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ORIGINAL RESEARCH

Rethinking the association between overweight/obesity and ADHD in children: a longitudinal and psychosocial perspective

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Objective: This study examines the association between attention-deficit/hyperactivity disorder (ADHD) and overweight/obesity in a large-scale longitudinal study of children, while controlling for a range of psychosocial factors.

Method: Data were obtained from Growing Up in Ireland, a nationally representative and longitudinal study of approximately 6500 children who were assessed at 9 and 13 years of age. Body mass index (BMI) was determined using measured height and weight, ADHD status was determined by parent reports of professional diagnoses and ADHD symptoms were measured using the Strengths and Difficulties Questionnaire (SDQ).

Results: The associations between ADHD status, ADHD symptoms (SDQ) and BMI category at age 9 and 13 years were evaluated using logistic regression. Adjustments were made for child factors (sex, developmental coordination disorder, emotional symptoms, conduct problems, birth weight and exercise) and parental factors (socio-economic status, parental BMI, parental depression, and maternal smoking and alcohol use during pregnancy). Logistic regression indicated that ADHD status was not associated with BMI category at 9 or at 13 years of age, but children with ADHD at 9 years were significantly more likely to be overweight/obese at 13 years than those without ADHD. However, when other child and parental factors were adjusted for, ADHD status was no longer significantly associated with weight status. Female sex, low levels of exercise, overweight/obese parents and prenatal smoking during pregnancy consistently increased the odds of childhood overweight/obesity.

Conclusions: While ADHD and overweight/obesity co-occur in general populations, this relationship is largely explained by a variety of psychosocial factors.

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Key words: ADHD, longitudinal, obesity, overweight, psychosocial.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is proposed as a risk factor for overweight and obesity (Fliers *et al.* 2013). ADHD is characterised by inattention, hyperactivity and impulsivity (American Psychiatric Association, 2013), and it has an estimated worldwide prevalence of 5.3% in populations under 18 years when using either Diagnostic and Statistical Manual (DSM) or International Classification of Diseases (ICD) criteria (Polanczyk *et al.* 2007). Associations between ADHD and overweight/obesity are reported in child and adolescent clinical (Erermis *et al.* 2004; Holtkamp *et al.* 2004; Agranat-Meged *et al.* 2005; Spencer *et al.* 2006; Swanson *et al.* 2006) and general population studies (Anderson *et al.* 2006; Lam & Yang, 2007). Cortese *et al.* (2015) meta-analysed 30 studies involving children and adolescents indicating significant associations between ADHD and obesity [odds ratio (OR) = 1.20].

It is hypothesised that ADHD and overweight/obesity overlap due to shared genetic vulnerabilities (Faith *et al.* 2013; Frazier-Wood *et al.* 2014) and dysregulation of dopamine reward pathways that are common to both presentations (Cortese & Vincenzi, 2011). Sanders (1983) hypothesised that hypoarousal may explain the behavioural symptoms of ADHD and this notion may also link ADHD with higher BMIs. For instance, individuals with ADHD may use energy-dense foods to maintain adequate arousal levels (Cortese & Vincenzi, 2011). Others speculate that the link between ADHD and overweight/obesity might also be mediated by mood disorders, and that consumption of foods high in fat, sugar and salt might temporarily improve mood (Davis *et al.* 2004).

Deficient attention, response inhibition and planning might lead to poor food choices (i.e. foods high in fat, sugar and salt) and binge eating in the absence of hunger (Cortese & Vincenzi, 2011; Nazar *et al.* 2016).

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Obesity and ADHD have both been linked with impulsive responding and a tendency to value immediate over delayed rewards (Sonuga-Barke *et al.* 1992; Patterson & Newman, 1993; Appelhans, 2009). The immediacy of fast food production means there is little-to-no delay between the initial desire for food and its satisfaction, potentially leading to a preference for fast food in this population. This preference may then be reinforced, as consumption of foods high in fat sugar and salt leads to stimulation of dopaminergic reward pathways (Davis *et al.* 2006; Cortese & Vincenzi, 2011). In support of this, Davis *et al.* (2006) report that ADHD symptoms, including impulsivity, are predictive of overeating behaviours which are in turn predictive of overweight/obesity.

A related body of research has implicated individual differences in sensitivity to reward, with some studies suggesting a link between obesity and a deficiency in reward systems, associated with hypodopaminergic functioning (e.g. Wang *et al.* 2001, 2004). Others claim the reverse – that risk of obesity is linked with heightened sensitivity to reward (e.g. Davis *et al.* 2004; Franken & Muris, 2005; Davis *et al.* 2007). Davis & Fox (2008) provided an apparent solution to this contradiction by describing an inverted-U shaped relationship between sensitivity to reward and BMI. BMI was positively associated with sensitivity to reward in the normal to overweight range, whereas the relationship was reversed in those in the obese range (BMI > 30). This suggests two possible routes to obesity arising from different levels of sensitivity to reward: those who are relatively sensitive to reward are highly motivated to seek out pleasurable foods and will struggle to resist the temptation of palatable foods that are high in sugar and fat. In contrast, individuals with an underactive dopamine reward pathway may 'self-medicate' with pleasurable foods to enhance dopaminergic activation and improve mood (Davis & Fox, 2008). It is this latter pathway which seems most likely to be at play in the ADHD–obesity relationship, as a reduction in sensitivity to reward, and corresponding deficits in dopamine reward pathways, has also been identified in ADHD (Volkow *et al.* 2009, 2011).

While these hypotheses, linking ADHD and overweight/obesity through dopaminergic dysregulation, motivational deficits and mood disorders, appear plausible, the association between ADHD and overweight/obesity may not be so clear-cut, as other clinical and population studies with children and adolescents have not found support for this link (Spencer *et al.* 1996; Biederman *et al.* 2003; Mustillo *et al.* 2003; Braet *et al.* 2007; Nigg *et al.* 2016).

In a recent population study of almost 45 309 children (10–17-year-olds), Nigg *et al.* (2016) found no significant overall association between parental reports of professional diagnoses of ADHD and BMI. Significant associations were only seen in adolescent girls (14–17 years; OR = 1.73), but the effect was attenuated when adjustments were made for depression (OR = 1.48) and when parental reports of depression and conduct disorder were controlled for, the effect was no longer significant (OR = 1.36). Nigg *et al.* (2016) also conducted a meta-analysis of 42 studies and reported that 32.6% of studies found a positive association between ADHD and overweight/obesity. Across these studies, they reported a monotonic increase in effect size as a function of increasing age, and that there was no reliable association between ADHD and overweight/obesity in pre-pubertal children. In addition, the effect was only reliable in adolescent females, not in adolescent males. The authors caution that age, sex and comorbid psychological difficulties may moderate the relationship between ADHD and overweight/obesity.

A host of other demographic and clinical factors relating to children and even their parents may be implicated in the ADHD–BMI link. In terms of child factors, developmental coordination disorder (DCD) is associated with lower levels of physical activity (Rivilis *et al.* 2011) and has a negative relationship with BMI, whereby poorer motor coordination is associated with higher BMI (Lopes *et al.* 2012). Since DCD and ADHD overlap in up to one-third of cases (Fliers *et al.* 2008), DCD may mediate the link between ADHD and overweight/obesity by reducing the child's level of physical activity (Fliers *et al.* 2013). Low birth weight is associated with ADHD (Mick *et al.* 2002), whereas high birth weight is associated with obesity (Yu *et al.* 2011). This variable may therefore serve as a confound in the relationship (Hanć *et al.* 2015). Exercise and reduced sedentary behaviours appear to protect against weight gain (Must & Tybor, 2005), but others suggest that this claim does not extend to boys with ADHD (Holtkamp *et al.* 2004). Depression (Biederman *et al.* 2010) and oppositional defiance (Fliers *et al.* 2013) predict weight status in children and are frequently diagnosed comorbidly with ADHD (Biederman *et al.* 2008; Connor & Doerfler, 2008); these variables must therefore be considered when evaluating the relationship between ADHD and BMI.

Parental psychosocial factors are also independently associated with both ADHD and overweight/obesity in children, so their impacts on the ADHD–BMI link must be considered. These include low socio-economic status (SES; Layte & McCrory, 2011; Russell *et al.* 2015), parental BMI (Rodriguez, 2010), parental depression (Lampard *et al.* 2014) and prenatal maternal smoking (Langley *et al.* 2005; Behl *et al.* 2012). Prenatal maternal alcohol use is associated with ADHD (Gronimus *et al.*

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2009) and low birth weight (McCarthy *et al.* 2013), so its impact on BMI is also of interest.

The majority of studies assessing the ADHD–BMI link are cross-sectional in nature, which restricts interpretation of findings. The evidence from longitudinal studies is mixed, warranting additional research. For instance, Cortese *et al.* (2013) suggested that men with childhood ADHD had significantly higher rates of obesity in adulthood compared to men without childhood ADHD, even after controlling for SES and lifetime mental health problems. In contrast, ADHD was not associated with obesity at annual follow-ups over 8 years (Mustillo *et al.* 2003); instead oppositional defiance disorder and depression (in boys) were related to weight status. Similarly, there was no increased risk for overweight in an ADHD sample over 10 years, but females with comorbid depression were at increased risk of weight gain (Biederman *et al.* 2010). Most recently, Nigg *et al.* (2016) reported no discernible association between ADHD and BMI in children (7– 13 years) assessed yearly over 2 years. Instead age, household income and stimulant use were significant weight-related factors.

These longitudinal studies hint at important implications, for example, the ADHD-overweight/obesity link may be more pronounced at certain developmental stages (e.g. in adulthood, when individuals may be assumed to have more control over their own health-related behaviours) and when comorbid factors are adjusted for in childhood, the effect appears to reduce. Moreover, the research described above suggests that the association between BMI categories and ADHD may be confounded by a wide range of demographic and psychosocial factors. The present study aims to clarify the nature of this relationship in a large and representative sample of young people. Data were obtained from a large cross-sectional and longitudinal cohort of children recruited to the Growing Up in Ireland (GUI) study. The relationship between ADHD and overweight/obesity was assessed at 9 and 13 years of age, and adjusted for the effects of both child factors (sex, birth weight, DCD, emotional symptoms (ES), conduct problems (CP), hyperactivity–inattention (HI), exercise) and parent factors (parental BMI, parental depression, SES, prenatal smoking, alcohol use). Each of these factors has previously been shown to independently influence body weight and ADHD status, and thus may be implicated in the apparent relationship between ADHD and overweight/obesity.

Research questions

1. Is ADHD status associated with BMI category at 9 years and at 13 years of age?
2. Is ADHD status at either age associated with BMI category after controlling for child and parent factors?
3. What factors at 9 years are associated with BMI category at 13 years?

Method

Participants

This study represents a secondary analysis of data from the GUI project, which examines factors that contribute to or undermine the well-being of Irish children. The GUI study received ethical approval from the Research Ethics Committee of the Health Research Board. Wave 1 data were collected between August 2007 and April 2009 for children aged 9 years. Four years later, wave 2 data were collected for the same children, then aged 13 years.

Children were recruited via a random sample of 1105 public, private or special education schools stratified for county, sex mix, disadvantaged status, religious denomination and size (82.3% school response rate). Information packs with consent and assent forms were dispatched to families. In total, 17 054 children were eligible to participate and consent was secured from 9645 (56.6% response rate). Consenting parents were contacted and 8655 home-based interviews were scheduled (89.7% response rate). Children with suspected or professional diagnoses of intellectual disability ($n = 136$) were excluded, leaving a maximum of 8432 individuals at wave 1. There were missing data across all variables of interest, so sample sizes vary throughout, but *n*s are listed where necessary.

Eighty-seven families were excluded from the dataset, either because families requested to be withdrawn from the study or because there was too much missing data. The final dataset included 8568 children, representing about one in seven 9-year-olds resident in Ireland at the time. Data for twins and triplets were collected separately and were not included in this study. At wave 2, data were collected for 7525 individuals (attrition rate of 12.2%).

The majority of children (94.6%) and parents/caregivers (92.6%) were Irish. Respondents were mostly biological parents of study children (95.8%), 3.1% were step-parents and 1% were other parents/caregivers. Respondents were mostly female (98.2%) and 51% of children were female.

Research design

The study was a cross-sectional and longitudinal, between- and within-subjects design. Unadjusted and adjusted logistic regression models were used to examine predictors of BMI categories in children.

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Procedure

Field workers interviewed children and parents/caregivers in their homes, and teacher interviews were conducted in schools. Interviews included survey questions, standardised questionnaires and open-ended questions regarding the children's health, use of health services, diet, exercise, lifestyle, activities, emotional health, well-being, education, attainments, family context, sociodemographics and neighbourhood/community. Key variables were extracted from the GUI dataset based on their comparability with the literature discussed in the introduction section.

Measures

Heights and weights (nearest millimetre and 0.5 kg) of children and their parents were measured by the experimenters during home-based interviews at both waves. BMI was calculated as weight (kilograms) divided by the square of the height (metres).

ADHD and DCD status were determined based on parent/caregivers' 'yes/no' responses about whether these difficulties had been diagnosed by a professional. Thus, categorisation is based on parental reports of professional diagnosis. ADHD symptoms (HI), ES and CP were measured with the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997), a 25-item measure for youths aged between 3 and 16 years. The SDQ has adequate reliability ($\alpha = 0.73$; Goodman, 2001) and validity (Becker *et al.* 2004), and the HI subscale predicts parent reports of professional diagnoses of ADHD (Russell *et al.* 2013). The psychometric properties of the SDQ in the GUI dataset are reported by Nixon (2012); in the present sample, reliabilities for the SDQ were acceptable (mothers $\alpha = 0.79$; fathers $\alpha = 0.86$). Three subscales (ES, CP and HI) are used; scores on each subscale range from 0 to 10, where higher scores reflect greater symptom severity. The total score was highly correlated with the variables of interest (i.e. ES, HI and CP) and was therefore excluded from the regression to avoid issues arising from multicollinearity. Peer problems and pro-social behaviour subscales were not included in the analyses, as these constructs have not been examined in relevant previous literature.

Birth weight (kilograms) was based on parental self-reports. The study child's engagement with exercise was assessed by asking parents how many times in the past 14 days the child engaged in at least 20 minutes of hard exercise enough to make him/her breathe heavily or for their heart to beat faster [adapted from Godin & Shephard (1985)]. Data were collected at both waves using categorical variables (none, 1–2, 3–5, 6–8 and 9–16 times). To facilitate analysis, exercise was recoded as a continuous variable by calculating the average number of times of hard exercise (0, 1.5, 4, 7 and 12.5 times).

Parental occupations were classified as professional/managerial, other non-manual/skilled manual or semi-skilled/unskilled manual (Central Statistics Office, 2006). These categories represent high, medium and low SES classes, respectively. Parents/caregivers completed the 8-item short version of the Centre for Epidemiologic Studies – Depression Scale [CES-D8 (Melchior *et al.* 1993)], at both waves. This measure screens for depressive symptoms in parents/caregivers over the previous week. Scores range from 0 to 24 (≥7 indicates depression). The CES-D8 correlates highly with the CES-D ($r = 0.93$), and it also has demonstrable internal reliability ($\alpha = 0.86$; Melchior *et al.* 1993). In the present sample, the CES-D8 had adequate internal reliability (mothers $\alpha = 0.88$; fathers $\alpha = 0.80$; Nixon, 2012). Mothers were asked how many cigarettes they smoked per day during pregnancy (0, 1–5, 6–10, 11–25 or >26) and for prenatal alcohol use they indicated 'never', 'occasionally', 'weekly' or 'daily'. Responses for prenatal smoking and alcohol use were collapsed to 'no' or 'yes'.

Statistical analyses

IBM SPSS (Version 20) was used for data analysis. Binary logistic regression was performed to assess the strength of the association between categorical and continuous psychosocial variables and BMI categories. BMI scores for parents/caregivers were categorised
as underweight (≤18.49 kg/m^2), healthy weight (18.5–24.9 kg/m^2) or overweight/obese (≥25 kg/m^2). For children and adolescents, BMI categories (underweight, healthy weight, overweight or obese) were calculated using age and sex norms from the International Obesity Task Force (IOTF; Cole & Lobstein, 2012). As the obese group was small ($n = 589$), the overall incidence of ADHD was low (~1%) and a large number of predictor variables were included in the logistic regression analyses described below, the obese group was collapsed with the overweight group ($n = 1576$) to prevent overspecification of the model. The underweight group ($n = 474$) was not included in the analyses; the logistic regression thus compared the healthy weight group with the combined overweight/obese group.

Three models were constructed: Model 1 assessed effects of these variables on BMI at 9 years (wave 1), while Model 2 assessed their effects on BMI at 13 years (wave 2). Model 3 evaluated the effects of baseline

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factors [static variables (e.g. sex, birth weight, maternal prenatal alcohol use, maternal prenatal smoking) and dynamic variables (e.g. ADHD status, DCD status, SDQ HI, SDQ ES, SDQ CP, CES-D8, parental BMI and SES)] at 9 years (wave 1) on BMI category at 13 years (wave 2). Static variables were included in this longitudinal analysis, so that the variance of dynamic variables would not be inflated in significant regression models.

The same procedure was followed for each of the three models. Firstly, unadjusted simple logistic regressions were performed to see if a host of individual factors were associated with BMI category. Secondly, ADHD status as well as all significant factors were subsequently entered into a multiple binary logistic regression (forced entry method). Corrections for multiple comparisons were made for each of the logistic regression models to constrain the family-wise alpha level to 0.05, for example, in all 3 models 13 comparisons were made (testwise $\alpha = 0.004$). Multicolinearity diagnostics were performed, and variables with tolerance <0.1 and variance inflation factors >10 were excluded from further regression analyses (SDQ-Total). The variables included in each model and the results of the regressions are reported in Tables 3–5. ORs are listed for all predictor variables. An OR greater than 1 indicates that, as the predictor increases, the odds of the outcome occurring increase. In contrast, a value that less than 1 indicates that, as the predictor increases, the odds of the outcome occurring decrease.

Results

Descriptive statistics and demographic details

Based on parental reports of professional ADHD diagnoses, 0.8% ($n = 71$; $n = 8568$) of the sample had a diagnosis at wave 1 and 1.2% ($n = 87$, $n = 7525$) had a diagnosis at wave 2. Of those with ADHD at wave 2, 43.1% ($n = 28$) also had ADHD at wave 1. An exact McNemar's test determined that there was no statistically significant difference in the proportion of children with ADHD across waves 1 and 2 ($p = 0.113$). Six children with ADHD at wave 1 dropped out by wave 2.

ADHD status was stratified by BMI categories (underweight, healthy weight and overweight/obese) and sex for 9-year-olds and 13-year-olds, and is reported in Table 1.

Descriptive statistics for the underweight group are listed in the interests of completeness, but this group was not included in subsequent regression analyses. In total, 65.6% of children at 9 years were in the healthy BMI range, 6.1% were underweight and 27.9% were

Table 1. *Frequencies of ADHD by sex, age and BMI category*

	BMI category (9 years)				BMI category (13 years)			
	Frequency	Underweight	Healthy weight	Overweight/obese	Frequency	Underweight	Healthy weight	Overweight/obese
All children	7746 (100%)	474 (6.1%)	5107 (65.9%)	2165 (27.9%)	6923 (100%)	358 (5.2%)	4709 (68%)	1856 (26.8%)
ADHD – no	7679 (99.1%)	470 (6.1%)	5068 (66%)	2141 (27.9%)	6845 (98.9%)	357 (5.2%)	4663 (68.1%)	1825 (26.7%)
ADHD – yes	67 (0.9%)	4 (6%)	39 (58.2%)	24 (35.8%)	78 (1.1%)	1 (1.3%)	46 (59%)	31 (39.7%)
Boys	3762 (100%)	218 (5.8%)	2616 (69.5%)	928 (24.7%)	3400 (100%)	162 (4.8%)	2425 (71.3%)	813 (23.9%)
ADHD – no	3709 (98.6%)	214 (5.8%)	2587 (69.7%)	908 (24.5%)	3346 (98.4%)	161 (4.8%)	2392 (71.5%)	793 (23.7%)
ADHD – yes	53 (1.4%)	4 (7.5%)	29 (54.7%)	20 (37.7%)	54 (1.6%)	1 (1.9%)	33 (61.1%)	20 (37%)
Girls	3984 (100%)	256 (6.4%)	2491 (62.5%)	1237 (31.1%)	3523 (100%)	196 (5.6%)	2284 (64.8%)	1043 (29.6%)
ADHD – no	3970 (99.6%)	256 (6.4%)	2481 (62.5%)	1233 (31.1%)	3499 (99.3%)	196 (5.6%)	2271 (64.9%)	1032 (29.5%)
ADHD – yes	14 (0.04%)	0 (0%)	10 (71.4%)	4 (28.6%)	24 (0.7%)	0 (0%)	13 (54.2%)	11 (45.8%)

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overweight/obese. BMI categories were similarly distributed at 13 years (under-weight = 5.2%; healthy weight = 68%; overweight/obese = 26.8%).

At wave 1, boys (77.8%) were more likely than girls (22.2%) to have ADHD [χ^2 (1, $n = 7272$) = 21.46, $p < 0.001$]. Similarly, at wave 2, boys (68.8%) were more likely than girls (31.2%) to have ADHD [χ^2 (1, $n = 6565$) = 11.86, $p < 0.01$]. In terms of weight status, girls (57.1%) were more likely than boys (42.9%) to be overweight/obese at wave 1 [χ^2 (1, $n = 7272$) = 42.53, $p < 0.001$]. Moreover, this sex difference in weight status was maintained at wave 2, again as girls (56.2%) were more likely than boys (43.8%) to be overweight/obese [χ^2 (1, $n = 6565$) = 31.52, $p < 0.001$].

Other demographic and clinical characteristics are presented in Table 2. Less than 1% of children had a parental report of a professional diagnosis of DCD (wave 1 = 0.8% and wave 2 = 0.7%). The majority of children at 9 years came from families classified in the high SES group (55.5%), the remainder were medium SES (35.5%) or low SES (9%). Approximately half of parents were overweight/obese (50.1%), 49% were in the healthy BMI range, while 0.9% were underweight. Average ratings of parental depression were in the normal range (<7). The majority of mothers reported not smoking (77.7%) or drinking alcohol (60.8%) during pregnancy. SES distributions at 13 years were similar to 9 years (high = 59.4%, medium = 33.8% and low = 7.2%). Once again, the majority of parents fell within the overweight/obese range but this saw an increase from 50.1% to 57% at 13 years. Average parental depression was in the normal range.

Dropout analysis

A total of 1043 children (12.2% of the sample) dropped out between wave 1 and wave 2. Independent *t*-tests and chi-square tests were used to compare wave 1 outcomes between dropouts and non-dropouts. Children who dropped out had higher BMIs at wave 1 ($M = 18.46$, S.D. = 3.12) than non-dropouts [$M = 18.06$, S.D. = 2.77; t (1072.62) = 3.59, $p < 0.001$]; dropouts had higher scores on the SDQ–hyperactivity–inattention (SDQ–HI) subscale ($M = 3.13$, S.D. = 2.40) than non-dropouts [$M = 2.93$, S.D. = 2.40; t (7261) = 2.32, $p < 0.05$]; dropouts had significantly lower birth weights than non-dropouts [t (1079.43) = −2.35, $p < 0.05$]; and parental depression (CES-D8) was higher among dropouts than non-dropouts [t (886.17) = 3.42, $p < 0.01$]. There were no differences between dropouts and non-dropouts in terms of SDQ CP [t (7267) = 0.03, $p > 0.05$], SDQ ES [t (7261) = 1.93, $p > 0.05$] and exercise [t (1112.47) = −1.19, $p > 0.05$]. The prevalence of ADHD between dropouts (1.3%) and non-dropouts (0.8%) was not significantly different [i^2 (1, $n = 7272$) = 1.73, $p > 0.05$].

Hypothesis 1: Is ADHD status associated with BMI category at 9 and at 13 years of age?

At wave 1, over 35.8% of children with ADHD were in the overweight/obese range, in comparison to 27.9% of children without ADHD (see Table 1). Moreover, 0.8% of those in the healthy weight range and 1.1% of those in the overweight/obese range had a diagnosis of ADHD. However, ADHD status at 9 years was not associated with BMI category. The SDQ–HI scale, a measure of ADHD symptoms, was also not associated with BMI category (see Table 3). Overweight/obesity prevalence among 13-year-olds with ADHD increased to 39.7% at wave 2, but only 26.7% of non-ADHD adolescents were overweight/obese (see Table 1). In addition, 1% of the healthy weight group had ADHD and 1.7% of the overweight/obese group had ADHD. Nevertheless, at 13 years, ADHD status and SDQ–HI were not significantly associated with BMI category (see Table 4).

Hypothesis 2: Is ADHD status associated with BMI category after controlling for psychosocial factors?

At wave 1, simple logistic regression indicated that there were significant effects of sex, DCD, SDQ (ES and CP), birth weight, exercise, SES, parental BMI, and maternal prenatal smoking and alcohol use, but not ADHD status. Next, all significant independent variables were entered into a multiple logistic regression to see if there was a combined association with the dependent variable (BMI category at 9 years; Table 3). This resulted in a significant model χ^2 (13) = 383.29, $p < 0.001$, $R^2 = 0.059$ (Cox and Snell), 0.084 (Nagelkerke), but there was no significant effect of ADHD status on BMI status. Overweight/obesity was less likely in males than females (OR = 0.73), as birth weight increased the odds of overweight/obesity increased (OR = 1.30), exercise reduced the odds of overweight/obesity (OR = 0.96), overweight/obesity was less likely in high SES *versus* low SES families (OR = 0.74), healthy weight parents were less likely to have overweight/obese children than overweight/obese parents (OR = 0.51), maternal prenatal smoking increased the odds of overweight/obesity (OR = 1.62) and maternal prenatal alcohol use reduced the likelihood of overweight/obesity (OR = 0.79).

At wave 2, sex, SDQ (ES and CP), exercise, parental BMI, parental depression, prenatal maternal smoking and alcohol use were each independently associated with BMI category in simple logistic regressions (Table 4). Again, all significant independent variables were entered into a multiple logistic regression to see

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Table 2. *Frequencies and mean scores of key child and parent factors for underweight, healthy weight and overweight/obese categories*

Variable	BMI category (9 years)			Variable	BMI category (13 years)		
	Underweight	Healthy weight	Overweight/obese		Underweight	Healthy weight	Overweight/obese
	Frequencies				Frequencies		
Sex ($n = 7746$)				Sex ($n = 6923$)			
Male (48.6%, $n = 3762$)*	5.8%	69.5%	24.7%	Male (49.1%, $n = 3400$)*	4.8%	71.3%	23.9%
Female (51.4%, $n = 3984$)*	6.4%	62.5%	31%	Female (50.9%, $n = 3523$)*	5.6%	64.8%	29.6%
DCD ($n = 7746$)				DCD ($n = 6923$)			
Yes (0.8%, $n = 62$)*	9.7%	46.8%	43.5%	Yes (0.7%, $n = 51$)*	5.9%	52.9%	41.2%
No (99.2%, $n = 7684$)*	6.1%	66.1%	27.8%	No (99.3%, $n = 6872$)*	5.2%	68.1%	26.7%
SES ($n = 7339$)				SES ($n = 5749$)			
High (55.5%, $n = 4071$)*	6.3%	69%	24.7%	High (59%, $n = 3434$)*	5%	67.4%	27.5%
Medium (35.5%, $n = 2606$)*	6%	63.5%	30.5%	Medium (33.8%, $n = 1810$)*	5.5%	68%	26.6%
Low (9%, $n = 662$)*	6%	58.8%	35.2%	Low (7.2%, $n = 505$)*	4.2%	69.5%	26.3%
Parental BMI ($n = 7341$)				Parental BMI ($n = 6577$)			
Underweight (0.9%, $n = 69$)*	14.5%	71%	14.5%	Underweight (0.7%, $n = 46$)*	13%	67.4%	19.6%
Healthy weight (49%, $n = 3597$)*	7.8%	72.3%	19.9%	Healthy weight (42.3%, $n = 2784$)*	7.6%	75.1%	17.3%
Overweight/Obese (50.1%, $n = 3675$)*	4.3%	60.3%	35.4%	Overweight/Obese (57%, $n = 3747$)*	3.5%	63.3%	33.1%
Smoking during pregnancy ($n = 7460$)				Smoking during pregnancy ($n = 6664$)			
Yes (22.3%, $n = 5799$)*	5.2%	59.9%	34.9%	Yes (21.6%, $n = 1440$)*	4.9%	59.1%	36%
No (77.7%, $n = 1661$)*	6.4%	67.7%	26%	No (78.4%, $n = 5224$)*	5.3%	70.6%	24.1%
Alcohol during pregnancy ($n = 7458$)				Alcohol during pregnancy ($n = 6663$)			
Yes (39.2%, $n = 2921$)*	5.9%	69.1%	25%	Yes (40.5%, $n = 2697$)*	5.7%	70.7%	23.6%
No (60.8%, $n = 4537$)*	6.2%	63.9%	29.8%	No (59.5%, $n = 3966$)*	4.9%	66.3%	28.8%
	Mean scores (S.D.)				Mean scores (S.D.)		
BMI ($n = 7746$)	13.55 (0.73)	16.59 (1.22)	21.69 (2.23)	BMI ($n = 6923$)	15.41 (0.63)	19.11 (1.61)	25.49 (2.79)
Percentage of total sample	6.1%	65.9%	27.9%	Percentage of total sample	5.2%	68%	26.8%
Birth Weight (kg) ($n = 7652$)	3.34 (0.56)	3.52 (0.60)	3.59 (0.61)	Birth Weight (kg) ($n = 6836$)	3.45 (0.58)	3.52 (0.60)	3.57 (0.62)
Exercise ($n = 7745$)	9.13 (4.20)	9.33 (3.97)	8.45 (4.18)	Exercise ($n = 6867$)	6.29 (4.30)	6.88 (4.32)	5.65 (4.06)
SDQ parent				SDQ parent			
Emotional ($n = 7742$)	1.90 (1.92)	1.91 (1.91)	2.17 (2.05)	Emotional ($n = 6923$)	1.97 (2.10)	1.62 (1.80)	1.96 (2.03)
Conduct ($n = 7736$)	1.13 (1.30)	1.19 (1.40)	1.36 (1.48)	Conduct ($n = 6923$)	1.00 (1.23)	1.03 (1.31)	1.19 (1.45)
Hyper/inattention ($n = 7734$)	2.88 (2.35)	2.91 (2.37)	3.06 (2.46)	Hyper/inattention ($n = 6923$)	2.32 (2.44)	2.46 (2.27)	2.60 (2.35)
Parental depression ($n = 7138$)	2.08 (3.20)	2.04 (3.23)	2.11 (3.43)	Parental depression ($n = 6433$)	2.28 (3.23)	2.27 (3.23)	2.63 (3.62)

ADHD, attention-deficit/hyperactivity disorder; DCD, developmental coordination disorder; SES, socio-economic status; BMI, body mass index; SDQ, Strengths and Difficulties Questionnaire.
* Denotes column percentages. All other percentages denote percentages of total sample.

Table 3. *Binary logistic regression for psychosocial variables associated with BMI at 9 years (Model 1)*

	Unadjusted simple regression analyses					Adjusted multiple regression analyses				
Variable	*n*	B	Wald	OR [95% CI]	*p*	*n*	B	Wald	OR [95% CI]	*p*
ADHD W1	7272	0.38	2.08	1.46 [0.87–2.45]	0.149	6328	−0.08	0.07	0.92 [0.50–1.69]	0.79
Sex	7272	−0.34	42.38	0.71 [0.65–0.79]	**0.000***	6328	−0.31	27.94	0.73 [0.65–0.82]	**0.000***
DCD W1	7272	0.80	8.73	2.21 [1.31–3.74]	**0.003***	6328	0.63	4.19	1.87 [1.03–3.41]	0.041
SDQ–ES	7269	0.07	26.00	1.07 [1.04–1.10]	**0.000***	6328	0.03	3.05	1.03 [1.00–1.06]	0.081
SDQ–CP	7263	0.08	19.03	1.08 [1.04–1.12]	**0.000***	6328	0.05	5.35	1.05 [1.01–1.10]	0.021
SDQ–HI	7263	0.03	5.80	1.03 [1.01–1.05]	0.016	–	–	–	–	–
Birth weight	7188	0.20	22.23	1.22 [1.12–1.33]	**0.000***	6328	0.27	30.81	1.30 [1.12–1.43]	**0.000***
Exercise W1	7271	−0.05	71.46	0.95 [0.94–0.96]	**0.000***	6328	−0.05	41.38	0.96 [0.94–0.97]	**0.000***
SES W1	6888	–	46.92	–	**0.000***	6328	–	14.01		0.001*
High *v.* low		−0.52	32.25	0.60 [0.50–0.71]	**0.000***	6328	−0.30	8.87	0.74 [0.61–0.90]	**0.003***
Medium *v.* low		−0.22	5.58	0.80 [0.67–0.96]	0.018	6328	−0.11	1.16	0.90 [0.74–1.09]	0.281
Parental BMI W1	6891	–	194.73	–	**0.000***	6328	–	133.72		**0.000***
Under *v.* O/Obese		−1.06	9.16	0.35 [0.18–0.69]	**0.002***	6328	−0.98	6.74	0.38 [0.18–0.79]	0.009
Healthy *v.* O/Obese		−0.76	190.11	0.47 [0.42–0.52]	**0.000***	6328	−0.67	130.33	0.51 [0.46–0.58]	**0.000***
Depression W1	6698	0.01	0.61	1.01 [0.99–1.02]	0.436	–	–	–	–	–
Smoke during pregnancy	7004	0.42	47.45	1.52 [1.35–1.71]	**0.000***	6328	0.48	47.65	1.62 [1.41–1.86]	**0.000***
Alcohol during pregnancy	7003	−0.25	21.86	0.78 [0.70–0.86]	**0.000***	6328	−0.23	15.24	0.79 [0.70–0.89]	**0.000***
Constant	–	–	–	–	–	6328	−0.92	19.34	0.40	**0.000***

ADHD, attention-deficit/hyperactivity disorder; DCD, developmental coordination disorder; SDQ, Strengths and Difficulties Questionnaire; ES, emotional symptoms; CP, conduct problems,; HI, hyperactivity–inattention,; SES, socio-economic status; BMI, body mass index; OR, odds ratio; W1, wave 1.
* Corrected α value = 0.004 (13 comparisons in this model and significant effects are highlighted in bold).

Table 4. *Binary logistic regression for psychosocial variables associated with BMI at 13 years (Model 2)*

	Unadjusted simple regression analyses					Adjusted multiple regression analyses				
Variable	*n*	*B*	Wald	OR [95% CI]	*p*	*n*	*B*	Wald	OR [95% CI]	*p*
ADHD W2	6565	0.54	5.39	1.72 [1.09–2.72]	0.020	5896	0.22	0.66	1.25 [0.73–2.13]	0.417
Sex	6565	−0.31	31.43	0.73 [0.66–0.82]	**0.000***	5896	−0.21	10.86	0.81 [0.72–0.92]	**0.001***
DCD W1	6565	0.69	5.50	1.98 [1.12–3.52]	0.019	5896	0.61	3.27	1.8 [0.95–3.56]	0.071
SDQ–ES	6565	0.10	45.94	1.01 [1.07–1.13]	**0.000***	5896	0.05	8.19	1.05 [1.02–1.09]	0.004
SDQ–CP	6565	0.09	18.72	1.09 [1.05–1.13]	**0.000***	5896	0.02	0.87	1.02 [0.98–1.07]	0.352
SDQ–HI	6565	0.03	5.25	1.03 [1.00–1.05]	0.022	–	–	–	–	–
Birth weight	6482	0.12	6.67	1.13 [1.03–1.23]	0.010	–	–	–	–	–
Exercise W2	6512	−0.07	107.11	0.93 [0.92–0.95]	**0.000***	5896	−0.05	48.75	0.95 [0.94–0.96]	**0.000***
SES W2	5456	–	0.77	–	0.682	–	–	–	–	–
High *v.* low		0.08	0.48	1.07 [0.87–1.34]	0.488	–	–	–	–	–
Medium *v.* low		0.03	0.08	1.03 [0.82–1.29]	0.784	–	–	–	–	–
Parental BMI W2	6227	–	177.91	–	**0.000***	5896	–	145.79	–	**0.000***
Under *v.* O/Obese		−0.59	2.41	0.55 [0.26–1.17]	0.121	5896	−0.62	2.50	0.54 [0.25–1.16]	0.114
Healthy *v.* O/Obese		−0.82	177.18	0.44 [0.39–0.50]	**0.000***	5896	−0.77	144.91	0.46 [0.41–0.52]	**0.000***
Depression W2	6507	0.03	14.75	1.03 [1.02–1.05]	**0.000***	5896	0.01	2.01	1.01 [1.00–1.03]	0.156
Smoke during pregnancy	6316	0.58	80.72	1.79 [1.57–2.03]	**0.000***	5896	0.55	62.09	1.74 [1.52–2.00]	**0.000***
Alcohol during pregnancy	6315	−0.26	20.26	0.77 [0.69–0.86]	**0.000***	5896	−0.23	13.27	0.80 [0.71–0.90]	**0.000***
Constant	–	–	–	–	–	5896	−0.43	32.20	0.65	**0.000***

ADHD, attention-deficit/hyperactivity disorder; DCD, developmental coordination disorder; SDQ, Strengths and Difficulties Questionnaire; ES, emotional symptoms; CP, conduct problems; HI, hyperactivity–inattention; SES, socio-economic status; BMI, body mass index; OR, odds ratio; W2, wave 2.
* Corrected α value = 0.004 (13 comparisons in this model and significant effects are highlighted in bold).

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Table 5. *Binary logistic regression for psychosocial variables at 9 years associated with BMI at 13 years (Model 3)*

	Unadjusted simple regression analyses					Adjusted multiple regression analyses				
Variable	*n*	*B*	Wald	OR and 95% CI	*p*	*n*	*B*	Wald	OR and 95% CI	*p*
ADHD status W1	5991	0.90	9.32	2.47 [1.38–4.42]	**0.002***	5263	0.61	3.40	1.90 [0.96–4.76]	0.065
Sex	5991	−0.32	31.43	0.73 [0.65–0.81]	**0.000***	5263	−0.29	19.02	0.75 [0.66–0.85]	**0.000***
DCD	5991	0.72	5.61	2.06 [1.13–3.73]	0.018	–	–	–	–	–
SDQ–ES W1	5989	0.07	26.51	1.08 [1.05–1.11]	**0.000***	5263	0.05	7.34	1.05 [1.01–1.08]	0.007
SDQ–CP W1	5983	0.08	14.52	1.08 [1.04–1.12]	**0.000***	5263	0.03	1.90	1.03 [0.98–1.08]	0.297
SDQ–HI W1	5984	0.04	13.86	1.05 [1.02–1.07]	**0.000***	5263	0.01	0.22	0.101 [0.98–1.04]	0.638
Birth weight	5920	0.07	1.85	1.07 [0.97–1.17]	0.173	–	–	–	–	–
Exercise W1	5990	−0.05	49.25	0.95 [0.94–0.97]	**0.000***	5263	−0.04	26.48	0.96 [0.95–0.98]	**0.000***
SES W1	5712	–	51.71	–	**0.000***	5263	–	13.93	–	**0.001***
High *v.* low	5712	−0.56	29.83	0.57 [0.47–0.70]	**0.000***	5263	−0.30	7.41	0.74 [0.60–0.92]	0.006
Medium *v.* low	5712	−0.18	3.03	0.83 [0.68–1.02]	0.082	5263	−0.08	0.51	0.92 [0.74–1.15]	0.477
Parental BMI W1	5691	–	156.25	–	**0.000***	5263	–	125.08	–	**0.000***
Under *v.* O/Obese	5691	−1.49	9.84	0.24 [0.09–0.57]	**0.002***	5263	−1.95	10.28	0.14 [0.04–0.47]	**0.001***
Healthy *v.* O/Obese	5691	−0.75	149.94	0.47 [0.42–0.53]	**0.000***	5263	−0.71	117.61	0.49 [0.43–0.56]	**0.000***
Parental depression W1	5581	0.02	3.35	1.02 [1.00–1.04]	0.067	–	–	–	–	–
Smoke during pregnancy	5772	0.61	82.02	1.83 [1.61–2.09]	**0.000***	5263	0.57	54.95	1.76 [1.52–2.05]	**0.000***
Alcohol during pregnancy	5772	−2.59	18.64	0.77 [0.69–0.87]	**0.000***	5263	−0.20	9.07	0.82 [0.72–0.93]	**0.003***
Constant	–	–	–	–	**–**	5263	−0.14	1.03	0.87	0.310

ADHD, attention-deficit/hyperactivity disorder; DCD, developmental coordination disorder; SDQ, Strengths and Difficulties Questionnaire; ES, emotional symptoms; CP, conduct problems; HI, hyperactivity–inattention; SES, socio-economic status; BMI, body mass index; OR, odds ratio; W1, wave 1.
* Corrected α value = 0.004 (13 comparisons in this model and significant are highlighted in bold). Underweight individuals were excluded from waves 1 and 2 for this analysis, which further reduced *n* values.

if there was a combined association with the dependent variable (BMI category at 13 years), resulting in a significant model [χ^2 (10) = 375.41, $p < 0.001$, $R^2 = 0.062$ (Cox and Snell), 0.089 (Nagelkerke)]. However, there was no significant effect of ADHD status on BMI status. Overweight/obesity was less likely in males than females (OR = 0.81), exercise reduced the odds of overweight/obesity (OR = 0.95), adolescent overweight/obesity was less likely for healthy weight parents than overweight/obese parents (OR = 0.46), maternal prenatal smoking increased the odds of overweight/obesity (OR = 1.74) and maternal prenatal alcohol use reduced the likelihood of overweight/obesity (OR = 0.80).

Hypothesis 3: What factors at 9 years are associated with BMI category at 13 years?

ADHD status, sex, SDQ (ES, CP and HI), exercise, SES, parental BMI, and prenatal maternal smoking and alcohol use recorded at wave 1 were each significantly associated with BMI category at wave 2 in simple logistic regressions (Table 5). All significant factors were entered in the multiple regression, and main effects were seen for sex, exercise, parental BMI, prenatal maternal smoking and alcohol use, χ^2 (12) = 332.73, $p < 0.001$, (Cox and Snell = 0.06, Nagelkerke = 0.09). No significant effect of ADHD status was observed. ORs for main effects were in the same direction as those described above (see Table 5).

Discussion

This study aimed to investigate the relationship between ADHD and overweight/obesity in a large longitudinal and cross-sectional sample of Irish children. ADHD status was not associated with BMI category at 9 or at 13 years, but ADHD at 9 years increased the odds of overweight/obesity at 13 years. However, when key child and parental psychosocial factors were adjusted for, this association weakened and was no longer significant. This suggests that the association between ADHD and overweight/obesity might be due to confounding variables, at least in general populations of children.

Notably, the overall rate of ADHD in this study was about 1%, which is considerably lower than the 5.3% reported elsewhere (Polanczyk *et al.* 2007) and could potentially affect statistical power. ADHD status in the GUI study is based on parental reports of professional diagnoses rather than an objective diagnostic measure. A large cohort study conducted in the United Kingdom using similar methods reported comparable ADHD rates of 1.4% (Russell *et al.* 2014);

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however, the reliability and validity of this assessment of ADHD status are undetermined. The low rates of ADHD observed in this sample may be due to a combination of factors thought to linked with under-diagnosis of the condition, including differing diagnostic assessments or diagnostic overshadowing (Taylor, 2017), the conservative culture of ADHD diagnosis in Ireland (Adamis *et al.* 2019) and parental difficulty in differentiating childhood behavioural disorders (Sayal *et al.* 2006). Despite this limitation, our findings are comparable to recent empirical and meta-analytic work conducted by Nigg *et al.* (2016), who concluded that there was no reliable association between ADHD and overweight/obesity in children. In the present study, factors that were consistently associated with overweight/obesity included female sex, low levels of exercise, parental overweight/obesity and maternal prenatal smoking. At 9 years, high birth weight and low SES were also associated with overweight/obesity.

In addition, females were consistently more likely to be overweight/obese than males at both developmental stages. Fliers *et al.* (2013) reported that the association between ADHD and weight status varied greatly according to sex and age, whereby middle childhood females (10–12 years) with ADHD were four times as likely to be obese as matched controls without ADHD. Nigg *et al.* (2016) reported similar results, but added that when depression and conduct disorder were controlled for, there was no association between ADHD and BMI. Higher levels of exercise consistently reduced the impact of overweight/obesity at 9 and 13 years, and greater exercise at 9 years was associated with reduced BMI at 13 years. This finding is in line with the energy expenditure hypothesis of overweight (Must & Tybor, 2005), which states that reduced levels of exercise lead to the accumulation of adipose tissue. As boys are reported to be more physically active than girls (Layte & McCrory, 2011), this may explain part of the sex differences observed across weight distributions.

Higher birth weights tend to be predictive of overweight later in life (Yu *et al.* 2011). In the present study, high birth weight was associated with overweight/obesity at 9 years but this effect attenuated over time. There were trends towards significant associations between CP and overweight/obesity at 9 years (although these did not reach statistical significance following correction for multiple comparisons), and elevations in ES were significantly associated with overweight/obesity at follow-up (13 years). It is interesting that psychological distress appeared to change from externalising to internalising problems between childhood and adolescence. Others have previously described the associations between increased weight and CP (Fliers *et al.* 2013), and depression in females (Biederman *et al.* 2010; Nigg *et al.* 2016), and a trajectory of increasing internalising symptoms has been associated with risk for subsequent mental health problems (Toumbourou *et al.* 2011).

In a large-scale general population study (van Egmond-Fröhlich *et al.* 2012), there was no independent relationship between ADHD symptoms and overweight/obesity when SES, parental BMI and parental smoking were controlled for, and this study reports similar findings. Low SES is associated with poor nutrition, increased media use and reduced physical activity (Barkley, 2008), which all have implications for weight status of parents and their children. Additionally, mothers who smoke during pregnancy tend to weigh more, have lower SES and have fewer years of education (Oken *et al.* 2008; Ino, 2010). In this study, low SES was associated with overweight/obesity at 9 years, but this effect diminished during adolescence. It is however important to note that the majority of participants in this sample reported their SES in the high or medium bracket, with only 9% of participants falling in the low SES category. These results therefore require replication in a sample with a more balanced socio-economic profile. Prenatal exposure to alcohol consistently reduced the odds of overweight/obesity, which is likely related to a failure to thrive and should not be interpreted as a protective factor. In sum, it seems then that parental variables are interrelated and their associations with childhood weight status are non-linear.

There are limitations of the GUI study that are noteworthy. Some of the measures were based on retrospective self-report (birth weight, exercise, parental smoking and alcohol use), so their reliability may be compromised. In addition, factors such as psychostimulant treatment (Poulton *et al.* 2013), diet (Wilborn *et al.* 2005), eating behaviours (Nazar *et al.* 2016), markers of immune system dysfunction (Lynch *et al.* 2017) and comorbid physical health problems (Lam *et al.* 2012) were not measured and deserve attention when considering biological confounders of the BMI and ADHD link. Finally, the generalisability of the study is limited by the fact that ADHD symptoms were assessed using the SDQ rather than an ADHD-specific measure. The SDQ has the considerable benefit of being short and easy to administer to large samples of parents and teachers. It is however important to note that it is a brief screening questionnaire for child psychopathology rather than a diagnostic instrument (Goodman *et al.* 1997), and is not specifically targeted towards the measurement of ADHD symptoms. The accuracy with which ADHD symptoms are measured in the GUI data is thus called into question. Moreover, the baseline differences between dropouts and non-dropouts (e.g. BMI, SDQ–HI, birth weight and

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parental CES-D8) affect generalisability of findings. Importantly, the present study employed a very large sample, providing adequate power to overcome noisy measures. Nevertheless, future cohort studies like the GUI – and indeed future waves of the GUI itself – should carefully consider the sensitivity and utility of the measures included. It is of course necessary in large cohort studies to balance comprehensiveness with efficiency in order to increase recruitment and sample retention, but future researchers may wish to consider whether the measures included are sufficient to accurately assess the psychological conditions of interest.

Despite these concerns, the GUI study is a large and representative sample of Irish children. Data were collected at two developmental stages, controlling for child and parental psychosocial factors. Previous studies (van Egmond-Fröhlich *et al.* 2012; Fliers *et al.* 2013) controlled for child and parental factors separately and were cross-sectional in nature. BMI scores for children and parents/caregivers are based on measured weight and height, not self-reports which can underestimate BMI (Merrill & Richardson, 2009). In addition, BMI categories for children and adolescents were age and sex-normed using gold standard IOTF criteria.

In conclusion, the results of this population study suggest that ADHD may not be associated with BMI category in a general population of Irish children when other confounding psychosocial factors are controlled for. While ADHD and overweight/obesity can co-occur, the association is weak within a general population, particularly in childhood. Female children appear to be most vulnerable to developing overweight/obesity, suggesting that they require targeted intervention. A family systems approach to treatment might be most suitable since exercise and ES were associated with BMI category, and there is good evidence for the effectiveness of these approaches to childhood obesity intervention (see Sung-Chan *et al.* 2013 for a review). From a prevention perspective, maternal obesity and prenatal smoking and alcohol use should be sensitively addressed through public health and primary care services.

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Conflicts of interest

Authors SÓD, JB and CG have no conflicts of interest to disclose.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committee on human experimentation with the Helsinki Declaration of 1975, as revised in 2008. The Growing Up in Ireland study protocol was approved by the Research Ethics Committee of the Health Research Board of Ireland.

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