.1392	.0629; .2156			
SEPARATION ANXIETY DISORDER	20			.0100	89%*	9.61*
T	14	.0499	.0234; .0763	.0018	74%*	
Interview	6	.2059	.1109; .3009	.0161	92%*	
Questionnaire						
PANIC DISORDER	11			.0009	72%*	3.12
Interview	8	.0113	.0032; .0194	<.0001	44%	
Questionnaire	3	.1160	.0001; .2318	.0121	87%*	

*Significant at p < .01 level

**Significant at *p*< .001 level

*** Significant at *p*< .0001 level

Depression. Statistically significant differences were observed between studies that had used interviews or questionnaires to measure unspecified depressive disorder, X^2 (1, k = 28) = 23.64, p = <.0001), and dysthymia, X^2 (1, k = 8) = 50.53, p = <.0001). Studies that used questionnaires exhibited higher weighted prevalence rates than those that conducted interviews for both disorders. For unspecified depressive disorder, measurement by questionnaire reported prevalence of 28.32% compared to interviews which yielded prevalence of 7.28%. For dysthymia questionnaires yielded a weighted prevalence of 9.63% compared to just 0.9% for interview-based measures. Significant heterogeneity was observed for both disorders. Full results can be found in Table 13 below.

Table 13

	The	Effect	of Measur	rement Meth	od on Dei	pressive Dis	order Preva	lence Rates
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Depressive Disorder	K	Prevalence	95% CI	Tau ²	I^2	Q
UNSPECIFIED DEPRESSIVE	28			.0275	95%*	23.64***
DISORDER	7	.0728	.0204; .1252	.0039	77%*	
Interview	21	.2832	.2165; .3499	.0257	94%*	
Questionnaire						
MAJOR DEPRESSIVE DISORDER	13			.0051	83%*	0
Tutoreione	10	.0856	.0452; .1261	.0031	82%*	
Questionnaire	3	.0853	0264; .1971	.0154	87%*	
DYSTHYMIA	8			.0016	86%*	50.53***
Interview	6	.0090	.0003; .0178	0	0%	
Questionnaire	2	.0963	.0739; .1187	< .0001	17%	

*Significant at *p* <.01 level

**Significant at p<.001 level

*** Significant at p<.0001 level

Heterogeneity Due to Participant Characteristics

Age. The impact of participant age upon the prevalence of each anxiety and depressive disorder was assessed by regressing the mean age for the study to the reported prevalence rate. Prevalence rates for specific phobia reduced by -.0336 for each year of age (z = -3.18, p < .01). Prevalence rates for major depressive disorder increased by 0.0136 for each year of age (z = 2.5234, p < .05). As can be seen in Table 14, no other anxiety or depressive disorders exhibited significant associations with participant age.

Table 14

Anxiety Disorder	Effect	95% CI	z	р
Unspecified anxiety disorder	0071	0289; .0146	6434	.5199
Generalised anxiety disorder	0091	0259: .0078	-1.0578	.2902
Specific phobia	- 0336	- 0543 0129	-3.18	0015**
Social anviety disorder	0550	0218: 0205	-5.10	.0015
Social allxiety disorder	0006	0218, .0203	0399	.9322
Separation anxiety disorder	.0070	0146; .0287	.6369	.5242
Panic disorder	.0020	0102; .0142	.3251	.7451
Agoraphobia	0077	0170; .0015	-1.6336	0.7510
Depressive Disorder	Effect	95% CI	z	р
Unspecified depressive disorder	.0085	0202; .0373	.5807	.5614
Major depressive disorder	.0136	.0030; .0242	2.5234	.0116*
Dysthymia	0008	0045; .0028	4381	.6613

The Impact of Participant Age Upon the Prevalence of Anxiety and Depressive Disorders

*Significant at p < .05 level

**Significant at *p* <.01 level

Sex. Differences in the prevalence of each anxiety and depressive disorder were compared by regressing the percentage of males in each sample to the reported prevalence rate. The association between proportion of male participants and prevalence did not achieve statistical significance for any of the anxiety or depressive disorders. For results, see Appendix 7 (Table 7.12).

Inclusion or Exclusion of People with Intellectual Disabilities. The impact of including people with intellectual disabilities was achieved by comparing the weighted average prevalence of anxiety and depressive disorders in studies that included and excluded people with intellectual disabilities. There was no significant difference between studies that excluded or included people with intellectual disabilities for any of the anxiety or depressive disorders (see Appendix 7, Tables 7.13 – 7.14 for full results).

The Impact of Publication and Small Study Biases

Publication bias is caused by the tendency for statistically significant results to be published more frequently than papers with non-significant results. Small study bias may also occur due to the tendency for studies with smaller sample sizes to show greater variability in their measurement of prevalence. Publication bias can be determined from visual reviewing funnel plots and checking for asymmetry (Egger, Smith, Schneider & Minder, 1997). Typically, if there is an absence of publication bias, the effects from studies with small sample sizes which show greater variability and will scatter more widely at the bottom of the plot compared to studies with larger samples which will lie closer to the overall meta-analytic average, creating an inverted funnel shape. If there is an absence of studies in the bottom left area of the plot (associated with small sample sizes and non-significant results), then it is likely there is some publication bias leading to an overestimation of the true effect. It has been argued that significant heterogeneity across prevalence rates for anxiety and depressive disorders may contribute to funnel plot asymmetry and create a false positive result when testing for publication bias (van Steensel et al., 2011). Further, it is debatable whether publication bias influences the rates of disorders reported in each study because samples were assessed to determine whether a disorder was present or not; thus, both null results and cases were reported, without manipulation of variables or significance testing (van Steensel et al.). Nevertheless, for completeness, funnel plots were reviewed and tests of publication bias were conducted as part of this meta-analysis.

Due to debate about the accuracy of visual funnel plot inspection (Terrin, Schmid, & Lau, 2005), a statistical test was also conducted. Egger's regression method was used with the standard error as moderator to the Random Effects Model (Egger et al., 1997). Funnel plots were inspected for each anxiety and depressive disorder separately. Where publication bias was evident, the effect of bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000). The procedure iteratively removes the most extreme small studies from the side of the funnel plot associated with positive effects, re-computing the effect size at each iteration until the funnel plot is symmetric about the (corrected) weighted average prevalence rate. Figure 12 presents the funnel plots for each anxiety disorder and (where relevant) the imputed studies from the trim and fill procedure. Funnel plots for depressive disorders are presented in Figure 13.

Unspecified Anxiety Disorder. There was no evidence of publication bias in the distribution of the prevalence of generalised anxiety. Egger's regression test was also not significant ($\beta = .3711, t = .17, p = .8636$).

Generalised Anxiety. There was evidence of publication bias in the distribution of the prevalence of generalised anxiety. Egger's regression test was also significant ($\beta = 5.099$, t =

4.65, p < .001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000); however, the trim and fill procedure did not impute any studies and therefore did not result in an adjustment to prevalence estimates. Using the Rosenthal algorithm, 4627 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 24 included studies).

Specific Phobia. There was evidence of publication bias in the distribution of the prevalence of specific phobia. Egger's regression test was also significant (β = 5.8964, *t* = 4.46, *p* < .001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000). The trim and fill procedure yielded a corrected prevalence estimate of 20.28% (95% CI 12.57%, 27.99%), evidencing an approximately 13.18% decrease relative to the uncorrected estimate of 23.36%. Using the Rosenthal algorithm, 1379 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 14 included studies).

Separation Anxiety. There was evidence of publication bias in the distribution of the prevalence of separation anxiety. Egger's regression test was also significant ($\beta = 3.9734$, t = 6.59, p < .0001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000); however, the trim and fill procedure did not impute any studies and therefore did not result in an adjustment to prevalence estimates. Using the Rosenthal algorithm, 1401 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 20 included studies).

Social Anxiety. There was evidence of publication bias in the distribution of the prevalence of social anxiety. Egger's regression test was also significant ($\beta = 4.2553$, t = 5.35, p < .0001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000); however, the trim and fill procedure did not impute any studies

and therefore did not result in an adjustment to prevalence estimates. Using the Rosenthal algorithm, 2900 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 23 included studies).

Panic Disorder. There was evidence of publication bias in the distribution of the prevalence of panic disorder. Egger's regression test was also significant ($\beta = 3.1706$, t = 6.32, p < .0001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000). The trim and fill procedure yielded a corrected prevalence estimate of 0.9% (5% CI -3.01%, 4.8%), evidencing an approximately 71.7% decrease relative to the uncorrected estimate of 3.18%. Using the Rosenthal algorithm, 145 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 11 included studies).

Agoraphobia. There was evidence of publication bias in the distribution of the prevalence of panic disorder; however, Egger's regression test could not be conducted as there were too few studies, thus there was insufficient power to distinguish chance from real asymmetry. Using the Rosenthal algorithm, 53 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 5 included studies).

Unspecified Depressive Disorder. There was evidence of publication bias in the distribution of the prevalence of unspecified depressive disorder. Egger's regression test was also significant ($\beta = 6.1679$, t = 5.45, p < .0001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000). The trim and fill procedure yielded a corrected prevalence estimate of 16.79% (95% CI 9.79%, 23.8%), evidencing an approximately 29.29% decrease relative to the uncorrected estimate of 23.73%. Using the Rosenthal algorithm, 8730 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 28 included studies).

Major Depressive Disorder. There was evidence of publication bias in the distribution of the prevalence of major depressive disorder. Egger's regression test was also significant ($\beta = 3.7387$, t = 6.68, p < .0001). The effect of publication bias was simulated using a trim and fill procedure (Duval & Tweedle, 2000); however, the trim and fill procedure did not impute any studies and therefore did not result in an adjustment to prevalence estimates. Using the Rosenthal algorithm, 604 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 13 included studies).

Dysthymia. There was evidence of publication bias in the distribution of the prevalence of dysthymia; however, Egger's regression test could not be conducted as there were too few studies, thus there was insufficient power to distinguish chance from real asymmetry. Using the Rosenthal algorithm, 212 unpublished null studies are required to reduce the meta-analytic effect to non-significance (based on the 8 included studies).

Funnel Plots of Anxiety Prevalence





Funnel Plots of Depression Prevalence



Unspecified Depression

Major Depressive Disorder

Dysthymia

Discussion

This meta-analysis aimed to estimate prevalence of specific anxiety and unipolar depressive disorders among young autistic people (5-25 years). A further aim was to determine the extent to which variance in prevalence rates could be accounted for by rater type (self, parent professional), study factors (design, recruitment location, measurement method) and participant characteristics (age, sex and intellectual ability). The subsequent paragraphs will discuss the results and impact of moderators, whilst considering limitations and potential implications of this meta-analysis.

Anxiety Disorder Prevalence Among Autistic Young People

Consistent with the results of van Steensel et al. (2011) and extending the findings to a broader age range encompassing autistic young adults, findings indicated disorder-level anxiety prevalence of 39.61%, which is significantly higher than the global prevalence of anxiety for neurotypical young people, estimated to be 6.5% (Polanczyk et al., 2015). Further consistency with van Steensel et al. was observed regarding the most and least prevalent disorders; specific phobia was the most common disorder (23.36%), whilst panic disorder was least common (3.18%). In this meta-analysis, second to specific phobia, social anxiety exhibited the highest prevalence (14.88%), followed by generalised anxiety (14.5%), separation anxiety (10.76%), again exceeding the 6.5% cumulative global prevalence for anxiety disorders. Although results were largely aligned with van Steensel et al., difference was observed in relation to agoraphobia prevalence which was significantly lower (5.94%) compared to that reported previously (16.6%). Result may present a genuine reduction in agoraphobia prevalence as three studies have been published since van Steensel et al., or, results may reflect application of slightly different inclusion criteria; for instance, Murris, Steerneman, Merckelbach, Holdrinet and Meesters (1998) reported prevalence of agoraphobia

to be 45.5%; however, the study was excluded from this meta-analysis as the sample included participants that were less than 5 years old. It is of note that the prevalence estimate reported by Murris et al. (1998) is significantly higher than the prevalence rates reported in the studies included in this meta-analysis. Given that agoraphobia prevalence rates were based on the fewest number of studies in both meta-analyses, results may have been easily influenced by one or two studies. Discrepancy between prevalence estimates may be clarified if more studies are conducted that measure co-morbidity of agoraphobia and ASD.

Although, these findings add to evidence for co-occurrence of anxiety disorders with ASD, the mechanisms underlying this association are yet to be established. Many relationships have been proposed in the literature; for instance, anxiety may result from ASDrelated environmental stressors or differences in executive functioning and emotional regulation, or anxiety and ASD symptoms may mutually exacerbate one another (Cai et al., 2018; Wood & Gadow, 2010). Further, recent work by Stark, Stacey, Mandy, Kringelbach and Happé (2021) highlights that the cognitive differences inherent to autism may be similar to those that are causally associated with anxiety in neurotypical people. For instance, cognitive inflexibility is a feature of ASD, whilst 'black and white thinking', associated with anxiety, refers to a binary, rigid cognitive style proposed to bias judgment and hinder adoption of more helpful cognitions.

It is likely that there are varied trajectories leading to different anxiety disorders, and given research indicating that autistic people experience atypical anxiety symptoms, use of more bespoke assessment methods and perhaps more exploratory, qualitative research exploring phenomenology may help to clarify the nature of the overlap between ASD and anxiety symptoms (Halim, Richdale & Uljarević, 2018; Kerns et al., 2014).

Moderators

Rating Type. Of the three rater types (parent, self, professional), there was a tendency for parents to rate higher, although this was not entirely consistent. Statistically significant differences between rater types were only observed for generalised, social and separation anxiety disorders. For generalised anxiety disorder and separation anxiety disorder, parent ratings were highest, followed by self-ratings, with professional ratings being the lowest. For both disorders, the parent-rated prevalence was more than double that of the professional-rated prevalence; the difference was largest for separation anxiety with ratings of 20.94% and 4.99% respectively.

These findings align with van Steensel et al. (2017), who found an association between parent report and higher prevalence. The observed discrepancy may indicate that rating type is confounded with measurement method (e.g., only professionals conduct interviews) or it may highlight that when assessed by professionals, symptoms of anxiety are overshadowed and seen as features of ASD rather than anxiety disorders in their own right. The propensity for studies to rely predominantly on professional ratings may mean that anxiety disorders are under-diagnosed in the studies included. Similarly, overreliance on parent ratings has also been discouraged due to parents tending towards over-reporting anxiety in their children (Bitsika et al., 2015a). In the case of generalised anxiety particularly, it has been suggested that self-assessment may be more accurate than parent report (Bitsika et al., 2015b). With this in mind, it is of note that far fewer studies included measures of selfreported anxiety. Only seven of the 40 studies that reported on unspecified anxiety included self-ratings. For specific phobia and agoraphobia, no self-ratings were collected in any of the studies, and for panic disorder (N = 1) and separation anxiety (N = 2) studies including selfratings were scant. Indeed, van Steensel et al. (2011) commented that there were so few studies that included self-report data, they were unable to include rater type as a moderator in the analysis. Reluctance to enquire with young autistic people about their experiences of anxiety may result from research suggesting that they are not able to identify and articulate their emotional experiences (Wood & Gadow, 2010), or that their symptoms are atypical and would be missed by generic mood measures (Halim et al., 2018; Kerns et al., 2014). Both scenarios indicate the importance of utilising measures and methodologies that are sensitive to detect anxiety within the autistic population.

Recruitment Location. Significant heterogeneity was observed within the findings of this meta-analysis and previous meta-analyses (van Steensel et al., 2011; 2017). A potential source of heterogeneity that was not investigated by van Steensel et al. (2017) was whether prevalence rates were moderated by the location from which participants were recruited. Within this meta-analysis findings indicated no significant difference in anxiety disorder prevalence whether participants were clinically referred or recruited from a community-based sample. One potential reason for this is that seven studies could not be entered into the subgroup analysis because they reported that participants were recruited from a research registry; thus, it was unclear whether clinically-referred participants were included in these samples. Clearer reporting regarding participant characteristics and recruitment may enable more definitive conclusions to be drawn in future.

Measurement Method. Significant differences were observed between the prevalence estimates of generalised, social and separation anxiety disorders according to whether questionnaire or interview-based measures were used. Although more studies used interviewbased measures, similar to van Steensel et al. (2011), for generalised and social anxiety, diagnosis was more likely if questionnaires were used. The same effect was observed for separation anxiety disorder, although not significant in the van Steensel et al. meta-analysis.
Additionally, van Steensel et al. reported that unspecified anxiety disorders were more likely to be diagnosed if interview-based measures were used; however, no such differences were observed in this meta-analysis. In fact, although not all differences reached significance, the consistent pattern observed was that use of questionnaires resulted in higher prevalence estimates.

Findings regarding interviews may reflect the tendency for professionals to diagnose anxiety disorders less frequently, stated above. Additionally, use of questionnaires may inflate prevalence rates if the measures detect symptoms that overlap with ASD features. Taking the example of social anxiety disorder, a qualitative study by Halim et al. (2018) found that the concerns autistic participants experienced in relation to social situations were different to that of neurotypical participants. The former, were inwardly focused and reported worries related to their ASD features; for instance, social communication and emotion recognition difficulties created uncertainty about how to behave socially. Whereas neurotypical participants were more outwardly focused on negative evaluation from others. Similar findings were reported in a systematic review regarding social anxiety in ASD (Spain, Sin, Linder, McMahon & Happé, 2018). Both groups might score highly on measures of social anxiety, particularly if parents are rating and have observed social difficulties in their child; but high scores could reflect different underlying causes and mechanisms. Again, this highlights the value of qualitative studies in helping to understand the phenomenology of anxiety in autistic people first-hand. It also emphasises again the importance of using mixed methodologies and measures that are designed to detect subtle differences between DSM-5 or ICD-11 defined anxiety disorders and potential variations of anxiety disorders experienced by autistic people.

Age. Significant decrease in prevalence of specific phobia with older age was observed. This may reflect the developmental risk period for specific phobia observed in

neurotypical children; phobias tend to develop in younger children, whilst other anxiety disorder become more prominent in adolescence (Becket et al., 2007). Results for all other anxiety disorders were not significant. Findings were inconsistent with van Steensel et al. (2011) who found that unspecified and generalised anxiety disorder prevalence increased with age, whilst separation anxiety prevalence decreased. The inconsistency here reflects that reported in the literature; some studies have reported increase in anxiety disorders with older age (for instance; Davis et al., 2011; Vasa et al., 2013), whilst others have found no effect (for instance; Lai et al., 2019; Niditch, Enrique, Kamps & Hill, 2012; Strang et al., 2012). Many of the studies included in the current meta-analysis collected data at one point in time, to clarify inconsistencies in the relationship between anxiety and age in autistic young people, it may be beneficial to adopt longitudinal designs that can explore changes in anxiety prevalence at particular developmental stages.

Sex. No significant differences were observed in prevalence rates resulting from the proportion of males included in studies. This was surprising given previously reported findings that neurotypical and autistic females are more likely to experience anxiety, compared to males (Jalnapurkar, Allen & Pigott, 2018; Sedgewick, Leppanen & Tchanturia, 2021), although it is of note that the meta-analysis by Lai et al. (2019) also found no relationship between sex and anxiety prevalence. However, these studies were based on adult samples and findings relating to young autistic people reported mixed findings in relation to sex differences (for a review see, Calderoni, 2022). Therefore, age may be a mediating factor in the relationship between sex and anxiety disorder prevalence in autistic people. Indeed, a seminal review reported that although co-occurring psychiatric conditions emerge in childhood, sex differences are only evident with age (Kirkovski, Enticott &Fitzgerald, 2013). A further factor to consider is that most studies in this meta-analysis used predominantly male

participants in the sample owing to the greater prevalence of autism among males; however, this may make it more difficult to detect subtle sex differences and make findings less generalisable to females. This may be improved by systematic reporting of prevalence rates by gender.

Intellectual Ability. Inclusion of people with intellectual disabilities did not significantly moderate prevalence rates for and of the anxiety disorders. Van Steensel et al. (2011) only found an effect for unspecified anxiety disorder; a relationship was found between lower IQ and higher prevalence of unspecified anxiety disorder. In the literature, both high and low IQ have been associated with increased anxiety in autistic children (Rosenberg, Kaufmann, Law & Law, 2011; Salazar et al., 2015; van Steensel et al., 2017; Mingins et al., 2021). It is possible that an effect was not found in the current meta-analysis because not all studies could be entered into the subgroup analysis due to unclear reporting. Some studies did not state whether people with intellectual disabilities were included or not. Given that approximately half of all autistic young people have a co-morbid intellectual disability (Charman et al., 2011), it is important to know how this relates to anxiety prevalence. Future studies should endeavour to clearly report whether people with intellectual disabilities were included in the sample or not.

Unipolar Depressive Disorder Prevalence Among Autistic Young People

This meta-analysis estimated the prevalence of unspecified unipolar depressive disorder among young autistic people to be 23.73%. This is significantly higher than the 10.6% prevalence estimate reported in the meta-analysis by Hudson et al. (2019), although the current meta-analysis included five more studies and the upper age limit was 25 years instead of focusing on young people <18 years. Additionally, this was the first meta-analysis to calculate prevalence estimates for specific unipolar depressive disorders separately. The

prevalence for major depressive disorder (8.32%), whilst the dysthymic disorder was less commonly reported among young autistic people (4.67%).

As demonstrated for anxiety, prevalence estimates across all depressive disorders significantly exceeded the global prevalence of unipolar depression (2.6%) among neurotypical young people. Unspecified depressive disorder prevalence specifically was almost was ten times the global estimate (Polanczyk et al., 2015). These findings are striking and highlight the scale of co-morbid mental health difficulties among young autistic people. Indeed, the risk factors associated with unipolar depression prevalence among young autistic people are numerous, encompassing genetic and environmental factors, negative life events and even features of ASD (DeFilippis, 2018). Acknowledging prevalence is crucial to the development of evidence-based, bespoke treatments for depression within the autistic population. Thus far, these treatments are lacking and require further attention (DeFilippis, 2018). Just five targeted intervention studies have been reported in the literature (Kim & Lecavalier, 2021). Inadequate provision of support implies that care needs are unmet, which is concerning as autistic young people are reported to experience suicidal ideation at a rate 28 times greater than their neurotypical peers (Mayes, Gorman, Hillwig-Garcia & Syed, 2013). Further, experience of depression in early adolescence contributes risk of suicide, emphasising the urgency for more effective early intervention (Culpin et al., 2018).

Moderators

Rating Type. There were statistically significant differences between prevalence estimates of all of the unipolar depressive disorders according to who was rating (self, parent, professional). For unspecified depressive disorder self-rated and parent-rated prevalence were highest, with little difference between the estimates, whilst professional-rated prevalence was significantly lower. These findings are consistent with Ozsivadjian et al. (2014) who reported agreement between self and parent ratings on questionnaires measuring depression. The same study found that autistic young people can accurately identify and report negative cognitions, which counters claims that subjective report is unreliable in the autistic population (Berthoz & Hill, 2005). Concerning low professional ratings, many of the diagnostic interviews that are typically used to diagnose depression are not validated for use with young autistic people and few adapted versions exist (Gjevik et al., 2011). Consequently, depressive symptoms may not be detected, particularly if symptoms are atypical. Further, phrasing of particular questions may preclude understanding of what is being asked, thus limiting accuracy.

For major depressive disorder, professional ratings were most frequently used. Although parent and professional ratings yielded similar prevalence estimates, only two studies used parent ratings. Self-ratings were significantly higher; but again, only one study used self-ratings. Reliance on professional assessment in studies may result in lower prevalence rates given the pitfalls noted above. Similar observations were noted for dysthymia, only two studies used parent ratings and none used self-ratings. Pertinently, the parent-rated prevalence estimate was significantly higher than that of professionals. When considering professional ratings across all unipolar depressive disorders, estimates are consistently low. In order to determine whether this reflects genuinely lower prevalence or this is an artefact of the method chosen, studies should endeavour to collect data from autistic young people and their care giver or parent.

Recruitment Location. No significant differences were observed between prevalence rates of unipolar depressive disorders depending upon the recruitment location. These findings are consistent with the meta-analysis conducted by Hudson et al. (2019); although adult samples were included in their meta-analysis, so this may not be comparable to younger autistic populations. The meta-analysis with autistic adults by Lai et al. (2019) did find an effect due to recruitment location, with those from clinical settings yielding higher prevalence estimates. However, Lai et al. combined community and research registry samples in their analysis. This may indicate that the lack of an effect for recruitment location is potentially due to excluding eight studies from the analysis when recruitment setting was described as a research registry or not reported. As it is not clear whether research registries do in fact contain clinical samples, clearer reporting is encouraged in future studies.

Measurement Method. Significant differences were observed between the prevalence estimates of both unspecified depressive disorder and dysthymic disorder according to whether questionnaire or interview-based measures were used. For both disorders, diagnosis was more likely if questionnaires were used, although for dysthymia this was based on far fewer studies. Similar to the pattern noted for anxiety disorders, these findings may reflect a tendency of professionals to diagnose less frequently. Equally, measurement methods may be confounded by the overlap between autism features and depressive symptoms, or measurement error. A further point to consider is whether questionnaires and interviews yield different prevalence estimates due to being developed for different purposes. For instance, questionnaires are often used as brief screening tools that are easily administered and require less resource than structured diagnostic interviews; because of this, when developing measures greater value may be placed on sensitivity, rather than specificity. Adaptation and validation of diagnostic interviews and questionnaire measures for young autistic people is greatly needed as inadequate diagnostic methods may delay or prevent access to treatment (Gjevik et al., 2011; Pezzimenti, Han, Vasa & Gotham, 2019). Consistent with recommendations made by Pezzimenti et al. (2019) the results of this meta-analysis reflect the importance of adopting a multi-method, multi-informant approach when assessing and diagnosing depressive disorders among young autistic people.

Age. Older age was found to significantly increase major depressive prevalence rates, but age did not significantly impact prevalence rates for unspecified depressive disorder or dysthymia. Research documenting prevalence of major depressive disorder in autistic populations is scant and no other meta-analyses have reported on major depressive disorder prevalence in young autistic people; however, findings here are consistent neurotypical trends that show increased prevalence of major depressive disorder with age (Schaakxs et al., 2018). The latter, non-significant results are consistent with Hudson et al. (2019) who observed no difference according to age; but not with the larger meta-analysis by Lai et al. (2019), who reported a relationship between older age and higher unipolar depression prevalence in adult samples. It is possible that age becomes a more significant moderator when there are greater proportions of participants above the age of 25; however, given the dearth of research regarding prevalence of specific unipolar depressive disorders among autistic people overtime, longitudinal studies are encouraged.

Sex. No significant differences were observed in unipolar depression prevalence rates resulting from the proportion of males included in studies. These results were consistent with Hudson et al. (2019), but again, not consistent with the meta-analysis by Lai et al. (2019) who reported higher proportion of females to be associated with higher depression prevalence. Similar to anxiety it may be that the relationship between depression and sex is moderated by age. Sex-based fluctuations in depression prevalence throughout adolescence have been reported in the literature; for instance, early adolescence has been associated with higher prevalence for females, with gender differences becoming less-evident in early adulthood (Gotham et al., 2015). As stated for anxiety, sex comparisons may be enhanced by including a larger proportion of female participants within studies. It may also be beneficial to explore sex differences at the

disorder-level, since most research focuses on depression in general, as opposed to major depression or dysthymia. Given the difference in course and duration of these disorders there are likely different implications for treatment.

Intellectual Ability. Similar to anxiety, no differences in unipolar depression prevalence rates were observed depending on whether people with intellectual disabilities were included in the sample or not. In the meta-analysis by Hudson et al. (2019) inclusion of people with intellectual disabilities was found to decrease prevalence estimates. Given that their meta-analysis explored lifetime prevalence and included adults in the sample, it may again show that age is a relevant factor in prevalence of depression in this group, although eight studies were excluded from analysis due to unclear reporting, which may have limited opportunity to find an effect. Although, mixed findings with regard to the relationship between intellectual disabilities and depression have been described previously (Chandrasekhar et al., 2015). Clarity may be gained from inclusion of people with differing levels of intellectual disability and clearer reporting as many studies report a mean IQ at best and the degree of disability may be important.

Limitations

Limitations should be considered when interpreting the findings of this meta-analysis. Firstly, larger heterogeneity was observed in the literature pertaining to anxiety and unipolar depressive disorders. Attempts were made to understand heterogeneity by exploring the impact of a number of moderators, one of which being informant or 'rater' type, which van Steensel et al. (2011) were unable to complete in their previous meta-analysis. However, significant heterogeneity remained suggesting that there may be some moderators that were not considered in this meta-analysis. The quality of studies should be considered when interpreting findings. Meta-analytic methods result in rigorous, comprehensive screening of the research literature; however, the pitfall of this is that findings are limited by the methodological limitations of the original studies. A number of studies were rated as high risk of generalisability bias due to recruiting predominantly high-functioning male samples, from clinical settings, which limits ability to generalised findings beyond these populations. Analyses were conducted to explore whether sex (proportion of male participants) and intellectual ability moderated prevalence rates; however, results may have been impacted by the inclusion and exclusion criteria for studies. Detection bias was also an area in which studies were frequently rated as high risk, due to utilising measures that had inadequate reliability and validity or that were not validated for use with autistic young people. High risk of detection bias creates uncertainty regarding whether particular assessment methods are genuinely measuring anxiety and depression, features of autism or some other phenomena, thus threatening validity.

The search was not restricted to particular geographical locations meaning that findings can be generalised globally. However, the search was limited to three research databases and only studies published in English. Therefore, it is possible that some studies reporting on prevalence have not been captured; though, results were largely consistent with previous meta-analyses (van Steensel et al., 2011; Hudson et al. 2019). Finally, few studies included measures of agoraphobia and dysthymia which means conclusions were based on relatively few papers and there may not have been sufficient power to detect the impact of moderators.

Future Directions

The sheer scale of co-morbidity among young autistic people highlights the need to shift the focus away from describing symptoms or co-morbidities, and towards the development of evidence-based adapted interventions. Given that the majority of studies were conducted before the COVID-19 pandemic, a further analysis, focused on studies published since the pandemic is needed in the near future to determine whether prevalence rates have been impacted.

A number of inequalities are evident in the literature, meaning that the extent to which autistic females and people with co-morbid intellectual disabilities experience mental illhealth may be underestimated. Future studies should focus on systematic reporting of prevalence rates according to participant sex and recruiting more representative samples. This will enable broader generalisations to be made, or perhaps highlight nuances associated with sex and IQ. Additionally, clearer reporting will enable more comprehensive analysis of these moderators. Further, longitudinal research may have particular value when exploring fluctuations in the prevalence of mental health conditions associated with age; such designs may also enable causal relationships to be delineated.

Regarding the differences observed depending on who was rating anxiety or depression, studies should aim to use a combination of methods, ensuring that attempts are made to enquire with young autistic people directly about their experiences. Indeed, uncertainty remains regarding whether the current assessment methods are actually measuring genuine mood disorders, features of ASD, or some other phenomena, given that most measures have not been developed with autistic people in mind. Qualitative studies may help to disentangle symptoms of autism, anxiety and unipolar depression, and deeper understanding may then inform the development of more appropriate measures.

Clinical implications

This meta-analysis has reported anxiety and depression prevalence among young autistic people that is disproportionate to their neurotypical counterparts. Considering known variation in prevalence estimates according to who is rating, the lack of adapted diagnostic measures (particularly for depression), and research indicating accuracy of self-ratings, there is value in collecting data from different perspectives (self, parent, professional), using varied methods (interview, questionnaire) to assess and diagnose co-morbid anxiety and depressive disorders among young autistic people.

Clinicians should be mindful of relying on one source of information or assessment tool. Whilst questionnaires may highlight mood difficulties in the first instance, probing of responses may be needed to check that interpretation of questions is as intended and to disentangle ASD symptomology from atypical mood difficulties. Equally, probing may highlight nuances related to the source of distress between autistic and neurotypical young people that are relevant for treatment; particularly in light of social anxiety research reporting that both groups score highly on measures, but the cognitive attributions underlying these scores differ in kind (Halim et al., 2018; Spain et al., 2018).

Despite greater depression prevalence in young autistic people, adapted evidencebased interventions are lacking. While this needs to be addressed empirically, in practice clinicians should be mindful of the numerous risk factors associated with unipolar depression prevalence among young autistic people, encompassing genetic and environmental factors, negative life events and even features of ASD (DeFilippis, 2018). As such, ongoing care and consideration of these factors should commence at the earliest possible stage.

This is the first meta-analyses to report on major depressive disorder prevalence in young autistic people. Findings are consistent with neurotypical trends that show increased prevalence of major depressive disorder with age (Schaakxs et al., 2018). Pertinently, many Child and Adolescent Mental Health Services in the United Kingdom support young autistic people up to the age of 18 years, at which point they are required to transition to adult services. Features of ASD, such as insistence on sameness, may exacerbate distress associated with this transition during a risk period major depressive disorder. Appropriateness of the timing of this transition is questionable for numerous reasons and clinicians should take great care when supporting young autistic people with this major change.

Conclusions

This meta-analysis reported prevalence rates for anxiety and unipolar depressive disorders among young autistic people that are disproportionate to neurotypical peers, highlighting the scale of mental ill-health within the young autistic population. Further, investigating prevalence separately according to informant revealed large variation in prevalence, emphasising different perspectives about what constitutes disorder-level anxiety and unipolar depression within the autistic population, or revealing lack of precision in measurement methods when detecting co-morbid conditions. Research into the overlap between ASD and common mental health conditions has a long way to go, particularly as females and people with intellectual disabilities may be understudied. Improvements could be made by greater inclusion of aforementioned groups, systematic reporting of prevalence rates according to participant sex, use of multiple assessment methods and methodologies (qualitative and quantitative), and longitudinal designs; however, the task of developing appropriate interventions to alleviate the distress of young autistic people must not be sidelined in pursuit of determining the exact nature of the relationship between ASD and anxiety or unipolar depression.

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Chapter 2

Empirical Research Paper

The Relationship Between Autistic Traits, Depressive Symptoms and Sleep

Quality

Word Count: 8, 205

Abstract

Background: Depression and sleep difficulties have been found to co-occur with autism and autistic traits, although the exact nature of this relationship is unclear.

Aims: Using the subscales of the Social Responsiveness Scale-Second Edition (SRS-2), this study aimed to examine a broad range of autistic traits and their unique interrelations with depressive symptoms and sleep difficulties.

Method: A total of 205 undergraduate students (age M= 19.1, SD= 2.9) took part in an online survey. Correlational analysis, multiple linear regression and mediation analysis were conducted to explore associations between measures of depressive symptoms, sleep difficulties, and autistic traits.

Results: All variables were highly correlated; however, only sleep difficulties and the 'restricted repetitive behaviour' (RRB) subscale of the SRS-2 remained in the regression model as predictors of depression. Mediation analysis revealed that the relationship between RRB and depression was partially mediated by sleep difficulties, but the predictive relationship between RRB and depression remained once sleep difficulties were controlled for.

Conclusions: Sleep difficulties and greater levels of autistic traits, particularly RRB, predict depressive symptoms. Implications for screening and treatment are considered and findings are discussed in the context of a biopsychosocial model.

Introduction

Autism Spectrum Disorder (hereon, referred to as autism) is marked by social communication and interaction differences and restricted or repetitive interests and behaviours (American Psychiatric Association, 2013). Autism is considered to exist at the extreme end of a spectrum of autistic traits, which are continuously distributed across the general population, reflecting natural variation in social function and behaviour patterns (Bralten et al., 2017; Constantino & Todd, 2003; Robinson et al., 2016).

To broaden understanding of the autism construct, research has taken a proportional approach, exploring how levels of autistic traits relate to mental health and functioning; for instance, prevalence of depression. Depression is four times more prevalent among autistic people (Hollocks, Lerh, Magiati, Meiser-Stedman & Brugha, 2019; Hudson, Hall & Harkness, 2019; Rai et al., 2018) and more common among those with a high degree of autistic traits (Kanne, Christ & Reiersen, 2009; Kunihira, Senju, Dairoku, Wakabayashi & Hasegawa, 2006). The reverse relationship has also been reported; neurotypical people diagnosed with depression have been found to present with higher levels of autistic traits (Matsuo et al., 2015). Additionally, from a genetic standpoint, mental health difficulties are commonly reported by family members of autistic people; although environmental factors may also play a part (Daniels et al., 2008; Jokiranta-Olkoniemi et al., 2016; Uljarevic, Nuske, & Vivanti, 2016). Despite the association between autism, autistic traits and depression being well-documented, the underlying mechanisms that drive the association are not well-understood. Research so far has implicated a range of factors.

Associations Between Subcomponents of Autistic Traits and Depression

Owing to the frequent co-occurrence of mood difficulties with autistic traits and autism, it is debated whether the symptoms observed represent true mood disorders or are more accurately defined as an expression of autism symptoms or traits. Indeed, the core features of autism and depression overlap considerably. Social communication and interaction differences and restricted interests and repetitive behaviours (RRBs) form the dyad of impairments upon which autism is diagnosed; however, features of the former, such as reduced social competence and motivation, and the latter, such as propensity to ruminate and rigid, repetitive cognition, are also observed in people with depression (Atherton, Nevels & Moore, 2015; Brookman-Frazee et al., 2018; Cai, Richdale, Dissanayake, Troller & Uljarević, 2018; Crane et al., 2013; Ishizuka, Ishiguro, Nomura & Inada, 2022; Joshi et al., 2010; Matsuo et al., 2015; Nolen-Hoeksema, 2000). Therefore, subcomponents of core autism impairments (RRB and social communication and interaction) are likely to be related to depression.

It is debated whether the relationship between autistic traits and depression is best explained by broad or more specific trait-based differences (Culpin et al., 2018; Gardiner et al., 2018; Ishizuka et al., 2022; Rai et al., 2018). For instance, some emphasise traits associated with RRB, such as reliance on maladaptive coping mechanisms (e.g., excess rumination, avoidance) and cognitive inflexibility (Andersen, Skogli, Hovik, Egeland & Øie, 2015; Gardiner & Larocci, 2018; Gotham, Bishop, Brunwasser & Lord, 2015; Pouw, Rieffe, Stockman & Gadow, 2013; Reiffe, De Bruine, De Rooij & Stockman, 2014); whilst others report that social communication and interaction differences are key (Rai et al., 2018).

Social communication and interaction differences in particular are associated with a range of complex factors found to be linked with depression. It is well-known that social acceptance and friendship enhance wellbeing, whereas disruption to formation and maintenance of relationships owing to social skill differences increases feelings of loneliness; a key mediator between autistic traits and mental health concerns (Kapp, 2018; Mazurek &

Kanne, 2010; Stice & Lavener, 2019). Additionally, awareness of skill differences among those with higher intellectual abilities may compound feelings of difference, isolation and rejection (Defilippis, 2018; LaFontana & Cillessen, 2010; Sterling, Dawson, Estes & Greenson, 2008); whilst, difficulties with verbally expressing inner experiences may make detection of depression less likely and delay or prevent access to support (Kanne, 2013; Magiati, Ozsivadjian & Kerns, 2017; Reilly, Senior & Murtagh, 2015; Uljarevic et al., 2016). This is concerning, as social communication and interaction differences have been associated with increased risk of harm to self (self-harm, suicidal ideation) and of harm from others (Borowsky, Taliaferro, & McMorris, 2013; Culpin et al., 2018). The experience of being bullied in particular has been found to enhance risk of depression among those with higher autistic traits, with frequency of bullying determining the severity of depressive symptoms (Borowsky, Taliaferro, & McMorris, 2013; Matthias, LaVelle, Johnson, Wu, & Thurlow, 2021; Zablotsky, Bradshaw, Anderson & Law, 2013).

In summary, autistic traits are likely to be related to depression and the relationship may be accounted for by shared variance in autistic traits; alternatively, particular traits related to either RRB or social communication and interaction may account for more of the variance in depressive symptoms (Culpin et al., 2018; Gardiner et al., 2018; Ishizuka et al., 2022; Rai et al., 2018). As such, a dimensional relationship may exist, with vulnerability to depression increasing as broad trait-based differences increase, due to greater overlap between the core features of autism (social communication and interaction differences and/or RRB) and features of depression (reduced social competence and motivation, rumination and repetitive cognition). Social skill differences may further compound risk by precluding access to support or protective relationships that buffer against feelings of loneliness. However, given the association between depression and sleep difficulties in the general population
(Marino et al., 2021), and the well-established association between sleep difficulties and features of autism (Schreck & Richdale, 2020), the role of sleep difficulties in this relationship must also be considered.

Sleep, Autism and Depression

Sleep difficulties are among the most common co-morbidities in autism and sleep is also known to be affected in people with higher levels of autistic traits and depression (Al-Beltagi, 2021; Schreck & Richdale, 2020). In neurotypical people, sleep problems and depression commonly co-occur, such that poor sleep is considered to be both a symptom of depression and a risk factor preceding depressive symptomology (Gregory & O'Connor, 2002; Lovato, Short, Micic, Hiller & Gradisar, 2017; Marino et al., 2021). Research indicates a two-fold increase in risk of depression associated with insomnia, and a four-fold increase in risk associated with sleep difficulties more generally (Baglioni et al., 2011; Jackson, Sztendur, Diamond, Byles & Bruck, 2014).

In autistic people, chronic sleep disruption is common across the lifespan (Croen et al., 2015; Richdale & Schreck, 2009, 2020; Uren, Richdale, Cotton & Whitehouse, 2019; Salmela, Kuula, Merikanto, Räikkönen & Pesonen, 2019; Stewart, Corbett, Ballard & Creese, 2020; Verhoeff et al., 2018). Sleep problems occur in approximately 50-80% of autistic children and 64% of autistic adults, compared to 9-50% of neurotypical children and 46% of neurotypical adults (Jovevska, Richdale & Lawson 2020; Richdale et al., 2009).

Meta-analytic findings have reported that autistic children experience reduced total sleep time, longer sleep onset latency and poorer sleep efficiency compared to neurotypical children (Díaz-Román, Zhang, Delorme, Beggiato, & Cortese, 2018). Whilst autistic adults and older adults have been reported to experience insomnia, frequent night time waking and excessive sleeping, in addition to aforementioned difficulties experienced by children (Baker

& Richdale, 2017; Carmassi et al., 2019; Croen et al., 2015; Goldman et al., 2017; Jovevska et al., 2020; Morgan, Nageye, Masi & Cortese, 2020; Schrek & Richdale, 2020).

Pertinently, poor sleep has been found to exacerbate impairments associated with autism. Shorter sleep duration and waking during the night were found to be associated with social communication and interaction differences and RRB (Schrek, Mulick & Smith, 2004). Further to this, those who slept for fewer hours per night exhibited reduced communication and socialisation skills and more stereotyped behaviour (Goldman et al., 2009; Hollway, Aman & Butter, 2013; Mazurek, Dovgan, Neumeyer & Malow, 2019; Taylor, Schrek & Mulick, 2012; Schrek et al., 2004). Greater bedtime resistance has also been associated with reduced reciprocal social interaction (Hollway et al., 2013), whilst stereotyped behaviour has been found to significantly predict sleep difficulties (Hundley, Shui & Malow, 2016), with improved behaviour noted once sleep difficulties were treated (Malow et al., 2012, 2014). It has been proposed that traits related to RRB such as cognitive inflexibility and ritualistic behaviour have potential to delay sleep onset or create emotional and physiological hyperarousal, precipitating the occurrence of insomnia (Harvey, Tang, & Browning, 2005; Mazzone, Postorino, Siracusano, Riccioni, & Curatolo, 2018; Tsai et al., 2021); whilst social communication and interaction deficits have a perpetuating role in sleep difficulties (Tsai et al., 2021).

Sleep problems have been reported to occur more frequently among people with higher levels of autistic traits (Verhoeff et al., 2018). Autistic traits in toddlerhood, childhood and adolescence were found to predict later sleep problems (Richdale et al., 2019; Samela et al., 2019; Uren, Richdale, Cotton, & Whitehouse, 2020). Whilst autistic traits in older adulthood were associated with daytime sleepiness, insomnia, reduced sleep quality and tiredness upon waking (Stewart et al., 2020). Mirroring research with autistic people, poor sleep, mental health difficulties and autistic traits have been found to interrelate (Hundley et al., 2016; Salmela et al., 2019; Stewart et al., 2020). For instance, older adults with higher degrees of autistic traits reported more severe sleep difficulties and depressive symptoms (Stewart et al., 2020). In adolescents, autistic traits independently predicted diminished sleep duration once anxiety and depression symptoms were controlled for (Salmela et al., 2019). The exact nature of this relationship is unclear; however, the reported interrelations between autistic traits, sleep and depressive symptoms implies that there is potential value to exploring these factors in synchrony (Stewart et al., 2020).

To summarise, depression has been associated with sleep difficulties and autistic traits, but it is not known whether sleep difficulties or higher levels of autistic traits (broad or trait-specific) independently contribute to predicting depression, whether the relationship is explained by shared variance between autistic traits and sleep difficulties, or whether sleep difficulties mediate the relationship between autistic traits and depression. Additionally, research regarding interrelations between depression, autistic traits and sleep thus far has predominantly focused on child and adolescent or older adult samples (Hundley et al., 2016; Salmela et al., 2019; Stewart et al., 2020); little is known about how these variables interact in young adults.

Rationale

Further investigation is necessary to examine a broad range of autistic traits and their unique interrelations with depressive symptomology and sleep difficulties. The issue of how particular autistic traits relate to depression among the general population has not been empirically established; further, little is known about the contribution of sleep difficulties to this relationship. Findings could clarify inconsistencies in the current literature and add to evidence regarding the interplay between specific autistic traits, sleep and risk of depression. By recruiting a sample of university students, findings could further our understanding of how these variables interact in young adults, and also potentially inform screening practices relating to student mental health. Pertinently, there are well-established interventions to improve sleep quality; if sleep quality was found to relate to autistic traits and depression in students, then improving sleep might be a good candidate to reduce mood difficulties within this population.

A further novel aspect of the current study is in the choice of measure of autistic traits. The Social Responsiveness Scale-Second Edition was selected in the current study as a measure of autistic traits in the general population (SRS-2; Constantino & Gruber, 2012). Previous studies have predominantly used the Autism Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin & Clubley, 2001) to examine the relationship between autistic traits and depression; however, the SRS-2 is deemed a more sensitive measure of aspects of social communication and reciprocity (Bruni, 2014). Therefore, the relationship between social function differences and depressive symptoms may not have been adequately explored previously (for instance, Ishiguro et al., 2022). It is of note that Rai et al. (2018) found social communication impairments to predict depression strongly when including a measure of social functioning. To the author's knowledge, this was the first study to use the SRS-2 subscales to measure the relationship between autistic traits, depression and sleep.

Aims

The current study had several aims. In the first instance, the study sought to replicate previous findings regarding positive correlation between a) depression and autistic traits (aim 1), b) depression and sleep difficulties (aim 2), c) autistic traits and sleep difficulties (aim 3).

Following replication, the study aimed to determine whether specific traits (related to RRB or social communication and interaction) independently contribute to this relationship, whether the relationship is explained by shared variance between broad autistic traits and whether sleep difficulties and autistic traits independently predicted depressive symptom scores (aim 4). Finally, the study aimed to understand the role of sleep difficulties in the relationship between autistic traits and depression (aim 5).

Method

Participants

Participants were 223 undergraduate psychology students recruited from the University of Birmingham. Recruitment took place via the university's online Research Participation Scheme system and participants were awarded 0.7 credits in exchange for their participation. Following exclusion of 18 participants due to incomplete or inattentive responses (see section titled 'Data Coding'), 205 participants comprised the final sample. Demographic data is presented in Table 1. Most participants were age 18-30; however, ages ranged from 18-51 years (M= 19.1, SD= 2.9). Participants were mostly female (89.75%) and Caucasian (61.95%). Minimal exclusion criteria were applied for the purpose of studying variation in autistic traits in the general population. All participants who took part in the Research Participation Scheme were invited to take part unless they had a diagnosed neurodevelopmental condition (such as autism) or any diagnosed mental health condition. No other inclusion or exclusion criteria were applied.

Table 1

Participant Demographics

Item	N (%)	Mean (SD)	Range
	(**)		
Age	205	19.12 (2.93)	18-51
C			
Gender			
Female	184 (89.75)		
Male	20 (9.75)		
Other	1 (0.48)		
Ethnicity			
White British	127 (61.95)		
Asian/Asian British	61 (29.75)		
Black	6 (2.43)		
British/African/Caribbean	7 (3.41)		
Mixed/multiple ethnic	3 (1.46)		
groups	1 (0.48)		
Arabian			
Prefer not to say			

Procedure

Participants were invited to complete an online survey via the Qualtrics platform (median response time 13.5 minutes) comprising an information sheet, a consent form, questions about demographics (age, gender, ethnicity), and five questionnaires, followed by a debrief sheet. Responses were recorded between 2nd and 30th of November 2021. The study was part of a wider project which aimed to investigate the impact of sleep on social and emotional functioning. Three questionnaires were relevant to this study; Pittsburgh Sleep Quality Index (PSQI), Social Responsiveness-Scale Second Edition (SRS-2), Patient Health Questionnaire-9 (PHQ-9). Data for the remaining two questionnaires (Generalised Anxiety Disorder Assessment, GAD-7; The Warwick-Edinburgh Mental Wellbeing Scale, WEMWBS) contributed to a related study and are not reported within this paper. The relevant parts of the survey (excluding the SRS-2 due to copyright reasons) can be found in Appendix 4.

Measures

The Patient Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, (2001)

The PHQ-9 is a 9-item abridged self-report measure of depressive symptoms severity developed from the Patient Health Questionnaire (Spitzer, 1999). Items are scored on a 4-point Likert-scale according to frequency of depressive symptoms during the past two weeks (0= 'not at all', 1= 'several days', 2= 'more than half the days', 3= 'nearly every day'). The maximum total score is 27. Scores of 0-4 indicate no depressive symptoms, whilst scores of 5-9 and 10-14 indicate mild to moderate depressive symptoms, respectively. Scores of 15-19 are classed as moderately severe and 20-27 as severe. Equal estimates of sensitivity and specificity are reported at 88% (Kroenke et al., 2001). The measure has good concurrent validity as it was based on criteria from the DSM-IV. Further, high internal consistency (Cronbach α = .89) and test-re-test reliability (.74) have been reported (Sun et al., 2020). High internal consistency was also observed when administering the PHQ-9 to the current study sample (Cronbach α = .88). Items from the PHQ-9 are shown in table 2.

Table 2

Patient Health Questionnaire-9 Example Items and Scoring

Item Example

Over the last two weeks how often have you been bothered by any of the following problems?

- 1 Little interest or pleasure in doing things
- 2 Feeling down, depressed, or hopeless
- 3 Trouble falling or staying asleep, or sleeping too much
- 4 Feeling tired or having little energy
- 5 Poor appetite or overeating
- 6 Feeling bad about yourself or that you are a failure or have let yourself or your family down
- 7 Trouble concentrating on things, such as reading the newspaper or watching television
- 8 Moving or speaking so slowly that other people could have noticed? Or the opposite being so fidgety or restless that you have been moving around a lot more than usual
- 9 Thoughts that you would be better off dead or of hurting yourself in some way

Social Responsiveness Scale–Second Edition (SRS-2; Constantino & Gruber, 2012)

The SRS-2 is a 65-item self-report measure that aims to identify differences in social behaviour synonymous with autistic traits. The measure is made up of five subscales; social awareness, social cognition, social communication, social motivation, and restricted interests and repetitive behaviours (RRB). Items relating to social functioning are scored on a 4-point Likert-scale denoting frequency over the previous six months (0='not true', 1='sometimes true', 2='often true', 3='almost always true'). The maximum total score is 195 with higher scores indicating greater difficulty. Raw scores can be transformed into T-scores that classify levels of social impairment. A T-score <59 (raw scores <67) is classified as typical, whilst a T-score of 60-75 indicates mild to moderate impairment and T-scores >75 is indicative of ASD. The SRS-2 demonstrates good internal consistency (Cronbach α , .76), discriminant validity and test re-test reliability (Bruni, 2014; Constantino 2003). High internal consistency was also observed when administering the SRS-2 to the current study sample (Cronbach α = .85). Classification of social communication impairments using the SRS-2 yield sensitivity and specificity of .85 and .75 respectively (Bölte et al., 2011). When the SRS-2 has been administered to university students, means scores of 31.57 and 41.28 have been reported (Denigris et al., 2017; South, Carr, Stephenson, Maisel & Cox, 2017). Example items from each subscale are displayed in Table 3.

Table 3

Subscale	Item	Example
Social awareness	2	'My facial expressions send the wrong message to others about how I actually feel'
Social cognition	5	'I do not recognise when others are trying to take advantage of me'
Social communication	18	'I have difficulty making friends, even when trying my best'
Social motivation	23	'I do not join group activities or social events unless prompted or strongly urged to do so'
Restricted interests and repetitive behaviours	4	'When under stress, I engage in rigid or inflexible patterns of behaviour that seem odd to people'

Social Responsiveness Scale-Second Edition Example Items and Scoring

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989)

The PSQI is a brief self-report measures that appraises sleep quality over the previous month. The measure consists of 19 items which assess seven components; subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. All items are scored on a scale of 0-3 (no difficulty to severe difficulty). Table 4 presents example items and scales. The maximum total score is 21, with higher scores indicating lesser sleep quality. Scores above the cut-off of five are proposed to accurately (sensitivity 89.6%, specificity of 86.5%) classify those with significant sleep difficulties (Buysee et al., 1989).

Table 4

Pittsburgh Sleep Quality Index Example Items and Scoring

Item	Scale Score		Component		
6. 'During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?'	'Not during the past month' 'Less than once a week' 'Once or twice a week' 'Three or more times a week'	0 1 2 3	Use of sleep medication		
7. 'During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?'	'Not during the past month' 'Less than once a week' 'Once or twice a week' 'Three or more times a week'	0 1 2 3	Daytime Dysfunction		
9. 'During the past month, how would you rate your sleep quality overall?'	'Very good' 'Fairly good' 'Fairly bad' 'Very bad'	0 1 2 3	Subjective sleep quality		

The measure has demonstrated high levels of internal consistency and test re-test reliability (Cronbach α , .83 and .85 respectively) when tested previously (Buysee et al., 1989). When administered to the current study sample, an acceptable level of internal consistency was observed (Cronbach α , .68). The PSQI is reported to provide a measure of sleep quality comparable to clinical and laboratory diagnosis (Buysee et al., 1989). More recent studies using the PSQI with undergraduate students have reported mean scores between 5.23 to 6.41 (Dietch et al., 2016; Famodu et al., 2018; Liu, Kahathuduwa & Vazsonyi, 2021; Rezaei et al., 2018).

Ethical Considerations

Ethical approval was obtained from the University of Birmingham Research Governance Team prior to conducting the study. The participant information sheet described how data would be stored and how participant anonymity and confidentiality of responses would be protected. Participants' right to withdraw without repercussions was also described within the information sheet. The measures were checked by the research team and it was determined that the questions themselves were unlikely to cause distress to participants. However, items relating to mental health and wellbeing may be considered sensitive; therefore, the information and debrief sheets included signposting to appropriate support. Additionally, the information sheet clarified that responses would not be used to screen for clinical conditions and scores above specified cut-offs would not necessitate diagnosis.

Data Coding

From a total of 223 responses, 18 response sets were excluded during the data cleaning process. Ten response sets were excluded on the basis of being incomplete. A further five were excluded for inattentive responding or exhibiting unreasonably brief response times. Responses of 'prefer not to say' were pro-rated for up to 20% of a participant's scores, three response sets exceeded this threshold resulting in exclusion. Following data cleaning, a total of 205 response sets remained.

In 61 cases, it was necessary to calculate the midpoint for PSQI questions 1-4 because participants responded with a range. Component scores were used to pro-rate responses in two cases when inadequate written responses were submitted. In 19 cases, sleep efficiency exceeded 100%; therefore, efficiency was recalculated using the percentage of reported actual sleep from a total of reported actual sleep and sleep latency combined. Component scores were not altered as a result.

In 44 cases, 'prefer not to say' was submitted as a response to 'other reason' for sleep disturbance. When the previous questions were answered, responses were coded as zero. It was assumed that no other reasons were relevant and the response was intended to mean 'not appliable'.

Regarding SRS-2 data, 16 'prefer not to say' responses submitted by 14 participants were pro-rated according to subscale scores.

Data Scoring

For the PSQI, seven component scores were calculated and then summed to obtain a total score (see Appendix 4d). For the SRS-2, 15 items were reverse-scored and then subscale scores were computed and summed to produce a total score. T-scores were calculated to aid interpretation of the data; however, no further statistical analysis was conducted using the scores. The PHQ-9 scores total score was calculated from a sum of scores for each item.

Statistical Analyses

SPSS 28.0 was used to conduct statistical analyses. Examination of plots revealed that SRS-2, PHQ-9 and PSQI data were positively skewed, which is to be expected when administering clinical measures to non-clinical populations. Non-normality was confirmed by significant Shapiro-Wilk tests. Spearman's rank correlations were computed to measure the strength of correlation between scores on the PHQ-9 and SRS-2, scores on the PHQ-9 and PSQI and scores on the SRS-2 and PSQI. Data were checked to determine whether assumptions for multiple regression were met (Dancey & Reidy, 2011; Field, 2009). As the PHQ-9 was the dependent variable, the square root transformation was calculated to obtain normality for regression analysis (Baguley, 2012; Field, 2009). The untransformed scores were used for all other variables. Scatterplots were inspected to confirm homoscedasticity, in addition to linearity between the dependent and predictor variables (residual plots can be found in Appendix 6.12, 6.13). No-multicollinearity of predictor variables was determined by variance inflation factors below 10 and tolerance values above .2 (Menard, 1995; Myers, 1990). Subsequently, multiple linear regression using an automatic modelling approach and the 'Enter' method in which variables were entered simultaneously was performed to explore

associations between depressive symptoms (PHQ-9 scores), sleep difficulties (PSQI total scores) and autistic traits (SRS-2 subscale scores). The 'Enter' method was chosen to explore the relationships between variables, no hypotheses were made about which variables would lead to a stronger prediction equation.

Based on the results of the regression analysis (reported below), mediation analysis was carried out to determine whether sleep mediated the relationship between RRB and depression and whether RRB continued to predict depression when controlling for sleep difficulties.

The requirements for mediation analysis were met; associations between the predictor variable, RRB and a) the dependent variable, depressive symptoms, b) the mediator, sleep difficulties, were evident, as was the association between the aforementioned mediator and dependent variables. Bootstrapped mediation analysis with 5000 samples was conducted using the PROCESS tool for SPSS (Field, 2013; Hayes, 2017). RRB subscale scores of the SRS-2 were entered as the X variable, the square root transformation of PHQ-9 scores as the Y variable, and PSQI total scores as the mediator in mediation model 4 (Hayes, 2017). The significance threshold was adjusted to p < .01 for all statistical tests in the analysis to correct for multiple comparisons.

Results

Sample Characteristics

Mann-Whitney tests were conducted to determine whether total scores across each measure differed according to gender. The one participant classified as 'other' for gender was excluded from this part of the analysis because it would be unlikely that anything meaningful could be concluded from comparing one participant to two other groups. Gender did not significantly impact scores on any of the measures; PHQ-9, $U(N_{\text{female}}=184, N_{\text{male}}=20,) =$

1467.5,
$$z = -1.488$$
, $p = .137$; SRS-2, $U(N_{\text{female}} = 184, N_{\text{male}} = 20,) = 1572.0, z = -1.069, p = .285$; PSQI, $U(N_{\text{female}} = 184, N_{\text{male}} = 20,) = 1767.5, z = -.291, p = .771$. Thus, it was not necessary to report results for each gender separately. Score summaries for each measure are presented in Table 5, below.

Table 5

	Max. Possible	Median Raw	Median T- Score	Interquartile Range	Min. and Max. Scores
PHO-9	27	8		8	0-27
SRS-2 Total Score	195	51	54	34	0-142 (38-86)
Social Awareness	24	6	49	4	0-17 (32-81)
Social Cognition	36	9	53	7	0-21 (37-74)
Social Communication	66	15	52	13	0-52 (37-88)
Social Motivation	33	12	57	10	0-27 (37-82)
RRB	36	8	53	8	0-34 (40->90)
PSQI	21	6		4	0-16

Descriptive Statistics for each scale (N = 205)

^a PHQ-9 depressive symptom severity: <5 non-clinical range, 5-9 mild range, 10-14 moderate range,
15-19 moderately severe, 20-27 as severe.

^b SRS-2 T-Scores: \leq 59 within normal limits of social functioning; 60 – 65 mild range of social impairment; 66-75 moderate range of social impairment; \geq 76, severe range of social

impairment (maximum \ge 90).

° PSQI Cut-off: >5 indicates poor sleep.

The median PHQ-9 score was eight, which falls in the upper end of the mild depressive symptoms range. Other studies conducted with university students have reported

similar scores both before (Fata Nahas, Elkalmi, Al-Shami & Elsayed, 2019; Urasaki et al., 2009) and after the COVID-19 pandemic (Giannopoulou, Efstathiou, Triantafyllou, Korkoliakou, & Douzenis, 2021). Scores were distributed across each range, representing varying levels of depressive symptoms within the general population. Whilst 26% of participants exhibited no depressive symptoms, 32% scored within the mild range, 21% within the moderate range, 12% in the moderately-severe range and 9% in the severe range.

The median SRS-2 total score was within the non-clinical range, indicative of healthy functioning and consistent with previous studies of neurotypical people (Han, Tomarken & Gotham, 2019). Although 75% of participants scored within the non-clinical range, approximately 25% of participants obtained scores reflective of social functioning and behaviour differences associated with autistic traits. Scores in the mild (14%), moderate (6%) and severe (2%) ranges were observed. The broad range of scores obtained within this sample was considered sufficient to explore distribution of autistic traits. Median subscale scores also fell within the non-clinical range. Again, a sufficient spread of scores was observed across non-clinical, mild, moderate and severe ranges.

Poor sleep quality as measured by the PSQI is indicated in scores that exceed the threshold of five. The median global PSQI score was six; approximately 60% of participants were considered to exhibit sleep difficulties. The results were consistent with other studies that have measured sleep quality within a student population (for instance; Maheshwari, & Shaukat, 2019; Zou et al., 2020; Ozcan & Acimis, 2020). The range of scores (0-16) reflected individual differences in sleep quality. Figures 1 and 2 present the distribution of scores alongside thresholds for impairment.

Figure 1

Scatterplot presenting distribution of SRS-2 T-scores and PHQ-9 total scores within the sample, alongside thresholds for impairment as

defined by each scale





Figure 2

Scatterplot presenting distribution of SRS-2 T-scores and PSQI total scores within the sample, alongside thresholds for impairment as

defined by each scale



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Correlation Analysis

Aim 1: Depressive Symptoms will be Positively Correlated with Autistic Traits

Spearman's rank correlation analysis was conducted using SRS-2 and PHQ-9 total scores to investigate the relationship between autistic traits and depressive symptoms respectively. SRS-2 total scores were positively and significantly correlated with PHQ-9 scores; $r_s(203) = .518$, p = < .001 (see Appendix 6.10).

Aim 2: Depressive Symptoms will be Positively Correlated with Sleep Difficulties

Spearman's rank correlation analysis was conducted using PHQ-9 and PSQI total scores to investigate the relationship between depressive symptoms and sleep difficulties respectively. There was a significant positive correlation between PHQ-9 and PSQI total scores; $r_s(203) = .523$, p = < .001 (see Figure 3).

Figure 3



Scatterplot of the Relationship Between PHQ-9 and PSQI Total Scores

Pittsburgh Sleep Quality Index Global Score

Aim 3: Autistic traits will be positively correlated with sleep problems

Spearman's rank correlation analysis was conducted using SRS-2 and PSQI total scores to investigate the relationship between autistic traits and sleep difficulties respectively. Higher scores on the PSQI indicated more sleep difficulties. There was a significant positive correlation between SRS-2 and PSQI total scores; $r_s(203) = .298$, p = < .001 (see Appendix 6.11).

Regression Analysis

Aim 4

To determine the relationships between depressive symptoms and a) autistic traits, b) sleep difficulties, using the 'Enter' method all SRS-2 subscales (social awareness, cognition, communication, motivation and RRB) and PSQI total scores were simultaneously entered as predictor variables in multiple linear regression, with square root transformed PHQ-9 total scores as the dependent variable. Table 6 presents Spearman's rank correlations between predictor variables, and intercorrelations between predictor variables.

Table 6

Correlations and Intercorrelations between PHQ-9 Scores and Predictor Variables (N = 205)

	Variables	1	2	3	4	5	6
1.	PHQ-9 score (square root)						
2.	Awareness score	.347**					
3.	Cognition score	.479**	.583**				
4.	Communication score	.480**	.583**	.693**			
5.	Motivation score	.442**	.373**	.477**	.725**		
6.	RRB score	.570**	.620**	.720**	.662**	.546**	
7.	PSQI score	.551**	.222*	.240**	.239**	.216*	.277**

*Significant at p < .01, **significant at p < .001

The association between depressive symptom scores on the PHQ-9 and predictor variables (SRS-2 subscales and sleep quality scores) was strong (Multiple R = .72). As shown

in Table 6, each SRS-2 subscale was positively and significantly related to depressive symptoms, but the only predictor that remained in the model was RRB. A significant regression equation was found, F(6, 198) = 35.199, p < .001), with an R² of .516, providing a model for depressive symptom scores significantly better than chance (Figure 4).

Figure 4





Together, SRS-2 subscale scores and sleep difficulties accounted for 50% of the variation in depressive symptoms scores (adjusted R^2). The model indicated that sleep difficulties and RRB were positively and significantly related to depressive symptom scores, but sleep difficulties were the stronger predictor. The regression coefficient for sleep difficulties was .146 (t = 8.034; p < .001; 95% CI [.110, .182]); whilst for RRB it was .066 (t = 4.296; p < .001; 95% CI [.036, .096]). Results of the model are presented in Table 7 below.

Table 7

Model	Variables Entered	R^2	Unstandardised Coefficients		Standardised Coefficients	t	Significance
			В	Std.	—		
				Error			
1.	Awareness	.516	033	.178	088	-1.301	<i>p</i> = .195
	Cognition		.020	.025	.084	1.052	p = .294
	Communication		.008	.019	.066	.715	<i>p</i> = .476
	Motivation		.019	.011	.109	1.472	<i>p</i> = .143
	RRB		.066	.013	.347	4.296	<i>p</i> < .001
	Sleep quality		.146	.015	.416	8.034	<i>p</i> < .001

Regression Model Summary

Aim 5: What Is the Role of Sleep Difficulties in the Relationship Between Autistic Traits and Depression?

Significant correlations between RRB and depressive symptoms, RRB and sleep difficulties, and depressive symptoms and sleep difficulties, satisfied the criteria for mediation analysis. The analysis found that sleep difficulties significantly mediated the association between RRB and depressive symptoms. RRB predicted depressive symptoms, b = .086, 95% CI [.066, .106], t(202) = 8.68, p < .0001. Both a and b paths were significant, meaning that RRB significantly impacted sleep difficulties (b = 0.15, 95% CI [.08, .22], t(203) = 4.1, p < .001), and sleep difficulties significantly impacted depressive symptoms (b = .15, 95% CI [.11, .19], t(202) = 8.18, p < .0001).

The indirect effect was significant, the 95% confidence interval did not include zero, confirming that sleep difficulties mediate the relationship between RRB and depressive symptoms, b = .02, 95% CI [.01, .04]. After accounting for the mediating role of sleep difficulties, RRB significantly predicted depressive symptoms, b = .02, CI [.01, .04], t(202) = 8.68, p = < .0001, meaning the relationship is not fully mediated. The standardised coefficient for the direct path was substantially larger than that for the indirect path; the direct effect of

RRB was the stronger predicter, than that accounting for sleep. Figure 5 summarises the mediation model.

Figure 5

Mediation Model of The Relationship Between RRB, Sleep Difficulties and Depression



Indirect effect = .0224

To summarise, each subscale of the SRS-2 correlated with depressive symptom scores and sleep difficulties, indicating that all variables are interrelated and the relationship between autistic traits and depression is explained by shared variance between broad autistic traits. RRB was the only SRS-2 subscale that remained in the regression model with sleep difficulties. The model demonstrated that RRB and sleep difficulties both independently contribute as predictors of depression. Finally, the mediation analysis revealed that the relationship between RRB and depression is partially mediated by sleep difficulties; however, the direct relationship between RRB and depression remained significant once sleep had been controlled for.

Discussion

This study measured interrelations between autistic traits, depressive symptoms and sleep difficulties in the general population. Consistent with previous studies, people with higher levels of autistic traits exhibited more depressive symptoms and more sleep difficulties (Hundley et al., 2016; Kanne et al., 2009; Kunihira et al., 2006; Salmela et al., 2019; Stewart et al., 2020; Verhoeff et al., 2018). Further, aligning with previous studies of the general population, a positive and significant association was observed for depressive symptoms and sleep difficulties (Baglioni et al., 2011; Gregory et al., 2002; Jackson et al., 2014; Lovato et al., 2017; Marino et al., 2021).

The findings of this study replicate previously reported association between high levels of broad autistic traits in clinically depressed people (Atherton et al., 2015; Ishizuka et al., 2022; Matsuo et al., 2015; Radke et al., 2019), and neurotypical university students with depressive symptoms (Kanne, Christ & Reiersen, 2009). However, the current study extends replication of findings to the use of the SRS-2 as a dimensional measure of autistic traits in the general population. The SRS-2 aligns with DSM-5 conceptualisation of autism and has been empirically cross-culturally validated and norm-referenced, demonstrating reliability and validity of the summative measure and its subscales (Duku et al., 2013; Shahrivar et al., 2020; Tehrani-Doost, Shahrivar, Torabi, Ansari, Haji-Esmaeelzadeh & Saeed-Ahmadi, 2020). Arguably, the SRS-2 provides more detailed, reliable insights about subcomponents of autistic traits in the general population than total scores on the AQ (Baren-Cohen et al., 2001), which has been heavily relied upon in previous studies (Frazier et al., 2014a, 2014b).

Findings bear similarity to studies reporting high rates of co-morbid depression among autistic people (Brookman-Frazee et al., 2018; Crane et al., 2013; Hollocks, Lerh, JMagiati, Meiser-Stedman& Brugha, 2019; Joshi et al., 2010). Such associations reflect the common features of autism and depression. Measures of autistic traits, such as the SRS-2, are designed to detect subcomponent traits of autism; for instance, differences in social functioning. Yet social withdrawal and differences in empathy and reciprocity are also characteristic of depression. Therefore, correlation between autistic traits and symptoms of depression is perhaps unsurprising, but less known are factors contributing to this shared phenomenology.

A novel aspect of this study involved using the SRS-2 at the subscale level, to explore whether particular traits were independently related to depressive symptoms. The value of using the SRS-2 here is that it allows for conceptualisation of autistic traits into a range of social and cognitive behaviours, abilities or predispositions, as opposed to focusing on whether a person meets a clinical threshold or not. The analysis highlighted significant positive correlations between each subscale of the SRS-2 and depressive symptoms, supporting the notion that shared variance across autistic traits conceptualised by SRS-2 subscales predict the relationship with depression. Subcomponent traits related to the dyad of impairments upon which autism is diagnosed (social awareness, cognition, communication and motivation, and RRB) were all associated with depressive symptoms.

Consistent with previous studies using different measures of autistic traits (e.g., the AQ), regression analysis revealed that RRB was the strongest trait-related predictor of depressive symptoms (Andersen et al., 2015; Gardiner et al., 2018; Gotham et al., 2015; Pouw et al., 2013; Reiffe et al., 2014). One potential explanation for this emerges from the well-established predictive relationship between adverse life events and depression in neurotypical and autistic people (Fung, Lunsky & Weiss, 2015). It is possible that subcomponent traits related to RRB which affect emotional regulation, such as cognitive inflexibility and repetitive cognition and behaviour, impede adjustment following adverse life events, creating the sense of being stuck in a cycle of negative thoughts and behaviours (Fung et al., 2015;

South et al., 2019). Pertinently, it has been reported that autistic children were found to experience more adverse life events in the year preceding depression onset (Fung et al., 2015; Ghazziuddin et al., 1995).

A further possibility is that insistence on sameness and stereotyped behaviours may reflect a preference for predictability in the environment and serve as maladaptive coping mechanisms for depressive symptoms; these coping mechanisms may hinder abstract thinking about how circumstances might be different in future and exacerbate distress (Joyce, Honey, Leekam, Barrett, & Rodgers, 2017; South et al., 2019; Stratis & Lecavalier, 2013). Therefore, if traits related to RRB are implicated in coping response then RRBs may occur at a higher rate during periods of depressed mood (Joyce et al., 2017). Indeed, in studies of autistic people, rates of self-injurious behaviour, which is considered to represent RRB, were found to predict depression and were conceptualised as an expression of internal distress (Muskett, Capriola-Hall, Radtke, Factor, & Scarpa, 2019).

In the final regression model, when controlling for RRBs, traits relating to social communication and interaction differences did not explain additional variance in depressive symptoms. This is likely in part explained by the high degree of overlap between RRB and, social and interaction difficulties (correlations .55 - .72 in the current sample). Though explained by shared variance with RRB, social communication and interaction difficulties clearly did correlate with depressive symptoms, consistent with previous evidence. This relationship has been demonstrated previously among a sample of adolescents; however, the study emphasised that social communication differences were associated with the highest risk for depression (Rai et al., 2018). Given that the majority of participants in the current study were young adults, it is likely that other factors, such as age, influence relationships between autistic traits and depression. Arguably, social skill deficits may be more impactful at a time

when fitting in and forming peer relationships have such high importance. Additionally, Rai et al. reported that the experience of being bullied during school years was found to mediate risk for depression among adolescents with higher autistic traits (Borowsky, Taliaferro, & McMorris, 2013). Therefore, it is possible that particular autistic traits may be more closely associated with development of depression at particular life stages. In the current study, of the autistic traits conceptualised by the SRS-2 subscales, only RRB remained in the regression equation as a predictor of depression. This implies that shared variance between social subcomponents of autistic traits and depression is also largely shared with individual differences in RRB. That is not to say that difficulties with social communication and interaction are not linked to depression, but rather those young adults experience these difficulties in the absence of restricted or repetitive behaviours or interests may not be more likely to experience a higher level of depressive symptoms than their peers.

With regard to sleep, consistent with other studies of the general population, sleep difficulties were found to be positively and significantly related to depressive symptoms (Baglioni et al., 2011; Dinis, J., & Bragança, 2018; Jackson, Sztendur, Diamond, Byles & Bruck, 2014). Previous findings defining directionality in this relationship are mixed; therefore, a reciprocal relationship has been proposed (Dinis et al., 2018). One way in which sleep difficulties might lead to depression is through lessening cognitive control and ability to disengage from negative cognition (Vanderlind et al., 2013). Conversely, depressive symptoms are numerous and as such, may impact sleep in a number of ways; one such example may be that excess rumination causes difficulty falling to sleep (Thorsteinsson, Brown & Owens, 2019).

In the autism literature, traits related to RRB, such as ritualistic behaviour have been found to cause bedtime resistance and difficulties settling at night time, with lesser sleep predicting more stereotypic behaviour (Schrek, Mullick & Smith, 2004; Richdale, 2001; Tsai et al., 2021); therefore, as well as contributing to depression (as discussed above), RRB could contribute to sleeping difficulties. Pertinently, the regression analysis in the current study revealed that sleep difficulties and RRB independently contributed to predicting depressive symptoms. Further, the mediation analysis confirmed that sleep difficulties partially mediate the relationship between RRB and depressive symptoms, but the direct relationship between RRB and depressive symptoms remained even when sleep difficulties were controlled for.

These novel findings support the notion that shared variance among autistic traits (predominantly RRB) and sleep difficulties both independently predict depressive symptoms, but the direct relationship between RRB and depressive symptoms accounts for more of the variance in depressive symptoms scores than that mediated by sleep difficulties.

Interconnectedness of these factors in people with higher levels of autistic traits may be understood in the context of the biopsychosocial model of sleep difficulties in autism. The model posits that sleep difficulties have an array of inter-related biopsychosocial determinants, including biological, genetic, environmental, psychological and behavioural factors. Any one or combination of these factors may have reciprocal relationships with autistic symptoms and have a precipitating or predisposing impact on sleep difficulties in autistic people (Richdale & Schrek, 2009; Schrek & Richdale, 2020).

Therefore, this may suggest that a reciprocal relationship exists between autistic traits and sleep quality, in which RRB contributes to sleep difficulties and subsequently sleep difficulties intensify RRB. Depression in neurotypical people has been associated with similar problems; for instance, settling difficulties and reduced sleep duration (Sheldon, Ferber & Kryger, 2005). Drawing on previous research indicating that sleep difficulties precede depression (Gregory et al., 2002; Lovato et al., 2017; Marino et al., 2021) and given the correlation between depressive symptoms and sleep in the current study and elsewhere (see Dinis et al., 2018 for a review), it is possible that difficulty sleeping contributes to depressive symptoms that become entrenched due to rigid and repetitive cognition. In addition, findings that report insomnia to worsen mental health symptoms (Baglioni et al., 2011; Jackson et al., 2014) may indicate a further reciprocal relationship between depressive symptoms and sleep disruption. The proposed explanation may account for reported improvements to mood once insomnia is treated (Paavonen, Nieminen-von Wendt, Vanhala, Aronen & von Wendt, 2003).

Consistent with conceptualisation of RRB as a coping mechanism and literature reporting that depressive symptoms are positively associated with features of RRB (Joyce et al., 2017; Muskett et al., 2019; Straits et al., 2013), depression may then also impact frequency and severity of RRB; thus, completing a vicious cycle between RRB, sleep difficulties and depressive symptoms. However, as suggested by Richdale et al. (2009), the relationship between autistic traits, depression and sleep difficulties is likely to be complex and multifaceted. The explanation here focuses on only one trait and one aspect of this model; therefore, further research encompassing more varied factors may enable deeper, more intricate understanding of the interplay between biopsychosocial factors.

Clinical Implications

The current study has replicated broad associations between degree of autistic traits, sleep difficulties and depression in a young adult population; whilst also expanding findings by demonstrating how variation in subcomponent traits of autism, particularly RRB, may be key to understanding risk of depression in a student population. Such findings demonstrate how the SRS-2 could helpfully contribute to mental health screening practices in universities by highlighting social and cognitive features relevant to depression, and enabling student

wellbeing services to identify students with higher degrees of RRB and sleep difficulties who may (now or in the near future) experience greater depressive symptoms than students with social communication and sleep difficulties and lesser degrees of RRB. Gathering this information could enable services to target resources to those in need, as well as proactively provide support at an earlier stage to those at greater risk. Given the interrelatedness of autistic traits, sleep difficulties and depressive symptoms observed in this study, offering interventions that focus on improving sleep quality may reduce mood difficulties and could be a practical starting point for student wellbeing services.

The findings here also highlight the importance of clinicians considering the potential impact of these factors on treatment. Cognitive behavioural therapy is a recommended treatment for depression. When considering maintenance cycles within this framework, RRBs may perpetuate depressive symptoms or create barriers to adopting more helpful cognitions or behaviours; therefore, working to enhance cognitive flexibility may be of benefit. Conversely, for some people it may be helpful to focus less on cognition and more on supporting them to regularly engage in a valued activity through behavioural activation.

Methodological Limitations

It is important to note strengths and limitations when considering the clinical implications of the current study. This study added to the literature by demonstrating associations between specific autistic traits, sleep difficulties and depressive symptoms when using the SRS-2. In its original and updated form, the Social Responsiveness Scale is a sensitive and reliable screening measures with robust psychometric properties and cross-cultural validity (Chan, Smith, Hong, Greenberg, & Mailick, 2017; Constantino et al., 2003; Duku et al., 2013; Shahrivar et al., 2020; Tehrani-Doost et al., 2020). The empirically derived subscales align with DSM-5 criteria and have been found to exhibit criterion and construct

validity, as well as better incremental validity than relying on the total score alone (Chan et al., 2017; Frazier et al., 2014a, 2014b). Previous studies have predominantly relied on the Autism Quotient (AQ; Baren-Cohen et al., 2001) to measure degrees of autistic traits in the general population. Unlike the SRS-2, the AQ lacks normative data and reliability of the subscales is questionable, indicating that further development of the instrument is needed (Stewart & Austin, 2009). Aside from the problem with building an edifice of what autistic traits relate to from a single measure, the AQ has been found to be less effective when used to measure autistic traits, compared to when used to discriminate between autistic and neurotypical people (Lundqvist & Lindner, 2017). Additionally, there is a propensity for studies to report the total score on the AQ, but this may be too simplistic and not accurately reflect the multi-faceted nature of the autism construct (Lundqvist et al., 2017).

Regarding methodology, use of an online survey facilitated the collection of data from a large cohort of people, enhancing representativeness of the sample. As with all designs, the cross-sectional nature of this study had particular costs and benefits. Despite mediation analysis showing the strength of variables to predict depression, causation cannot be inferred leading to mainly descriptive interpretation of findings, limited to the variables studied. However, the benefits of a cross-sectional design in this context should not be underestimated. It enabled detailed information on the relationship between co-occurring variables relevant to depression in the short-term; for instance, measuring RRB and sleep difficulties can provide useful information about whether a person is currently depressed, their likelihood of developing depression in the near future and which social or cognitive features to be aware of when screening for depression.

The correlational nature of the study, combined with reliance on self-report measures, may be considered a limitation due to the possibility of common-method variance. Commonmethod variance refers to the variance attributable to the method itself rather than the variables being measured and can lead to false relationships being observed between variables (Podsakoff, MacKenzie, Lee & Podsakoff, 2003). However, relationships between autism, autistic traits, depression and sleep are well documented in the literature from studies using differing methodology (for instance; Rai et al., 2018; Shanahan, Copeland, Angold, Bondy, & Costello, 2014). Additionally, common method variance may be less relevant since the current study found differing relationships for different variables.

In relation to use of self-report measures in this study, although the pitfalls of relying on self-report data are well-known, in clinical practice such measures are commonly used owing to practicality and contribution of useful information to clinical decision making; thus, findings highlight the use of screening for autistic traits (predominantly RRB) and sleep difficulties when identifying students that may be at risk of developing depression.

When selecting measures, consideration was given to the psychometric properties, with each demonstrating good reliability and validity. Additionally, it is not clear whether objective measures would enhance the strength and reliability of conclusions in the current study. For instance, autism assessment is commonly conducted through behavioural observations and structured interviews; however, these same methods do not measure autistic traits and still involve a degree of subjectivity from the rater. Additionally, objective sleep measures such as actigraphy may be less impacted by inaccurate recall (Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Gotham et al., 2015), but aside from being costly and requiring greater commitment from participants, discrepancy is also evident in the reporting of sleep factors when using these methods (Fekedulegn et al., 2020), and measuring habitual sleep parameters does not necessarily capture a person's experience of their sleep. With regard to subjective measurement of depressive symptoms, there are also costs and benefits. Whilst it could be argued that a mental health professional may more accurately assess levels of depressive symptoms given exposure to varied presentations and severities of mental distress, it is also claimed that subjective measures provide greater access to a person's inner world (Paykel & Norton, 1986).

Future Directions

Measuring autistic traits and co-occurring factors in the general population is useful because it facilitates the dimensional study of co-occurring phenomena that is not confined to diagnostic boundaries. This transdiagnostic approach moves away from one focused on core deficits and will likely lead to the development of a more balanced, complex and accurate conceptualisation of the autism construct (Astle & Fletcher-Watson, 2020).

The current study was the first to use the SRS-2 subscales to explore associations between autistic traits, sleep difficulties and depressive symptoms. Given that the SRS-2 has more favourable psychometric properties than the AQ when measuring autistic traits, particularly at the subscale level, a more refined understanding of how subcomponent traits covary with biopsychosocial factors may be gained from using the SRS-2 in place of the AQ in future studies. Replicating the findings of the current study with a larger sample and broader participant age range would help to generalise conclusions more widely to adults of ranging ages. When considering the findings of Rai et al. (2018) who reported that social interaction differences strongly predicted depression among adolescents, it may be useful to conduct a longitudinal study which explores stability of interrelations over time and developmental trends that may be influenced by age-relevant contextual factors.

It is likely that many factors interact to result in the constellation of differences observed in autistic people or people with higher levels of autistic traits; therefore, studies considering a broad range of factors will be beneficial. To date, research supports the biopsychosocial model proposed by Richdale et al. (2009). In order to gain a more comprehensive understanding of the mechanisms underlying associations between autistic traits, sleep and depression, it will be important for future studies to explore biological and genetic processes in unison with physiological, psychological and behavioural factors (Schreck et al., 2020)

Conclusions

The results of this study propose that greater levels of autistic traits, particularly RRB, predict likelihood of experiencing depressive symptoms; however, this relationship is complex and likely subject to multi-factorial biopsychosocial influences. Among such influences, are sleep difficulties, which were found to partially mediate the relationship between RRB and depressive symptoms, although the direct relationship between RRB and depressive symptoms remained once sleep difficulties were controlled for. Use of the SRS-2 subscales facilitated a more nuanced exploration of the association between autistic traits and depression, indicating that propensity towards features of RRB (e.g., cognitive inflexibility and repetitive cognition) predicts depression more so than social communication and interaction differences. Findings highlight the potential benefit of measuring autistic traits, particularly RRB, when screening for depression or identifying those at the greatest risk. Additionally, it is important to consider the perpetuating impact of RRB on depression and how features of RRB may impact treatment. A biopsychosocial approach incorporating the SRS-2 subscales to explore interrelations between subcomponents of autistic traits and co-occurring phenomena is needed to reach a more comprehensive understanding.

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Chapter 3

Press Release for Literature Review

Word Count: 593

University of Birmingham News Release

Anxiety And Depression Rates Among Young Autistic People Exceed Global Rates for Young People

Anxiety and depression are highly prevalent among young autistic people, emphasising the need for appropriate treatment and support.

A recent review of 56 studies published in scientific literature estimated that approximately 40% of young autistic people experience significant anxiety and 24% experience significant depression. Previously reported rates of anxiety and depression among young people without Autism are much lower at 7% and 3% respectively (Polanczyk et al., 2015).

The most common types of anxiety experienced by young autistic people were reported to relate to extreme fears of specific objects or situations (23%), particularly social situations (15%), constant feelings of worry (15%) and fear of separation from a valued person, such as their parent (11%). Persistent sadness in both milder (5%) and more severe (8%) forms were also more common.

It is not known why young autistic people experience anxiety and depression more frequently than their peers. However, some theories point to similarities in thinking style between people with autism and people with anxiety and depression; for instance, rigid or biased thinking style, sometimes referred to as 'black and white thinking' (Stark, Stacey, Mandy, Kringelbach & Happé, 2021). Another possibility is exposure to stress or adverse life events (DeFilippis, 2018); it has been found that the experience of being bullied is more common among young autistic people and that negative life events predate development of depression (Forrest, Kroeger & Stroope, 2020).

The review explored whether anxiety and depression rates were affected by differences relating to participant characteristics such as age and sex, and whether participants with intellectual disabilities were included in the study or not. The only factor found to have an impact was age. Rates of phobia were less common in older children; however, severe depression was more common with age, indicating that older age, or greater exposure to difficult life events present a risk factor for severe depression.

An interesting finding was that rates of anxiety and depression among young autistic people differed according to who was reporting. For both anxiety and depression, parents tended to provide the highest ratings, followed by the young person's own rating and then the rating of a mental health professional. However, it was highlighted that few studies actually asked young people to report on their own mental health and most studies used professional ratings, which may underestimate how common anxiety and depression are within young autistic people.

Variability in the way studies were conducted may have had an impact on the rates of anxiety and depression reported. Also, it is unclear whether findings in relation to sex and intellectual disability would have been different if studies had been clearer when describing their participants. It will be important for these factors to be explored further in future. Concerningly, since the COVID-19 pandemic, mental distress in young people is reported to have increased. It would be helpful to update anxiety and depression rates as new evidence emerges to see whether there have been significant changes over the course of the pandemic (Santomauro et al., 2021).

ENDS

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Chapter 4

Press Release for Empirical Research Paper

Word count: 621

University of Birmingham News Release

Student Mental Health: Screening for Autism Traits and Sleep Problems Predicts Which Students Are at Risk of Developing Depression

A research study involving 205 university students revealed that students who reported more sleep problems and who scored higher on a questionnaire measuring 'traits' that are associated with features of autism, were more likely to report symptoms of depression.

Having traits of autism is not the same as having a diagnosis of autism. To greater or lesser degrees these traits exist in all of us. When thinking about this, it may help to imagine lots of volume dials with different settings that represent the natural variation of autism traits in the population.

Results showed that differences in social skills (also a feature of autism) were associated with depression, but the trait that predicted depression most related to a feature of autism known as 'repetitive and restricted behaviour' (RRB).

RRB involves a more rigid thinking style, specific interests, tendency towards repetitive thoughts or patterns of behaviour and difficulty tolerating uncertainty or change. Sleep problems predicted depression too, and were found to play a role in the link between RRB and depression, but when examined altogether, the strongest predictor of depression was scoring highly for RRB.

Why might this be the case? One possible explanation is that people with more or higher levels of these traits have particular styles of thinking and behaving which overlap in some ways with the thought and behaviour patterns observed in people with depression, but we don't yet know why. For instance, people with depression may ruminate more often, experience negative thoughts that reoccur repeatedly, or report feeling stuck in negative cycles of behaviour. RRB also involves repetitive thoughts and patterns of behaviour, so having a predisposition to this style of thinking and behaving may increase the risk of developing depression over and above other people.

Additionally, previous research has shown that sleep problems intensify RRB, which might be why sleep problems were implicated in the relationship between RRB and depression in this study; however, at this stage it is not clear whether other biological, psychological or social factors are also involved (Schrek, Mulick & Smith, 2004).

So, what does all of this mean? Well, for staff at universities and people who work in mental health roles, it means that looking out for RRB and sleep problems is important. Occasional screening for autism traits via a questionnaire known as the Social Responsiveness Scale – Second Edition (SRS-2; Constantino & Gruber, 2012); and perhaps also screening for sleep problems by using a questionnaire like the Pittsburgh Sleep Quality Inventory (PSQI; Buysse et al., 1988), might help universities to identify those who are showing signs of depression or who are risk of developing depression in the near future. Additionally, when treating

depression, it might be helpful to know whether a person scores highly for RRB as treatment may need adapting slightly. For instance, Cognitive Behavioural Therapy is a recommended talking therapy for depression which helps people to become aware of distressing patterns of thoughts and behaviour and adopt more helpful ways of coping. People that score highly for RRB may find it harder to make these changes than others.

This study was the first to relate specific traits of autism to depression and sleep problems in university students, as previous studies have focused on children and broad autism traits. Of course, more research needs to be conducted to replicate these findings; however, measuring variation in these traits in the population may help to deepen understanding of how and why mental health difficulties co-occur with them so frequently.

ENDS

For media enquiries please contact Lisa Blatchford, School of Psychology, University of Birmingham, tel:

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Chapter 5: Appendices

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Appendix 1: Letter from ethics committee granting full ethical approval for the

research

From: Susan Cottam (Research Support Services)
Sent: 02 November 2021 10:49
To: Andrew Surtees (Psychology) <
Subject: Ethics Application ERN_09-719AP28

Dear Dr Surtees

Re: "The relationship between sleep, well-being and social cognition" Ethics Application ERN_09-719AP28

Thank you for the above application to use Programme of Work ERN_09-719P. This has now been considered by the Science, Mathematics, Engineering and Technology Ethical Review Committee.

On behalf of the Committee, I can confirm a favourable ethical opinion for this application.

I would like to remind you that any substantive changes to the nature of the study as described in the Application for Ethical Review, and/or any adverse events occurring during the study should be promptly brought to the Committee's attention by the Principal Investigator and may necessitate further ethical review.

Please be aware that whilst Health and Safety (H&S) issues may be considered during the ethical review process, you are still required to follow the University's guidance on H&S and to ensure that H&S risk assessments have been carried out as appropriate. For further information about this, please contact your School H&S representative or the University's H&S Unit at <u>healthandsafety@contacts.bham.ac.uk</u>.

If you require a hard copy of this correspondence, please let me know.

Kind regards

Mrs Susan Cottam

Research Ethics Manager Research Support Group University of Birmingham Email:

Video/phone: If you would like to arrange a Teams/Zoom/telephone call, please email me and I will get in touch with you as soon as possible.

Web: <u>https://intranet.birmingham.ac.uk/finance/RSS/Research-Support-Group/Research-Ethics/index.aspx</u>

Postal address: Mrs Sue Cottam, Finance Office, University of Birmingham, c\o Room 106 Aston Webb, B Block, Edgbaston, Birmingham, B15 2TT.

Appendix 2: Participant Information Sheet

Before taking part in this study, it is important for you to understand why it's being conducted and what it will involve.

What will my participation involve if I agree to take part?

You are being invited to take part in an online research study that aims to help us better understand the relationship between sleep quality, mood and social cognition. You will be asked to answer questions about the quality of your sleep, your emotions, wellbeing and how you interact in social situations, across three questionnaires. Participation in the study typically takes around 20 minutes. You will be rewarded 0.4 credits on completion of this study. Anyone aged 18-65 can take part. Participation is voluntary and you are free to withdraw at any point should you choose to do so, by exiting the questionnaire. Withdrawal will not incur any penalty. You will receive credit for the proportion of questions you have completed.

What will happen to my responses?

Your responses to the questions will be stored in an electronic database alongside the answers of other participants in this study. You will be known by your Participation ID only, and your data will not be stored with any personally identifiable data about you. You have the right to ask that any data you have contributed be withdrawn within two weeks after completing this study. To do so, please contact the research team with your RPS ID number. Anonymised data will be stored for up to 10 years before being destroyed, in accordance with the University of Birmingham policy. You maintain the right to refuse to respond to any question that is asked of you by exiting the questionnaire.

What are the potential benefits and risks of me taking part?

By taking part in research studies, you gain valuable experience of being a participant and understanding of how psychological research takes place. There are no known risks for you in this study. Questions in the study will be related to anxiety, sleep, well-being, emotions and social cognition. We do not expect this to cause distress in any participants. On the chance that it does, support can be sought from Birmingham Healthy Minds (https://www.bsmhft.nhs.uk/ourservices/29irmingham-healthy-minds/self-referral/), Birmingham Nightline (https://www.guildofstudents.com/studentgroups/societies/nightline/) or from your General Practitioner (doctor).

This project follows protocols approved by the University of Birmingham Research Ethics Committee. If you have any questions as a result of reading this information sheet, please feel free to contact the researchers via email: Komal: Lottie: or the Principal Investigator, Dr Andrew Surtees (Lecturer in

Psychology): a.surtees@bham.ac.uk

Appendix 3: Consent Form

By signing below, you agree that:

- I have read and understood the Participant Information Sheet.

- I understand that I maintain the right to withdraw at any point in the study and can refuse to answer any question without any consequence.

- I understand that I can request for my data to be withdrawn and destroyed within two weeks after completing the study.

- I understand the purpose and nature of the study. I have had the opportunity to ask questions and have had them answered.

– I understand that all personal information will remain confidential and data will be anonymised.

- I understand that I am free to contact any of the researchers or the Principal Investigator for further clarification and information.

- Having read the above, I consent to taking part in this study

Appendix 4: Questionnaire used for the Empirical Research Paper

4a. Demographic Questions

- 1. What is your age? If you'd prefer not to answer, please type 'prefer not to say'.
- 2. What gender do you identify with?
 - a. Male
 - b. Female
 - c. Non-binary
 - d. Other
 - e. Prefer not to say
 - f. Please specify your ethnicity.

a. White

- b. Asian/Asian British
- c. Black/African/Caribbean/Black British d. Mixed/multiple ethnic groups
- e. Other ethnic group, please specify:
- f. Prefer not to say

4b. Patient Health Questionnaire (PHQ-9)

PATIENT HEAL	TH QUESTIONNAIRE	- 9
(PHQ-9)		

Over the <u>last 2 weeks</u> , how often have you been bothered by any of the following problems? (Use "✔" to indicate your answer)	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
 Feeling bad about yourself — or that you are a failure or have let yourself or your family down 	0	1	2	3
 Trouble concentrating on things, such as reading the newspaper or watching television 	0	1	2	3
 Moving or speaking so slowly that other people could have noticed? Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual 	0	1	2	3
 Thoughts that you would be better off dead or of hurting yourself in some way 	0	1	2	3
For office codi	NG <u>0</u> +	+	·+	

=Total Score: _____

4c. Pittsburgh Sleep Quality Index (PSQI)

Pittsburgh Sleep Quality Index (PSQI)

Buysse, D. J., Reynolds III, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1988). The Pittsburgh Sleep Quality Index: A New Instrument for Psychiatric Practice and Research. *Psychiatry Research, 28*, 193-213.

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

Requiring written responses.

- 1. During the past month, what time have you usually gone to bed at night?
- 2. During the past month, how long (in minutes) has it usually taken you to fall asleep?
- 3. During the past month, what time have you usually gotten up in the morning?
- 4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

Scale: Not during the past month; less than once a week; once or twice a week; three or more times a week.

- 5. During the past month, how often have you had trouble sleeping because you...
 - a. Cannot get to sleep within 30 minutes
 - b. Wake up in the middle of the night or early morning
 - c. Have to get up to use the bathroom
 - d. Cannot breathe comfortably
 - e. Cough or snore loudly
 - f. Feel too cold
 - g. Feel too hot
 - h. Have bad dreams
 - i. Have pain
 - j. Other reason(s), please describe
- 6. During the past month how often have you taken medicine to help you sleep (prescribed or "over the counter")?
- 7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

Scale: No problem at all; only a very slight problem; somewhat of a problem; a very big problem.

8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

Scale: Very good; fairly good; fairly bad; very bad.

9. During the past month, how would you rate your sleep quality overall?

Appendix 4d: PSQI Scoring Instructions

In scoring the PSQI, seven component scores are derived, each scored 0 (no difficulty) to 3 (severe difficulty). The component scores are summed to produce a global score (range 0 to 21).

Component 1: Subjective sleep quality-question 9

Response to Q9	Component 1 score
Very good	0
Fairly good	1
Fairly bad	2
Very bad	3

Component 2: Sleep latency-questions 2 and 5a

Response to Q2	Component 2/Q2 subscore
\leq 15 minutes	0
16-30 minutes	1
31-60 minutes	2
> 60 minutes	3

Response to Q5a	Component 2/Q5a subscore
Not during the past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3
Sum of Q2 and Q5a subscores	Component 2 score

Sum of Q2 and Q5a subscores	Component 2 se
0	0
1-2	1
3-4	2
5-6	3

Component 3: Sleep duration-question 4

Response to Q4	Component 3 score
> 7 hours	0
6-7 hours	1
5-6 hours	2
< 5 hours	3

Component 4: Sleep efficiency-questions 1, 3, and 4

Sleep efficiency = (# hours slept/ # hours in bed) X 100%

hours slept – question 4

hours in bed – calculated from responses to questions 1 and 3

Sleep efficiency	Component 4 score
> 85%	0
75-84%	1
65-74%	2
< 65%	3

Component 5: Sleep disturbance-questions 5b-5j

Questions 5b to 5j should be s	scored as follows:
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Sum of 5b to 5j scores	Component 5 score
0	0
1-9	1
10-18	2
19-27	3

Component 6: Use of sleep medication-question 6

Response to Q6	Component 6 score
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Component 7: Daytime dysfunction-questions 7 and 8

Response to Q7	Component 7/Q7 subscore
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3
Response to Q8	Component 7/Q8 subscore
No problem at all	0
Only a very slight problem	1
Somewhat of a problem	2
A very big problem	3
Sum of Q7 and Q8 subscores	Component 7 score
0	0
1-2	1
3-4	2
5-6	3

Global PSQI Score: Sum of seven component scores.

Appendix 5: Debrief Form

Debrief form

This study aimed to investigate whether anxiety mediates the influence of sleep quality on social cognition. Differences in social cognition are often associated with Autism Spectrum Disorder.

Participation in this study involved the completion of some questionnaires that may ordinarily be used during preliminary screening for clinical conditions, including anxiety disorder and autism symptom severity. Scores from these tests would not be a sufficient basis for clinical diagnosis, and they do not serve those purposes in this study. However, they may still have had an impact on you. If you wish to seek further information, you might consider the following outlets, or speak with your General Practitioner (doctor).

Birmingham Healthy Minds

Birmingham Healthy Minds is an NHS primary care psychological therapies service that works closely with Birmingham GPs (https://www.bsmhft.nhs.uk/ourservices/birminghamhealthy-minds/self-referral/)). BHM offers advice, information and brief psychological talking therapies for people aged 16 and over, who are often feeling anxious, low in mood or depressed.

Birmingham Nightline

Birmingham Nightline is a confidential, non-judgemental and non-directive listening and information service run by students for students. You can contact Nightline from 8pm-8am every night of term for email, phone and instant messaging services, and from 6pm-12am every night of term for face-to-face contact at the Chaplaincy. Emails are also checked regularly throughout vacation periods. Contact details can be found on the back of student ID cards for students at both University of Birmingham and Aston University, or on online portals (my.bham)

Mind

Mind offers confidential help on a range of mental health problems by providing high quality information, including on anxiety and sleep deprivation. Helpline: 0300 123 3393

National Autistic Society

The Autism helpline provides impartial, confidential information and advice concerning Autism. Helpline: 0808 800 4104

If you wish to withdraw your data, or have any questions concerning this research, please contact the researchers via email: Komal: , Lottie:

or the Principal Investigator, Dr. Andrew Surtees:

Appendix 6: Supplementary Figures

Figure 6.1

Baujat diagnostic plot of sources of heterogeneity for unspecified anxiety disorder



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.
Baujat diagnostic plot of sources of heterogeneity for specific phobia



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.

Baujat diagnostic plot of sources of heterogeneity for social anxiety



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.

Baujat diagnostic plot of sources of heterogeneity for agoraphobia



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.

Baujat diagnostic plot of sources of heterogeneity for panic disorder



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.

Baujat diagnostic plot of sources of heterogeneity for generalised anxiety disorder



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.



Baujat diagnostic plot of sources of heterogeneity for unspecified depressive disorder

Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.

Baujat diagnostic plot of sources of heterogeneity for dysthymia



Note. The vertical axis reports the influence of the study on the overall effect and the horizontal axis reports the discrepancy of the study with the rest of the literature.



Empirical Paper: Scatterplot of the Relationship Between SRS-2 and PHQ-9 Total Scores

Figure 6.11

Empirical Paper: Scatterplot of the Relationship Between SRS-2 and PSQI Total Scores



Empirical Paper: Normal P-P Plot of Regression Standardised Residual with PHQ-9 Square Root as the Dependent Variable



Empirical Paper: Scatterplot Confirming Homoscedasticity Between the Dependent and Predictor Variables



Appendix 7: Supplementary Tables

Tables 7.1 - 7.10

The following tables summarise the subgroup analyses conducted on the anxiety disorder prevalence rates for the risk of bias ratings of "low risk" and "any risk"

Table 7.1

Unspecified anxiety disorder

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	p
Selection bias	.3982	.3310; .4653	27	.3926	.2845; .5008	18	.01	.9323
Performance bias	.4085	.3428; .4742	32	.3656	.2394; .4917	13	.35	.5541
Detection bias	.3159	.2357; .3961	17	.4427	.3666; .5187	28	5.05	.0246
Statistical bias	.3984	.3345; .4623	38	.3834	.2241; .5426	7	.03	.8637
Reporting bias	.3904	.3268; 4540	40	.4484	.3053; .5915	5	.53	.4677
Generalisability bias	.4867	.3052; .6682	6	.3822	.3205; .4438	39	1.14	.2850

Table 7.2

Specific phobia

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	p
Selection bias	.2337	.1533; .3140	11	.2376	.0178; .4574	3	0	.9739
Performance bias	.2311	.1537; .3086	13	.3000	.0160; .5840	1	.21	.6467
Detection bias	.1863	.0907; .2819	8	.2926	.1859; .3994	6	2.11	.1460
Statistical bias	.2357	.1506; .3207	12	.2261	.0624; .3897	2	.01	.9188
Reporting bias	.2336	.1589; .3082	14			0		
Generalisability bias	.3627	.2762; .4492	3	.2087	.1259; .2915	11	6.35	.0117

Table 7.3Generalised Anxiety Disorder

	Low	Risk		Апу	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.1529	.1027; .2030	21	.1261	.0531; .1992	8	.35	.5544
Performance bias	.1386	.0925; .1848	23	.1746	.0798; .2693	6	.45	.5040
Detection bias	.0918	.0361; .1475	10	.1716	.1197; .2236	19	4.21	.0401
Statistical bias	.1437	.0959; .1916	21	.1498	.0650; .2346	8	.02	.9024
Reporting bias	.1076	.0721; .1431	26	.2797	.2062; .3532	6	17.05	<.0001
Generalisability bias	.01679	.1060; .2298	4	.1417	.0958; .1876	25	.44	.5049

Table 7.4Separation Anxiety Disorder

	Low	⁷ Risk		Any Risk				
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.1099	.0522; .1675	16	.1021	.0341; .1701	6	.03	.8641
Performance bias	.0692	.0304; .1080	16	.2219	.1532; .2905	6	14.4	.0001
Detection bias	.0175	.0056; .0294	8	.1560	.0936; .2185	14	18.24	< .0001
Statistical bias	.0920	.0442; .1397	15	.1486	.0442; .2531	7	.94	.3336
Reporting bias	.0937	.0490; .1385	19	.2099	.0430; .3678	3	1.74	.1878
Generalisability bias	.1241	.0545; .1936	4	.1077	.0536; .1619	18	.13	.7159

Table 7.5

Social Anxiety Disorder

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.1537	.1040; .2033	20	.1322	.0416; .2228	5	.17	.6839
Performance bias	.1305	.0885; .1725	18	.2086	.0907; .3265	7	1.49	.2215
Detection bias	.0712	.0299; .1125	7	.1750	.1216; .2285	18	9.06	.0026
Statistical bias	.1393	.0938; .1848	17	.1737	.0699; .2774	8	.35	.5526
Reporting bias	.1511	.1022; .2000	22	.1301	.1301; .1648	3	.47	.4911
Generalisability bias	.1175	.0622; .1728	4	.1539	.1037; .2041	21	.91	.3398

Table 7.6

Panic Disorder

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.0109	.0029; .0190	8	.1086	- .0113; .2284	4	2.54	.1112
Performance bias	.0127	.0047; .0206	10	.2112	.0412; .3813	2	5.23	.0222
Detection bias	.0121	- .0027; .0269	3	.0542	.0108; .0977	9	3.23	.0721
Statistical bias	.0139	.0048; .0230	9	.1390	.0254; .3034	3	2.22	.1363
Reporting bias	.0318	.0112; .0525	12			0		
Generalisability bias	.0127	.0120; .0373	1	.0401	.0119; .0684	11	2.06	.1509

Table 7.7 Agoraphobia

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.0695	.0283; .1108	4	.0330	0037; .0697	1	1.68	.1943
Performance bias	.0594	.0271; .0917	5			0		
Detection bias	.0300	.0040; .0561	2	.0838	.0517; .1159	3	6.51	.0107
Statistical bias	.0594	.0271; .0917	5			0		
Reporting bias	.0594	.0271; .0917	5			0		
Generalisability bias	.1392	.0629; .2156	1	.0458	.0226; .0690	4	5.27	.0218

Unspecified Depressive Disorder

	Low	Risk		Any	Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	p
Selection bias	.2092	.1425; .2759	21	.2897	.1705; .4088	11	1.33	.2482
Performance bias	.2148	.1533; .2764	27	.3623	.1868; .5377	5	2.42	.1201
Detection bias	.2732	.1941; .3522	14	.2117	.1244; .2990	18	1.05	.3064
Statistical bias	.2343	.1640; .3046	25	.2492	.1268; .3716	7	.04	.8361
Reporting bias	.2500	.1844; .3155	28	.1476	.0147; .2806	4	1.83	.1760
Generalisability bias	.1899	.1034; .2764	1	.2392	.1769; .3014	31	.82	.3647

	Lo	w Risk		An	y Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	<i>X</i> ²	р
Selection bias	.0965	.0400; .1529	11	.0506	.0199; .0813	4	1.96	.1618
Performance bias	.0832	.0436; .1228	15			0		
Detection bias	.0816	.0285; .1347	5	.0868	.0275; .1462	10	.02	.8973
Statistical bias	.1054	.0500; .1607	11	.0339	.0118; .0561	4	5.51	.0189
Reporting bias	.0933	.0461; .1405	13	.0389	.0080; .0698	2	3.58	.0586
Generalisability bias	.1333	.0473; .2193	1	.0806	.0389; .1223	14	1.17	.2796

Dysthymia

	Lov	v Risk		Any	y Risk			
	EFFECT	95% CI	k	EFFECT	95% CI	k	X^2	р
Selection bias	.0306	.0056; .0556	7	.0755	.0165; .1345	3	1.89	.1697
Performance bias	.0467	.0190; .0745	10			0		
Detection bias	.0110	0042; .0261	3	.0580	.0244; .0935	7	5.68	.0171
Statistical bias	.0312	.0060; .0564	7	.0741	.0129; .1353	3	1.62	.2034
Reporting bias	.0269	.0066; .0471	8	.1052	.0800; .1304	2	22.57	<.0001
Generalisability bias			0	.0467	.0190; .0745	10		

Table 7.11

Results relating to the effect of recruitment location on prevalence rates for depressive disorders

	X ²	Р
Unspecified depressive disorder	.03	.8551
Major depressive disorder	.18	.6747
Dysthymia	4.19	.0406

Anxiety Type	Fem	ales	Males			
	EFFECT	95% CI	EFFECT	95% CI	Z	р
Unspecified Anxiety Disorder	.6602	.0290; 1.2914	.6570	.0333; 1.2807	.0032	.4082
Generalised anxiety Disorder	.0415	4947; .4117	0392	.4872; - .5341	.0023	.3974
Specific Phobia	.4987	2536; 1.251	.4958	2479; 1.239	.0029	.5104
Social Anxiety Disorder	.5223	0990; 1.1435	.5178	0962; 1.132	- .0044	.2301
Separation Anxiety Disorder	.2868	1185; .6922	.2844	-1162; .6851	.0024	.3257
Panic Disorder	.1097	0873; .3067	.1086	0862; .3035	- .0011	.3285
Agoraphobia	-2.394	-5.06; .3402	-2.33	-5.0009; .3370	.0275	.0793
Depression Type	Fem	ales		Males		
	EFFECT	95% CI	EFFECT	95% CI	z	р
Unspecified depressive disorder	.6223	.0887; 1.156	.6178	.0903; 1.145	- .0045	.1557
Major depressive disorder	.4093	.0037; .8149	.4055	.0046; .8064	- .0038	.1127
Dysthymia	.3099	2154; .8351	.3068	02124; .8261	- .0030	.3256

The Impact of the Proportion of Male Participants on the Prevalence of Anxiety and Depressive Disorders

	X ²	Р
Unspecified Anxiety	1.03	.3112
Generalised Anxiety Disorder	.87	.3499
Specific Phobia	.03	.8715
Social Anxiety Disorder	.17	.6807
Agoraphobia*	-	-
Separation Anxiety Disorder	1.59	.2074
Panic Disorder	4.75	.0293

Results relating to the effect of including or excluding people with intellectual disabilities in studies reporting on anxiety

Note: All studies reporting prevalence of agoraphobia included people with intellectual disabilities.

Table 7.14

Results relating to the effect of including or excluding people with intellectual disabilities in studies reporting on depression

	X ²	Р
Unspecified Depressive Disorder	.67	.4139
Major Depressive Disorder	3.02	.0821
Dysthymia	.44	.5049