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Abstract
The UK prevalence of parent-reported autism spectrum disorder (ASD) and attention deficit/hyperactivity disorder (ADHD) were estimated from the Millennium Cohort Study. Case definition was if a doctor or health care professional had ever told parents that their child had ASD and/or ADHD. Data were collected in 2008/2009 for 14,043 children. 1.7 % of children were reported as having ASD (95 % CI 1.4–2.0) at mean age 7.2 years (SD = 0.2; range = 6.3–8.2). 1.4 % reportedly had ADHD (95 % CI 1.2–1.7), and 0.3 % had both ASD and ADHD (95 % CI 0.2–0.5). After adjusting for socio-economic disadvantage, only male sex ($p < 0.001$ for both conditions) and cognitive ability, $p = 0.004$ (ASD); $p = 0.01$ (ADHD) remained strongly associated. The observed prevalence of parent-reported ASD is high compared to earlier UK and US estimates. Parent-reported ADHD is low compared to US estimates using the same measure.

Keywords (separated by '-') Attention deficit hyperactivity disorder - Autism - Prevalence - Co-morbidity - Pervasive developmental disorder - Autism spectrum disorder

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2 **Prevalence of Parent-Reported ASD and ADHD in the UK:**
3 **Findings from the Millennium Cohort Study**

4 **Ginny Russell · Lauren R. Rodgers ·**
5 **Obioha C. Ukoumunne · Tamsin Ford**

6
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9 spectrum disorder (ASD) and attention deficit/hyperactivity
10 disorder (ADHD) were estimated from the Millennium
11 Cohort Study. Case definition was if a doctor or health care
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13 and/or ADHD. Data were collected in 2008/2009 for
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15 ASD (95 % CI 1.4–2.0) at mean age 7.2 years (SD = 0.2;
16 range = 6.3–8.2). 1.4 % reportedly had ADHD (95 % CI
17 1.2–1.7), and 0.3 % had both ASD and ADHD (95 % CI
18 0.2–0.5). After adjusting for socio-economic disadvantage,
19 only male sex ($p < 0.001$ for both conditions) and cogni-
20 tive ability, $p = 0.004$ (ASD); $p = 0.01$ (ADHD) remained
21 strongly associated. The observed prevalence of parent-
22 reported ASD is high compared to earlier UK and US
23 estimates. Parent-reported ADHD is low compared to US
24 estimates using the same measure.

25
26 **Keywords** Attention deficit hyperactivity disorder ·
27 Autism · Prevalence · Co-morbidity · Pervasive
28 developmental disorder · Autism spectrum disorder

Introduction

The last 20 years have seen steady increases in the esti-
mated prevalence of both autism spectrum disorder (ASD)
and attention deficit hyperactivity disorder (ADHD) in
childhood (Boyle et al. 2011). Despite exclusion clauses in
diagnostic criteria for ASD relating to ADHD (World
Health Organization 1993; American Psychiatric Associa-
tion 2000) considerable symptom overlap between these
conditions has been reported (Simonoff et al. 2008; Re-
iersen and Todd 2008).

Estimates of the prevalence of both conditions world-
wide vary widely (Newschaffer et al. 2007; Brown et al.
2001; Polanczyk et al. 2007). Knowledge of the number of
children identified with these disorders is crucial for
planning and commissioning services and studying the
process of identification in clinical practice. Nevertheless,
there is no UK public health record that gives a definitive
number of children with a diagnosis of either condition.
Researchers have therefore estimated prevalence in the
community in a variety of ways.

Screening instruments combined with assessments and
parent-reported clinical diagnosis resulted in an estimated
ASD prevalence of 1.57 % for children aged 5–9 in 2004 in
the UK in a sample from primary schools (Baron-Cohen
et al. 2009). An earlier UK cohort study screened the ‘at-risk
of ASD’ population with parent and teacher assessment
instruments, producing a estimate of 1.16 % of children
having an ASD (Baird et al. 2006). A population-based
sample estimated the UK prevalence of both ASD (0.9 % in
2004) and ADHD (approximately 1.5 %) using both semi-
structured interviews and an instrument designed to identify
DSM diagnoses in 5–15 years olds (Green et al. 2003).
Polanczyk et al. (2007) systematic review of the worldwide
prevalence of ADHD found recorded rates ranging from 1 to

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63 18 %. This wide variation is likely to be in part due to a lack
64 of standardisation in case ascertainment.

65 Clinical practice varies widely between cultures and
66 even within countries (e.g. Reid et al. 2002). Several
67 studies have addressed the issue of cross-cultural differences
68 in labelling, for example, in South Korea, some
69 researchers have argued under-diagnosis of ASD is due to
70 strong stigma attached to the disorder (Grinker et al. 2012).
71 Others have argued that cultural, social and developmental
72 context elicit differences in impact and expression of
73 symptoms and behaviours (Caron et al. 2012; Singh 2011;
74 Norbury and Sparks 2013). Objective measures to diagnose
75 that reach across cultures are therefore hard to establish.
76 Taylor and Sandberg (1984) questioned why measured
77 rates of ADHD in the UK were lower than the US, sparking
78 further debate as to whether this really was the case
79 (Faraone et al. 2003). Malacrida (2004) discusses the
80 reluctance of European clinicians and parents to utilise the
81 ADHD label and administer pharmaceutical treatment
82 (usually methylphenidate) compared to US counterparts.
83 Polanczyk et al. (2007) however, found no differences
84 between European and US rates of ADHD in their sys-
85 tematic review. In the US, 6.3 % of all children aged 5–9
86 were reported by parents to have an ADHD diagnosis in
87 2008–2010 (National Center for Health Statistics 2012).

88 Both diagnoses have been associated with socio-econ-
89 omic factors. In the US, studies based on the National
90 Health Interview Survey data, and others, show that ASD
91 prevalence is lower among groups of lower socio-economic
92 status (Fountain et al. 2011; Kogan et al. 2009). By contrast,
93 higher rates of ADHD have been observed for socially dis-
94 advantaged groups (Pastor and Rueben 2008; Akinbami
95 et al. 2011; Bøe et al. 2012; Hjern et al. 2010). A range of
96 other factors, including child's sex, maternal depression
97 older motherhood, intellectual disability, and ethnicity
98 also been associated with both conditions (Akinbami et al.
99 2011; Banerjee et al. 2007; Kogan et al. 2009; Lesesne et al.
100 2003; Pastor and Rueben 2008; Russell et al. 2011; Sandin
101 et al. 2012; Scahill et al. 1999). Birth complications and
102 prenatal risk factors have been linked to both conditions
103 (Gardener et al. 2009; Linnert et al. 2003). It is important to
104 establish whether some groups of children are more likely to
105 be identified, as differing contexts may lead to children
106 missing out on health services, or to over-identification.

107 The aims of our study were to estimate the prevalence of
108 parent-reported ASD and ADHD in the UK and examine
109 association between recognition of these disorders and
110 socio-demographic, child-based and contextual factors. The
111 prevalence of both conditions was estimated using data from
112 the Millennium Cohort Study (MCS), a large UK popula-
113 tion-based birth cohort study. ~~ASD and ADHD status were
114 measured over a 13 months period between 2008 and 2009
115 when the children were around 7 years old from parent-~~

report of whether either condition was identified by a doctor
or other health professional. The same measure was used by
the US National Health Interview Survey Sample Child
questionnaire to identify developmental disabilities in the
United States (for example, Kogan et al. 2009; Boyle et al.
2011; Pastor and Rueben 2008). Parents reported on iden-
tified ASD and ADHD in their children over a 13 months
period between 2008 and 2009.

Methods

The MCS is a UK-representative birth cohort study using a
disproportionate stratified cluster sampling design. Sam-
pling of electoral wards (the clusters) was stratified by UK
country (England, Scotland, Wales and Northern Ireland),
and further stratified by ethnic group composition (whether
at least 30 % of the population fell into the categories
“Black” or “Asian”) and level of Child Poverty in Eng-
land, and by level of social disadvantage in Scotland,
Wales and Northern Ireland (Hansen and Joshi 2010).
There was further implicit stratification by region (within
country), and by electoral ward size. Details of the sam-
pling design are documented in detail elsewhere (Plewis
2007). Children born between 1st September 2000 and 11th
January 2002 and listed on the Child Benefit Records
(which had near universal take up) were eligible for the
study. Data were first collected when children were
9 months old (1st wave), including hospital birth records
and socio-demographic and family circumstances. Subse-
quently, further data were recorded concerning the chil-
dren's health and development when the children were
3 years old (2nd wave), 5 years old (3rd wave) and 7 years
old (4th wave). Within the total MCS cohort of 15, 918
% responded to the questions about ASD
Consistent with other studies using these
data (Totsika et al. 2011), families with twins or triplets
where all the siblings participated were excluded (252
twins, 11 triplets) as outcomes would be expected to be
correlated within families.

Outcome Measures

The outcome measure of ASD or ADHD status was based
on responses to the MCS question duplicated from the US
National Health Interview Survey questionnaire reported in
previous studies (Akinbami et al. 2011; Boyle et al. 2011;
Kogan et al. 2009; Pastor and Rueben 2008). The main
carer was asked if a child had ADHD or ASD identified by
doctor or health professional. In 96.7 % of cases the carer
was the child's mother, who in over 99 % of cases was
resident at home with the child all of the time. This mea-
sure was used in a face to face interview in each child's

add 's' (make plural)

164 home by trained interviewers, with the wording of the
165 question read out verbatim:

- 166 • Has a doctor or health professional ever told you that
167 (sample child) attention deficit hyperactivity dis-
168 order (ADHD)? Has a doctor or health professional ever
169 told you that (sample child) had Autism, Asperger's
170 syndrome or autistic spectrum disorder?

171 Data on ASD and ADHD status were collected at both
172 waves 3 and 4. Wave 4 ASD/ADHD status data, (mean age
173 of child 7 years old), were analysed in this study. A posi-
174 tive or negative response to the above question was taken
175 as the case definition for diagnosis of ASD or ADHD. Data
176 were coded as missing where a response of 'don't know' or
177 'not applicable' was recorded.

178 Potential Predictors

179 Several variables that had previously been found to be
180 associated with ASD or ADHD were available. Child-
181 based measures included sex, age and cognitive ability of
182 children, which was recorded at age three using a series of
183 tests administered by trained researchers during individual
184 visits to all children's homes. The cognitive test used was
185 the Bracken School Readiness Assessment (Bracken 1999).
186 The test comprised six subtests that assess a child's ability
187 to identify colours, letters, numbers, shapes and to describe
188 and compare objects (e.g., by size). These assessments
189 were individually administered in computer-assisted inter-
190 views. The test has been used as an intellectual screening
191 instrument (Laughlin 1995). Other child-based factors were
192 derived from linked UK Birth Registration and Maternity
193 Hospital Episode Data, including birth weight, gestation
194 length (i.e. before 280 days if premature birth), type of
195 delivery, and length of labour. Mothers responding to the
196 9 months interview were asked to give written consent to
197 birth registration and hospital maternity records being
198 added to the survey. This interview also recorded tobacco
199 use during pregnancy.

200 Family-based background factors including the age of
201 the mother at childbirth, the ethnicity of the family into
202 which the child had been born and family size were
203 reported at waves 1–4. A measure of maternal mental
204 health was taken from mothers' reports of whether they had
205 ever been diagnosed with depression or anxiety by wave 4.
206 Indicators of family socio-economic status (SES) were
207 family income (adjusted for the number of children per
208 family), housing tenure, number of full time carers at
209 child's home (single parent or couple), and mother's
210 highest educational qualification. Families were classed as
211 living in poverty if their income was equal to or less than
212 60 % of the median household income for the UK popu-
213 lation at wave 4.

Statistical Analysis

214
215 Demographic characteristics for the study sample overall,
216 by ASD status and by ADHD status, were reported.
217 Logistic regression was used to examine the association
218 between ASD/ADHD status and the following potential
219 predictors: child's sex, cognitive ability at age 3, birth
220 weight, and exact age of child in months, pre and perinatal
221 factors (child characteristics); maternal education, maternal
222 age at childbirth, ethnicity, equivalised family income,
223 family size, family structure, housing tenure, poverty level
224 and whether mothers had been diagnosed with depression
225 (family characteristics).

226 In the logistic regression models, continuous predictors
227 were rescaled (divided by 2 standard deviations), so that
228 odds ratios (OR) indicate the relative increase in odds of
229 being identified with the condition, corresponding to a 2
230 standard deviation increase in the predictor. This trans-
231 formation enables comparison of strength of association
232 across continuous and binary predictors (Gelman 2008).
233 Unadjusted logistic regression models were fitted in which
234 just one predictor at a time was included. Multivariable
235 (adjusted) logistic regression models were then fitted in
236 which predictors significant at the 10 % level in the
237 unadjusted analyses were included as covariates.

238 Estimates of the prevalence of ASD and ADHD and the
239 logistic regression analyses were weighted to take account
240 of the disproportionate stratified sample of electoral wards
241 and attrition/non-response by the 4th wave when the study
242 outcomes were measured, making the sample representa-
243 tive of the UK population (Plewis 2007). Standard errors in
244 the logistic regression were calculated using first-order
245 Taylor linearisation to take account of the correlation of
246 responses between children within electoral ward clusters.
247 All analyses were performed using Stata 12 software. The
248 complete case analyses reported here include only partic-
249 ipants with data for both the outcome and all predictors in
250 the model. The numbers of observations analysed exceeded
251 13,000 for all but two predictors (from a possible 13,586
252 responses to the question about ASD and 13,574 respond-
253 ing to ADHD status); the exceptions were maternal
254 depression (n = 8,443) and ethnicity (n = 11,883).

Results

255
256 For 96.7 % of those participating at wave 4, the main
257 respondent on the outcome measure of ASD or ADHD was
258 the child's mother. At the birth of the child, mothers had a
259 mean age of 28 years (range 13–48 years). The mean age
260 of children when outcome measures were taken was
261 7.2 years (SD = 0.2; range 6.3–8.2). Table 1 illustrates the
262 demographic profile of the sample.

Table 1 Descriptive statistics: child- and family-based background factors for children by ASD and ADHD status

Characteristic	ASD (N = 86-209)	No ASD (N = 8,363-13,377)	ADHD (N = 59-173)	No ADHD (N = 8,384-13,401)	Comorbid ASD and ADHD (N = 8-44)	No diagnosis ASD or ADHD (N = 8,306-13,231)
<i>Child characteristics</i>						
Male (%)	83.9	50.2	82.2	50.3	93.0	49.9
Birth weight in kg, mean (SD)	3.4 (0.6)	3.4 (0.6)	3.3 (0.6)	3.4 (0.6)	3.4 (0.5)	3.4 (0.6)
Age in years at wave 4, mean (SD)	7.2 (0.3)	7.2 (0.2)	7.2 (0.2)	7.2 (0.2)	7.2 (0.3)	7.2 (0.2)
Cognitive ability—wave 2, mean (SD)	43.8 (34.6)	58.2 (30.3)	44.1 (30.4)	58.3 (30.4)	40.2 (32.9)	58.4 (30.3)
Number of cigarettes smoked in pregnancy, mean (SD)	1.3 (3.3)	1.0 (3.4)	1.9 (4.0)	1.0 (3.4)	1.4 (3.0)	1.0 (3.4)
Length of labour in hours, mean (SD)	8.2 (8.8)	9.2 (11.1)	10.2 (14.0)	9.1 (11.0)	7.7 (8.4)	9.1 (11.0)
Days gestation, mean (SD) 280 = due date	274.9 (17.1)	277.6 (13.4)	274.5 (17.8)	277.6 (13.4)	276.8 (14.0)	277.6 (13.3)
Delivery type (%)						
No problems	8.4	69.0	68.5	69.0	80.0	69.0
Forceps/breach/vacuum	8.7	9.7	7.3	9.7	2.5	9.7
Caesarean	23	21.3	24.2	21.3	17.5	21.3
<i>Family characteristics</i>						
White British (%)	92.4	86.5	90.9	86.5	94.6	86.5
Family size—wave 4						
Only child (%)	17.7	12.9	16.8	12.9	15.9	12.8
1 sibling (%)	43.5	45.2	39.9	45.3	45.5	45.3
2 siblings (%)	25.4	27.1	26.6	27	22.7	27.1
More than 2 siblings (%)	13.4	14.8	16.8	14.8	15.9	14.8
Maternal age at childbirth, mean (SD)	27.9 (5.9)	28.7 (5.8)	26.2 (5.8)	28.8 (5.8)	26.2 (5.4)	28.8 (5.8)
Maternal education—wave 1						
No qualifications (%)	17.1	16.6	25.4	16.5	20.9	16.5
School level (%)	62.3	56.3	60.4	56.3	62.8	56.3
Degree or higher (%)	20.6	27.1	14.2	27.2	16.3	27.2
Mother depression/anxiety—wave 4 (%)	10.5	6.6	8.5	6.6	0	6.6
Family income in £—wave 4, mean (SD)	351.2 (209.2)	382.2 (228.0)	312.9 (179.9)	382.8 (228.1)	324.2 (172.2)	383.1 (228.2)
Below poverty line—wave 4 (%)	35.4	30	42.8	29.9	40.9	29.8
Single parent family—wave 4 (%)	34.9	20.9	37	20.9	40.9	20.7
Housing tenure—wave 4						
Social housing (%)	31.9	23.2	40.9	23.1	36.4	23
Rent private (%)	13.2	8.8	15.2	8.8	15.9	8.8
Home owner (%)	54.9	68	43.9	68.1	47.7	68.3

N range is given as not all children have recorded data for every characteristic. Where data were missing they were excluded from the analysis

263 After excluding twins and triplets, at wave 4, there were
 264 13,586 responses concerning ASD status and ADHD status
 265 of children. Of these children, 209 were reported to have
 266 ASD and 173 to have ADHD. Forty-four children report-
 267 edly had both ASD and ADHD. The prevalence for ASD
 268 was 1.7 % (95 % CI 1.4–2.0) overall; 2.5 % for boys and
 269 0.5 % for girls, giving a male to female ratio of approxi-
 270 mately 5–1 for ASD. Prevalence of ADHD was 1.4 %
 271 (95 % CI 1.2–1.7) overall; 2.2 % of boys and 0.5 % of
 272 girls, giving a male to female ratio of approximately 4–1.
 273 The proportion of children with both conditions was 0.3
 274 (95 % CI 0.2–0.5). 19.9 % of the children with ASD also
 275 had ADHD (95 % CI 13.2–26.6) while 24.1 % of the
 276 children with ADHD had ASD (95 % CI 18.9–32.0).

277 At wave 3 children were approximately 5 years of age
 278 (range 4.9–5.5 years). Not surprisingly, more children had
 279 been identified with both conditions by age seven. The
 280 prevalence of ASD for 5 years olds was 0.9 and 0.9 % for
 281 ADHD. Drop-out from wave 3 to wave 4 was slightly
 282 greater for those with ASD and/or ADHD than for other
 283 children. Nineteen percent of those with ADHD at wave 3
 284 were missing at wave 4 (26/134), compared to 13 %
 285 (1,932/14815) missing from the rest of the sample, while
 286 20 % (26/131) of those with ASD at wave 3 were missing
 287 compared to 13 % (1,933/14,826) of non-respondents
 288 without the diagnosis.

289 Table 2 reports the odds ratios (OR) of having ASD for
 290 the unadjusted and adjusted analyses. For factors significant

Table 2 Logistic regression of ASD status on background factors

Predictors	Unadjusted ^a		Adjusted ^b	
	OR (95 % CI)	<i>p</i>	OR (95 % CI)	<i>p</i>
Child characteristics				
Male	5.02 (3.19–7.90)	<0.001	4.94 (2.58–9.44)	<0.001
Birth weight	0.94 (0.60–1.47)	0.78		
Age at wave 4	0.91 (0.64–1.29)	0.58		
Cognitive ability—wave 2	0.41 (0.26–0.63)	<0.001	0.49 (0.30–0.79)	0.003
Number of cigarettes	1.18 (0.98–1.41)	0.08	0.99 (0.78–1.27)	0.95
Length of labour	0.88 (0.64–1.21)	0.42		
Days gestation	0.64 (0.45–0.92)	0.01	0.69 (0.44–1.09)	0.12
Delivery %		0.91		
No problems at birth	Reference			
Forceps/breach/vacuum delivery	0.91 (0.43–1.97)			
Caesarean	1.07 (0.72–1.59)			
Family characteristics				
White British	0.82 (0.37–1.80)	0.62		
Family size—wave 4		0.46		
Only child	Reference			
1 sibling	0.70 (0.41–1.19)			
2 siblings	0.70 (0.41–1.20)			
More than 2 siblings	0.62 (0.34–1.15)			
Maternal age at childbirth	0.80 (0.57–1.23)	0.20		
Maternal education—wave 1		0.12		
No qualifications	Reference			
School level	1.12 (0.72–1.72)			
Degree or higher	0.70 (0.39–1.24)			
Maternal depression/anxiety diagnosis—wave 4	1.85 (0.86–3.94)	0.11		
Family income—wave 4	0.68 (0.48–0.95)	0.02	1.42 (0.89–2.28)	0.14
Below poverty line—wave 4	1.27 (0.90–1.79)	0.17		
Single parent family—wave 4	1.87 (1.30–2.68)	0.001	1.11 (0.62–2.01)	0.72
Housing tenure—wave 4		0.001		0.03
Social housing (%)	Reference		Reference	
Rent private (%)	0.93 (0.55–1.60)		0.82 (0.38–1.75)	
Home owner (%)	0.51 (0.35–0.74)		0.47 (0.27–0.81)	

Odds ratios (ORs) shown for a 2 standard deviation increase in continuous predictors

^a Sample size ranges from 8,449 to 13,586 in unadjusted analyses

^b Sample size is 10, 230 in adjusted analysis

291 at the 10 % level in the unadjusted analysis, the right hand
 292 column of Table 2 shows adjusted odds ratios which take
 293 interdependencies between predictors into account. In the
 294 unadjusted analyses there was strong evidence that boys and
 295 those with lower scores on the school readiness assessment
 296 (lower cognitive ability) at 3 years were more likely to have
 297 an ASD. Increasing tobacco use in pregnancy and a more
 298 premature birth were also associated with ASD. Birth
 299 weight, length of labour, method of delivery and the child's
 300 exact age when the wave 4 data were recorded did not appear
 301 to be associated with the odds of having ASD.

302 Several measures of socio-economic disadvantage in the
 303 children's family background were associated with ASD.
 304 Children from families with lower income were more

likely to have ASD. Children living in social housing and
 those from single parent families were also more likely to
 have ASD (Table 2). There was little evidence of associ-
 ation between the other family-based factors that were
 examined and ASD.

In the adjusted model, lower cognitive ability and male
 sex were the factors most strongly associated with ASD,
 together with one measure of socio-economic status: social
 housing. Families living in social housing were still around
 twice as likely to have a child with ASD compared to
 families that own their homes.

Table 3 reports the logistic regression for the children
 with ADHD. In the unadjusted analysis, the same child-
 based factors that were significantly associated with ASD

Table 3 Logistic regression of ADHD status on background factors

Predictors ^a	Unadjusted ^b		Adjusted ^c	
	OR (95 % CI)	<i>p</i>	OR (95 % CI)	<i>p</i>
Child characteristics				
Male	4.26 (2.77–6.56)	<0.001	4.56 (2.55–8.14)	<0.001
Birth weight	0.84 (0.57–1.25)	0.39		
Age at wave 4	1.16 (0.86–1.57)	0.32		
Cognitive ability—wave 2	0.40 (0.26–0.62)	<0.001	0.54 (0.34–0.88)	0.01
Number of cigarettes smoked prenatal	1.36 (1.19–1.56)	<0.001	1.10 (0.90–1.36)	0.35
Length of labour	1.36 (1.01–1.83)	0.04	1.35 (1.00–1.81)	0.05
Days gestation	0.65 (0.48–0.88)	0.006	0.67 (0.48–0.93)	0.02
Delivery %		0.38		
No problems at birth	Reference			
Forceps/breach/vacuum	0.69 (0.37–1.29)			
Caesarean	1.14 (0.78–1.66)			
Family characteristics				
White British	1.46 (0.74–2.89)	0.28		
Family size—wave 4		0.13		
Only child	Reference			
1 sibling	0.56 (0.34–0.91)			
2 siblings	0.68 (0.40–1.14)			
More than 2 siblings	0.78 (0.45–1.34)			
Maternal age at childbirth	0.46 (0.33–0.65)	<0.001	0.63 (0.38–1.04)	0.07
Maternal education—wave 1		<0.001		0.91
No qualifications	Reference		Reference	
School level	0.62 (0.41–0.94)		0.97 (0.50–1.90)	
Degree or higher	0.32 (0.19–0.56)		0.86 (0.37–2.01)	
Maternal depression/anxiety—wave 4	1.03 (0.39–2.72)	0.95		
Family income—wave 4	0.52 (0.35–0.77)	0.001	1.21 (0.72–2.04)	0.47
Below poverty line—wave 4 ^a	1.64 (1.12–2.39)	0.01		
Single parent family—wave 4	2.06 (1.41–3.00)	<0.001	1.29 (0.72–2.29)	0.39
Housing tenure—wave 4		<0.001		0.42
Social housing (%)	Reference		Reference	
Rent private (%)	0.81 (0.46–1.42)		1.15 (0.50–2.63)	
Home owner (%)	0.37 (0.26–0.54)		0.73 (0.42–1.28)	

Odds ratios (ORs) shown for a 2 standard deviation increase in continuous predictors

^a Poverty was not included in the adjusted model as it is derived from the family income variable which was also significant at the 10 % level in the unadjusted analysis

^b Sample size ranges from 8,443 to 13,574 in unadjusted analyses

^c Sample size is 9,808 in adjusted analysis



Table 4 Logistic regression of comorbid status on background factors

Predictors	Unadjusted ^a		Adjusted ^b	
	OR (95 % CI)	<i>p</i>	OR (95 % CI)	<i>p</i>
Child characteristics				
Male	18.77 (4.58–76.88)	<0.001	23.54 (3.49–158.60)	0.001
Birth weight	0.93 (0.48–1.77)	0.82		
Age at wave 4	1.09 (0.57–2.10)	0.79		
Cognitive ability—wave 2	0.32 (0.13–0.78)	0.01	0.39 (0.18–0.86)	0.02
Number of cigarettes smoked during pregnancy	1.27 (0.95–1.70)	0.11		
Length of labour	0.78 (0.41–1.50)	0.48		
Days gestation	0.79 (0.40–1.58)	0.51		
Delivery %		0.35		
No problems	Reference			
Forceps/breach/vacuum	0.23 (0.03–1.67)			
Caesarean	0.98 (0.42–2.27)			
Family characteristics				
White British	1.93 (0.46–8.12)	0.37		
Family size—wave 4		0.63		
Only child	Reference			
1 sibling	0.76 (0.28–2.04)			
2 siblings	0.48 (0.15–1.47)			
More than 2 siblings	0.76 (0.25–2.37)			
Maternal age at childbirth	0.61 (0.34–1.09)	0.10		
Maternal education—wave 1		0.39		
No qualifications	Reference			
School level	1.02 (0.43–2.39)			
Degree or higher	0.54 (0.18–1.62)			
Maternal depression/anxiety—wave 4	NA ^c			
Family income—wave 4	0.51 (0.24–1.07)	0.08	1.25 (0.60–2.60)	0.54
Below poverty line—wave 4 ^a	1.86 (0.88–3.94)	0.10		
Single parent family—wave 4	2.88 (1.45–5.70)	0.003	1.83 (0.62–5.36)	0.27
Housing tenure—wave 4		0.006		0.35
Social housing (%)	Reference			
Rent private (%)	1.10 (0.35–3.44)		1.52 (0.39–5.98)	
Home owner (%)	0.43 (0.20–0.93)		0.61 (0.27–1.39)	

Odds ratios (ORs) shown for a 2 standard deviation increase in continuous predictors

^a Sample size ranges from 7,769 to 13,275 in unadjusted analyses

^b Sample size is 10,114 in adjusted analysis

^c NA (not applicable): no mothers of children with both ASD and ADHD reported depression or anxiety

319 were also associated with ADHD; lower cognitive ability
 320 and male sex. In addition, three pre- and perinatal factors
 321 were associated with ADHD: prematurity, smoking during
 322 pregnancy and longer labour. There was little evidence of
 323 associations between ADHD status and the exact age of
 324 child at the fourth MCS wave, or their birth weight. Several
 325 family-based socio-economic measures of disadvantage
 326 were strongly linked to ADHD: lower income, lower
 327 maternal education and poverty. Mothers who were
 328 younger when the study child was born, families living in
 329 social housing and single parent families had greater odds
 330 of having a child with identified ADHD. There was,

331 however, no significant association between ADHD and
 332 ethnicity, maternal depression or family size.

333 In the adjusted analysis sex and cognitive ability were
 334 most strongly associated with ADHD. Boys were still
 335 over four times more likely to have ADHD than girls
 336 (OR = 4.56, 95 % CI 2.55–8.14). Each drop of two stan-
 337 dard deviations in the Bracken school readiness assessment
 338 was associated with an almost two-fold increase in the odds
 339 of having ADHD. In the adjusted analysis, length of ges-
 340 tation: our proxy for prematurity, and longer labour were
 341 still related to the ADHD outcome, but not as strongly as
 342 cognitive ability and sex.



343 Finally, Table 4 reports results for the group of children
344 who were reported as having both ASD and ADHD. In
345 these analyses, only male sex and cognitive ability
346 increased odds of having co-morbid ASD and ADHD after
347 adjustment for interdependencies. Caution is needed
348 interpreting these findings due to low numbers in the
349 comorbid category (Table 1).

350 Discussion

351 The estimated prevalence of ASD of 1.7 % is high com-
352 pared to other UK and US estimates which have ranged
353 from 0.9 to 1.6 % in recent literature (Baird et al. 2006;
354 Baron-Cohen et al. 2009; Kogan et al. 2009; Zaroff and
355 Uhm 2011). The finding suggests an increasing trend in the
356 UK to apply the ASD label which may be due to a com-
357 bination of greater awareness, successive diagnosis of
358 younger children, broadening criteria (Fombonne 2001,
359 2009) and/or lessening of social stigma associated with the
360 label (Gray 2002). One debate surrounding the rising
361 prevalence of developmental disorders concerns whether
362 rises reflect real increases in frequency and severity of
363 symptoms, or whether they are entirely an artefact of
364 changing diagnostic criteria and increased awareness.
365 Some people affected by these conditions, and some
366 researchers, believe that shifts in diagnostic categorisation
367 do not entirely explain rising prevalence. An underlying
368 concern among these people is that environmental influ-
369 ences may be partially to blame (Russell and Kelly 2011).
370 It is beyond the scope of this study to address what the
371 triggers for increasing prevalence may be.

372 In contrast, the estimated prevalence of ADHD in the
373 UK at 1.5 % is very similar to previous estimates for
374 ADHD and hyperkinetic disorder in the UK based on
375 research diagnosis (Ford et al. 2003; Green et al. 2005).
376 Such estimates are low compared to the European ADHD
377 prevalence of 3–5 % given in the meta-analysis of Pola-
378 nczyk et al. (2007). However, the meta-analysis did not
379 include UK estimates. In addition, in the current study, a
380 substantial proportion of children with ADHD may not
381 have been diagnosed by age seven (Kieling et al. 2010);
382 therefore we would expect around half the population that
383 eventually receive an ADHD diagnosis to be undetected in
384 the study age range of 6–8. There have been debates about
385 whether the prevalence of ADHD is lower in the UK than
386 the US (Charach et al. 2011; Faraone et al. 2003; Taylor
387 and Sandberg 1984). Our findings suggest the ADHD
388 diagnosis is not as often used by UK doctors and/or health
389 professionals as it is in the USA (Boyle et al. 2011; Pastor
390 and Rueben 2008; Akinbami et al. 2011); whereas the
391 autism spectrum as a diagnosis is on the ascent in the UK.
392 The nearest comparator in the US for ADHD is in

5–9 year-olds from 2008 to 2010 using the same parent-
report measure. This gives a prevalence estimate for the
USA of 6.3 % with ADHD (National Center of Health
Statistics 2012, Table 46). The current study uses the same
parent-report measure in 6–8 years-olds in 2008–2009 and
derives a UK estimate of 1.4 % for ADHD. The compar-
atively sparse use of ADHD label in the UK may be due to
lower numbers of children with symptoms in the UK, or
more likely, apprehension regarding ADHD diagnosis and/
or impact of diagnosis on children and their families, or
persistent concerns regarding its treatment with stimulant
drugs (Malacrida 2004). The current diagnostic classifica-
tions suggest the diagnosis of ASD rules out a diagnosis of
ADHD, so that children with hyperactive behaviour in
combination with social difficulties may be more likely to
be diagnosed as having ASD in the UK and ADHD in the
USA (APA 2000; WHO 1993).

However, our findings suggest that ASD and ADHD
labels are used together in a small but noteworthy pro-
portion of the clinical child population, despite the exclu-
sionary criteria of diagnostic criteria (APA 2000; WHO
1993). In doing so, clinical practice is consistent with other
studies that show ASD and ADHD often co-exist (Simo-
noff et al. 2008; Reiersen and Todd 2008). ~~Indeed, recent
debates have addressed whether the two conditions should
be considered as different manifestations of one over-
arching disorder (van der Meer et al. 2012; Hattori et al.
2006). These and other studies lend weight to proposed
revisions to DSM-5 and ICD-11 that will see exclusivity
criteria between the conditions removed.~~

The high estimates for ASD may reflect measurement
error. Whether a child 'had ever been said to have an ASD
by doctor or health professional' may have been over-
inclusive. This was the major limitation to the study. Par-
ents may have inferred a positive answer in cases where
ASD or ADHD was suggested by a ~~school psychologist or~~
health worker but not confirmed by further assessment. The
slightly increased drop-out in the ASD and ADHD groups
between waves suggests that our figures for ASD and
ADHD may be slightly underestimated at wave 4. The
effect of drop-out should be the same for reports of ADHD
and ASD; so they do not explain low estimates of ADHD
relative to ASD. US studies using the NHIS question to
parents have shown discrepancies between 'current' and
'previous' diagnoses of autism (Kogan et al. 2009), sug-
gesting a current diagnosis may become invalid as children
mature. Children may no longer meet diagnostic criteria
after symptomatic behaviours at preschool or kindergarten
(Fein et al. 2013; Turner and Stone 2007; Russell et al.
2012); early misdiagnosis may be partially accountable for
ASD over-identification.

A major strength of the current study was the ability to
compare parent-reported ASD and ADHD across social

446 strata. Male sex and lower cognitive ability were the
 447 strongest predictors of both conditions and there was a
 448 tendency for socially disadvantaged groups to have higher
 449 proportions with ADHD, consistent with previous findings
 450 (Akinbami et al. 2011; Banerjee et al. 2007; Hjern et al.
 451 2010; Kogan et al. 2009; Pastor and Rueben 2008; Scahill
 452 et al. 1999). It is unclear whether this effect is due to
 453 differential reporting about the same level of difficulties
 454 between low and high SES groups or whether children
 455 in different socio-economic groups have truly varying
 456 symptom levels, perhaps due to increased stressors in low
 457 SES households, ~~or early environmental insults more~~
 458 ~~common in low SES groups~~ (Boyle et al. 2011). Some US
 459 studies have found a relationship between measures of
 460 social and economic advantage and having a child with
 461 ASD (Fountain et al. 2011; Kogan et al. 2009), in contrast
 462 to our findings which found a link with socio-economic
 463 disadvantage in unadjusted analysis. The results did not
 464 show any link between ASD and older motherhood, or
 465 diagnosed maternal depression, unlike other studies (Dan-
 466 iels et al. 2008; Sandin et al. 2012). There is little evidence
 467 of an association between ASD and ethnicity in studies
 468 outside the US (Zaroff and Uhm 2011). Despite the over-
 469 sampling of ethnic populations in MCS, numbers were too
 470 low to give a meaningful picture of identification within
 471 specific ethnic groups for either disorder: but this is not to
 472 say such associations do not exist.

473 Conclusions

474 The prevalence for clinically identified ASD reported by
 475 parents is higher than previously estimated. Our findings do
 476 suggest that the proportion of children recognised with
 477 ADHD by doctors in the UK is lower than the proportion of
 478 children diagnosed in the US (1.4 % in this UK estimate as
 479 opposed to 6.3 % recorded in the closest US comparator).
 480 This difference in clinical practice in UK settings may be
 481 due to truly lower levels of symptoms, or differing cultural
 482 factors in consideration of the ADHD label. Our study
 483 underlines the need to establish whether trends are under-
 484 pinned by increasing risk, or merely reflect changes in
 485 diagnostic practice. On-going work to establish which
 486 groups of children are most often identified with each
 487 condition is important as differing contexts may lead to
 488 children either missing out on health services, and/or or
 489 over-diagnosis.

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