**The Association Between Childhood Attention Deficit Hyperactivity Disorder (ADHD) Problems and Obesity at Age 42: Using the Prospective 1970 British Cohort Sample**

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Abstract

Backgrounds: Previous studies suggest an association between childhood Attention Deficit Hyperactivity Disorder (ADHD) and adulthood obesity. However, there are no longitudinal studies investigating this for both genders into the fourth decade of life. Obesity is an important healthcare burden, and identifying risk factors could help with early prevention.

Methods: This prospective, longitudinal study uses 8282 cases from the 1970 British cohort study. The relationship between age 10 ADHD and age 10 and age 42 obesity was assessed using logistic regression, for both genders. The association between children with only inattention and only hyperactivity/impulsivity, and adulthood obesity was also assessed.

Results: There is no significant association between childhood ADHD and age 10 obesity. For males with childhood ADHD, there was a significant relative risk of 1.40 (1.04, 1.87) p=0.026 for obesity at age 42, after adjusting for social class. For females, the association was not statistically significant (OR=1.35 (0.93, 1.95), p=0.113) after adjusting for conduct problems. Children with inattention only showed a significant association with obesity (OR=1.37 (1.09, 1.73), p=0.007).

Interpretation: There is a significant independent association between male childhood ADHD problems and adulthood obesity and it is the inattention aspect involved. Further research is needed to identify the causal factors.

Introduction

Attention Deficit and Hyperactivity Disorder (ADHD) is one of the most common childhood psychiatric disorders, affecting 5-10% of children worldwide (Nigg, 2013). It is characterised by three core symptom clusters: hyperactivity, impulsivity and inattention (Erskine et al, 2016). Diagnostic and statistics manual of mental disorders (DSM-V) describes three presentations of ADHD; inattentive (ADD), hyperactive-impulsive and combined (presents with all three symptoms) (American Psychiatric Association, 2013).

In approximately 65% of childhood cases, ADHD problems persist into adulthood (Cortese et al, 2013). Research shows that childhood ADHD can predispose people to multiple problems in adulthood, regardless of persistence. There is a well-established association between childhood ADHD and poorer social outcomes, such as higher criminality and poorer educational achievement and employment outcomes (Erskine et al, 2016, Klein et al, 2012). Recently, there was a systematic review of 278 longitudinal and case-controlled studies exploring the consequences of ADHD and Conduct Disorder in adulthood (Erskine et al, 2016). The review found that the majority of evidence about the long-term outcomes of ADHD centres around adverse social effects. There have also been longitudinal studies showing higher levels of substance use disorder, antisocial personality disorder and depression in adults who had childhood ADHD (Klein et al, 2012).

There has been some research focusing on the association between ADHD and long term physical health outcomes, finding increased rates of physical injuries, sleep disorders, sexually transmitted diseases and global decline of health (Nigg, 2013). Many American studies have shown an association between childhood ADHD and obesity, however evidence has been varied in both the results and the robustness of the methodology (Holtkamp et al, 2004).

Obesity is an important outcome to investigate. It is the presence of excess body fat and can be defined as a Body Mass Index (BMI) greater than 30 (Marcus and Wildes, 2009). The prevalence of adulthood obesity in England was reported at 26.9% in 2017, which has increased by 12% since 1993 (Public Heath England, 2017). Obesity is associated with multiple health conditions including cardiovascular disease, type II diabetes mellitus, hypertension, strokes, osteoarthritis and some cancers (Must et al, 1999). Cardiovascular disease accounts for the most non-communicable deaths in the UK (17.5 million per year) followed by cancers, and diabetes is fourth with 1.5 million deaths per year (Non-Communicable Diseases, 2015). Obesity is therefore a significant cause of both morbidity and mortality (Cortese et al, 2016) and is estimated to cost the National Health Service (NHS) £3.2 billion pounds per annum (Allender and Rayner, 2007). Obesity needs to be addressed by identifying the risk factors in order to develop appropriate interventions (Fuemmeler et al, 2011).

There have been inconsistencies in the research surrounding ADHD and obesity, as some studies have found no association, whereas others have. For example, a retrospective US chart review looked at 140 charts of children with ADHD aged 3-18 years from a tertiary care clinic (Curtin et al, 2005). No difference was found in obesity rates of children with ADHD compared to an age-matched reference population. However, it is possible that these results are influenced by methodological flaws, for example, height and weight in the child’s chart may not have been accurately recorded or up to date measures, reducing the validity of the results. In contrast, there are many studies that support a potential association between childhood ADHD and obesity. A cross-sectional American study, examining the association between ADHD and obesity in children looked at 46,000 participants aged 10-17 from the 2003 National Survey of Children’s Health in the United States (Chen et al, 2010). This is a large sample size which gives the study high statistical power. The obesity rate amongst children with ADHD was 18.9% compared to those without ADHD (12.2%). This study suggests an association between obesity and ADHD in children but does not elicit the direction of the association.

Other studies have investigated the association between childhood ADHD and obesity in adulthood. For example, a cross-sectional study of 6735 participants aged 18-44, from the Psychiatric Epidemiology Survey (Pagoto et al, 2009) found that 29.4% of adults with childhood ADHD were obese, compared to 21.6% of adults without childhood ADHD and so there was a significant association between ADHD and obesity in adults. The association was seen after covarying for social class and depression, which are important confounding factors to consider as they are associated with both ADHD and obesity (Nigg, 2013). A broad age range was used so the study does not elicit whether ADHD affects obesity differently at different stages of adulthood. Furthermore, although these cross-sectional studies are helpful for identifying the association, in order to appropriately tackle this issue, the direction of association needs to be established if possible.

A meta-analysis was undertaken of 43 longitudinal and cross sectional studies, exploring the association between childhood ADHD and obesity in adulthood (Nigg, 2013). A third of the studies did not show a statistically significant association, however the pooled odds ratio showed an increased relative risk of children with ADHD developing obesity for females in young adulthood (over 18), but not in males or children. The results are consistent with a longitudinal study examining the obesity rates in patients with ADHD during their childhood and young adulthood, which found a significant association between childhood ADHD and obesity in young adulthood for females but not for males (Castaneda et al, 2016). The above studies suggest that the association between ADHD and obesity may differ with age and gender.

In contrast, Cortese et al (2016), in a meta-analysis of 42 studies, found that there was a significant increased risk of obesity for participants with ADHD in both childhood and adulthood. The risk was greater in adulthood (70% increased risk compared to 40% increased risk in childhood). The different results could be accounted for by individual methodological differences in the studies included in the meta-analyses. Both meta-analyses included small and unpublished studies as well as retrospective, cross-sectional studies, all of which may not have given reliable results, which could account for the difference in results. It is important to build upon these studies and study the association between ADHD and obesity in both children and in older adulthood, and separately for males and females to elicit any differences.

Although scarce, there have been some longitudinal studies following participants past early adulthood. A longitudinal prospective American study looked at a clinical sample of 655 children, followed up until age 38 (Anderson, 2006). There were losses to follow up resulting in 454 participants. The study looked at the association of ADHD, oppositional defiant disorder and conduct disorder (together assessed as disruptive disorder) and obesity. Social class and medication were covaried for. The study found a significantly higher mean BMI at age 38 for patients with disruptive disorder compared to those without in males and females. However, this does not elicit an independent association between ADHD and obesity. Although the study was longitudinal, a clear direction of association was not found, as patients with disruptive disorder also reported increased BMI in childhood. There may have also been selection bias due to the losses to follow up, potentially reducing the validity of the results.

Another longitudinal prospective study followed up 207 patients for 33 years, finding significantly higher rates of obesity in men with childhood ADHD compared to without (41.4% vs 21.6%, p=0.001), after covarying for social class and mental health disorders (Cortese et al, 2013). There was no significant difference in obesity rates between men with persistent or remitted ADHD, which suggests that children with ADHD have an increased risk of obesity in adulthood, regardless of whether the ADHD persists. This is an important finding for showing a relationship between ADHD and obesity further into adulthood. However, the study may not be representative of the population since it studied caucasian, white males and the sample size was relatively small. It is possible that this association may be different in females at this stage of adulthood. Furthermore, like many of the above studies, a clinical sample was used, which studies participants with more severe cases of ADHD. It is important to discover whether this association affects less severe and even subthreshold cases.

There is relatively little research exploring the specific subtypes of ADHD and obesity in adulthood. It was previously hypothesised that hyperactivity in ADHD would be associated with reduced weight, and may be a protective factor for obesity (Holtkamp et al, 2004). Holtkamp et al (2004) undertook a study using a clinical sample of 97 boys with ADHD and concluded against the original hypothesis that hyperactivity would be a protective factor for obesity, as they found that there were higher levels of obesity in ADHD patients. Furthermore, they found it was the inattentive subtype mediating this association. Castaneda et al (2016) undertook a longitudinal population based study, and also concluded that the inattentive subtype of ADHD was associated with obesity in early adulthood and the hyperactivity/impulsivity subtype was not significantly associated. However, in contrast, it has been thought that the impulsivity component of ADHD may be mediating the association between ADHD and obesity, in terms of leading to disordered eating patterns (Nigg, 2013).

As seen above, most studies have covaried for potential confounding factors such as social class and mental health problems, which have not changed the significance of the association (Cortese et al, 2013). However, there may be other factors involved in the association. Obesity is thought to be heritable, as well as having environmental influences (Cortese and Vincenzi, 2011). Therefore, ideally a genetically sensitive design such as a twin study would show us whether there is a genetic influence to the association between ADHD and obesity. In the absence of this, parental BMI can be used as an indication of familial influences. Whether this affects the association will be important information to gather, as if there is a familial influence for children with ADHD having an increased risk of obesity, then management must take this into consideration.

The primary aim of our study is to investigate the association between childhood ADHD problems and obesity in adulthood (age 42) in both males and females. It is important to test the relationship in both genders separately as previous studies have found differing associations depending on gender (Nigg, 2013). The association between ADHD problems and obesity at age 10 will also be assessed, to elicit whether the association begins in childhood, or later in life, as there has been contrasting evidence (Niggs, 2013, Cortese et al, 2016). The 1970 British Cohort Study has been used, which includes data collected from birth. This data is beneficial as it gives us a large sample size which is representative of the British population, reduces recruitment issues and selection bias often associated with clinical samples, and obtains a near equal sample of males and females which is harder to do with clinical samples. To the best of our knowledge, this will be the first UK study to investigate this association into the fourth decade of life for both genders, using population based data. We hypothesise that there will be a significantly increased risk of obesity for participants with childhood ADHD problems, after adjusting for confounders, for females but not males, based on previous studies (Nigg et al, 2016, Castaneda et al, 2016). The secondary aim of the study is to elicit whether there is a relationship between specific symptoms of ADHD (inattention, or hyperactivity/impulsivity) and obesity in adulthood, which has been found in previous studies in younger adults (Holtkamp et al, 2004). Previous research has confounded for just a few variables such as social class and mental health conditions. Therefore, this study will explore which additional factors may influence the association, and will confound for these appropriately.

Methods

*Design*

This is a prospective, longitudinal study, using data from the 1970 British Cohort Study (Elliot and Shepherd, 2006). Our study looks at the associations between childhood ADHD problems at age 10 (independent variable) and development of obesity in adulthood (measured by BMI>30 at age 42) (main dependent variable) and obesity at age 10. The study looks at the relationship separately for females and males and the association with obesity is secondarily explored for the two subtypes of ADHD (hyperactive/impulsive and inattentive subtype).

*Participants*

The 1970 British Cohort Study collected information for 17 198 cases born in 1970 across Britain (Elliot and Shepherd, 2006). The participants were followed up at ages 5, 10, 16, 26, 30, 34, 38 and 42. Data has been collected on several factors to do with health, education, family and social influences. Our study looks at data at ages 10 and 42. At age 10, there was data for 15,479 participants, however, 625 cases were omitted as they were deceased, leaving 14854 cases. At 42 years, there were 9842 participants. There were 8282 participants with data for both BMI at age 42 and ADHD problems at age 10.

*Materials and Procedure:*

The datasets used in this study were downloaded from the UK data service website (UK Data Service, 2017). Informed consent for interviews and the use of medical records was collected for the 1970 British cohort study (Murray et al, 2010). Ethical approval was not required for our secondary analyses of the existing data. The specific variables chosen to keep from the age 10 dataset were mother’s report of child’s hyperactivity, mother’s report of child’s inattention, child’s height in millimetres, child’s weight in grams, mothers’ report of child’s conduct problems, father’s social class, mother’s height in metres, mother’s weight in kilograms, father’s height in metres, father’s weight in kilograms and from the 42- year dataset, height in metres and weight in kilograms.

*Variables*

**ADHD Variable**

The dataset included the mother’s report on inattention and hyperactivity/impulsivity of the child at 10 years old. This was an appropriate time because ADHD problems are said to be detected before age 7 (Holtkamp et al, 2004). The questionnaires completed by mothers included 6 items from the Rutter A(2) questionnaire (Rutter et al, 1970) and the Conners rating scale (Murray et al, 2010), measured on a visual analogue scale and giving a total score out of 100. The items included were 1) being impulsive or excitable, 2) very restless, hardly ever still, 3) overactive behaviour, 4) failure to finish tasks, 5) inattentive or easily distracted and 6) difficulty concentrating on specific tasks. The questionnaires were such that inattention and hyperactivity/impulsivity could be extracted. The top 15% for each subtype was taken to mean that the child had these problems (for both inattention and hyperactivity/impulsivity independently). The sum of the values for inattention and hyperactivity/impulsivity was computed to create a variable which equates to overall ADHD problems reported by the mother. This was an appropriate measure for ADHD problems because both the Rutter A(2) and Conners rating scales are reliable and valid (Murray et al, 2010) and evidence shows that mothers’ self-reports are a reliable and valid method for assessing ADHD problems (Faraone et al, 1995).

The secondary aim of the study was to assess whether it is the inattentive or hyperactive/impulsive subtype more strongly associated with obesity in adulthood and so variables for inattention only and hyperactivity/impulsivity only were also computed. Of the 1126 participants with a score of inattention, 59.1% had inattention only, and these participants were 8.0% of the total population. Of the 1134 participants with reported hyperactivity/impulsivity, 59.3% had hyperactivity/impulsivity only, and these participants were 8.1% of the total population. 5.6% of the total participants had both inattention and hyperactivity/impulsivity (ADHD problems).

**Body Mass index**

Data was available for height and weight of the participants. At age 10, height and weight was measured by community medical officers, health visitors or school nurses. The participant’s mother and father’s height and weight and the participant’s measures at age 42 were self-reported. Units were converted into metres and kilograms for height and weight respectively and BMI was calculated via weight divided by height squared (Marcus and Wildes, 2009).

**Obesity at age 42 and age 10**

Obesity in adulthood is defined as BMI>30 (Marcus and Wildes, 2009) and so BMI at age 42 was computed into a categorical variable of BMI over 30, where 0 equates to a BMI of under 30, and 1 equates to BMI of over 30.

In children, BMI is interpreted differently, and the BMI-for-age-cut offs were used and the 95th percentile for the specific age group equates to obesity (CDC, 2017). This method is reported to have high sensitivity and specificity (CDC, 2017). For this dataset, obesity equates to BMI>21.36. This cut off was used to create a categorical variable of obesity, where 0 equates to a BMI of under 21.36 and 1 equates to a BMI of over 21.36.

**Social Class**

Father’s social class at age 10 was reported based upon occupation, using the National Statistics Socio-economic Classification (Chandola and Jenkinson, 2000). Social Class was classified from 1 to 6: 1= Professional occupations, 2 = Managerial and technical occupations, 3 =Intermediate skilled occupations non-manual, 4 = Intermediate Skilled occupations, manual, 5 =Partly skilled occupations and 6= Unskilled occupations. This variable was dichotomised into higher and lower social class where higher includes categories 1, 2, 3 and 4, and lower includes categories 5 and 6.

**Conduct Problems**

Conduct problems were reported at age 10 by the participant’s mother, also using the Rutter A2 behaviour rating scale (Rutter et al, 1970). There were 6 items used to detect conduct problems which included: destroys belongings, fights with other children, takes others’ belongings, disobedient, tells lies and bullies (Murray et al, 2010). The conduct problems were then dichotomised into the top 10% compared to the remainder.

*Analysis Plan*

Analysis was conducted using SPSS (IBM SPSS Statistics Version 23). The demographics of the cohort were analysed. ADHD prevalence is higher in males (Gaub and Carlson, 1997) and obesity is more common in females (Kopelman, 2000) and so analysis was performed separately for men and women to elicit any differences, which have been found in previous studies (Castaneda et al, 2016). Firstly, age 10 BMI was compared between children with and without ADHD problems using an independent t test for both males and females separately. Logistic regression was then run to assess the association between ADHD problems and obesity at age 10.

The mean BMI at age 42 was also described, and the differences in mean BMI at age 42 were analysed using an independent t test. The primary aim of the study was to see whether childhood ADHD predisposes people to obesity at age 42. To achieve this, firstly associations were analysed between participants with and without ADHD problems with various covariates, to elicit the potential confounders. For the continuous variables of birth weight, maternal and paternal BMI, independent t tests were calculated to compare the means between participants with and without ADHD problems. These continuous variables were normally distributed and so this is an appropriate test to elicit group differences between children with and without ADHD problems (See appendix A1). Cohen’s d (Rosenthal, 1994) was used to calculate the effect sizes to show the extent of the differences between the mean scores. The Cohen’s d benchmark was used for interpretation of the effect sizes, where a small effect size is d>0.20, moderate effect size is d>0.50 and large effect size is d>0.80 (Durlak, 2009). The associations were also analysed for the categorical variables of lower social class and conduct problems, using logistic regression to calculate odds ratios and 95% confidence intervals. Associations between the covariates and participants with and without obesity at age 42 were also analysed using t tests and odds ratios and 95% confidence intervals. Appropriate confounders in men and women were selected based upon which variables were significantly associated with both childhood ADHD problems and obesity at age 42. Logistic regression was used to assess the association between childhood ADHD problems and obesity at age 42, repeating the regression with the appropriate confounders. Logistic regression was also run for childhood inattention only and obesity at age 42 and hyperactivity/impulsivity only and obesity at age 42, to identify if it is a specific subtype of ADHD responsible for the association, which had been found in previous studies (Castaneda et al, 2016).

*Attrition*

The 8282 participants with data available for both childhood ADHD problems and obesity at age 42 may represent a sample that is not representative of the initial population. Table 1 compares the original data with the analytic sample. Crude comparison shows that in the original sample, there is a predominance of males compared to females (51.8% to 48.2%), however, in the analytic sample there is female predominance (52.2% females compared to 47.8% males). The number of cases of ADHD problems, conduct problems and particularly lower class participants also dropped. Logistic regression confirmed that male gender, ADHD problems, conduct problems and lower social class all increased the likelihood of non-response at age 42 and showed that this was not the case for mean birthweight, maternal or paternal BMI. Despite the 51.8% decrease in total number of participants, the resulting 8282 participants is still a very large sample size, which is important for the generalisability of the results and the statistical power.

Table 1: Comparison of original data with analytic sample (excluding missing data)

|  |  |  |
| --- | --- | --- |
|  | Original Sample | Analytic sample |
| Number of cases | 14854 | 8282 |
| Males | 7695 (51.8%) | 3962 (47.8%) |
| Females | 7159 (48.2%) | 4320 (52.2%) |
| ADHD problems | 851 (6.5%) | 461 (5.6%) |
| Low Family Social Class | 2000 (16.4%) | 1074 (14.4%) |
| Conduct Problems | 1311 (9.9%) | 700 (8.5%) |
| Mean birthweight | 3307.80 | 3314.4 |
| Mean Maternal BMI | 23.45 | 23.38 |
| Mean Paternal BMI | 24.49 | 24.43 |
|  |  |  |

Results

Of the 8282 participants with available data for ADHD problems and BMI at age 42, 52.2% were female and 47.8% were males. 461 participants had childhood ADHD problems (5.6%); 186 of whom were female (40.3%) and 275 were male (59.7%). 1134 participants had reported hyperactivity/impulsivity (13.7%) and 1126 participants had reported inattention (13.6%). 673 (8.1%) reported hyperactivity/impulsivity only and 665 (8.0%) had inattention only.

The range of BMI at age 10 was 10.20 to 30.86 and the mean BMI was 16.88, which using the BMI-for-age-Cutoffs, is a healthy weight. 4.5% (n=374) of the population were obese at age 10. For males without ADHD problems, the mean BMI at age 10 was 16.76 (SD=1.94) compared to 16.59 (SD=1.69) for males with ADHD problems (t(3524)=1.36, p=0.175, d=0.09). For females without ADHD problems, the mean BMI at age 10 was 17.01 (SD=2.24) for participants without ADHD problems and 17.22 (SD=2.39) for participants with ADHD problems. t(3868)=-1.19, p=0.234, d=0.09. The t tests for both males and females show no significant difference in BMI at age 10 between children with and without ADHD problems.

Logistics regression was run to show whether there was a significant association between ADHD problems and obesity at age 10. For both genders, OR=0.87 (0.54, 1.41), p=0.570, for males OR=0.55 (0.22, 1.35), p=0.191, and for females OR=1.30, (0.72. 2.22), p=0.384, so there was no significant association between ADHD problems and obesity at age 10.



**Figure 1: Histogram of BMI across the population**

Figure 1 shows the distribution of BMI at age 42 across the population and the red line represents a BMI>30 (obesity). The mean BMI across the population was 26.85 which is overweight and 19.5% (n=1613) of this population were obese (BMI>30). For males without childhood ADHD problems, the mean BMI at age 42 was 27.41 (SD=4.58), compared to 27.96 (SD=4.83) for males with ADHD problems. t(3675)=-1.87, p=0.06, d=0.12, showing no significant difference in mean BMI between males with and without childhood ADHD problems. For females without childhood ADHD problems, the mean BMI at age 42 was 26.21 (SD=5.74), compared to 27.81 (SD=5.75) for those with childhood ADHD problems. The results of the t test showed a significant difference in mean BMI between females with and without childhood ADHD problems, with a small effect size, t(3778)=-3.40, p=0.001, d=0.28.

**Childhood covariates and childhood ADHD problems**

Table 2 shows the associations of different covariates with childhood ADHD problems. An independent t test was used to compare the means for the birthweight, maternal and paternal BMI of children with and without childhood ADHD problems. Effect sizes were also calculated to show the group differences. The table shows that males with ADHD problems had significantly lower birthweights and lower family social class and were much more likely to have conduct problems (OR=7.51 (5.74, 9.83, p=0.000) compared to males without childhood ADHD problems. The difference between birthweights of males with and without ADHD problems was significant but a small effect size (d=0.19). There was no significant difference in maternal or paternal BMI.

Females with ADHD problems did not show a significant difference in birthweight, maternal, or paternal BMI compared to participants without ADHD problems. Females with ADHD problems were of significantly lower family social class and much more likely to have conduct problems. This was again a large odds ratio (10.78 (7.73, 15.75, p=0.000). For both lower social class and conduct problems, the odds ratio is larger for females than males, however there is overlapping of the confidence intervals, suggesting that the association with ADHD problems and these two factors is similar for males and females.

**Table 2: Childhood ADHD: associations with childhood covariates**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | Males (n=3692) | | | | |
|  | ADHD Problems | | T test | Cohen’s d | |
|  | No  (n=3687) | Yes  (n=275) |  |  | |
| Covariates |  |  |  |  | |
| Mean Birthweight | 3381.64 SD=523.59 | 3281.81 SD=548.36 | t(df=3666)=2.90, p=0.004 | 0.19 | |
| Mean Maternal BMI | 23.34 SD=3.74 | 23.50 SD=3.85 | t(df=3798)=-0.66, p=0.51 | 0.04 | |
| Mean Paternal BMI | 24.41 SD=2.97 | 24.53 SD=3.02 | t(df=3615)=-0.57, p=0.57 | 0.04 | |
|  |  |  | OR (95% CI) | |  |
|  |  |  |  | |  |
| % Low family social class | 13.3 | 19.8 | 1.61 (1.16, 2.23), p=0.005 | |  |
| % Conduct problems | 8.7 | 19.8 | 7.51 (5.74, 9.83), p=0.000 | |  |

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Females (n=4320) | | | |
|  | ADHD problems | | T test | Cohen’s d |
|  | No  (n=4134) | Yes  (n=186) |  |  |
| Covariates |  |  |  |  |
| Mean Birthweight | 3261.69 SD= 493.89 | 3204.59 SD= 571.14 | t(4022)=1.45, p=0.148 | 0.11 |
| Maternal BMI | 23.40 SD=3.85 | 23.43 SD=3.70 | t(4165)=-0.08, p=0.93 | 0.008 |
| Paternal BMI | 24.44 SD=2.96 | 24.45 SD=3.09 | t(3951)=-0.05, p=0.96 | 0.003 |
|  |  |  | OR (95% CI) |  |
| % Low family social class | 14.6 | 24.2 | 1.87 (1.29, 2.70), p=0.001 |  |
| % Conduct problems | 5.1 | 36.5 | 10.78 (7.73, 15.05),  p=0.000 |  |

**Childhood covariates and adult obesity**

Associations between the covariates and obesity at age 42 were then analysed, as seen in Table 3. Table 3 shows that male and female participants who were obese at age 42 had significantly higher maternal and paternal BMI. There was also a significantly higher rate of lower family social class amongst participants who were obese at age 42. However, in females, there was a significantly higher rate of childhood conduct problems amongst participants with obesity at age 42, whereas this was not significant for males.

**Table 3: Obesity age 42: associations with childhood ADHD problems and childhood covariates**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Males (n=3692) | | | |
|  |  | | | |
| Obesity | | T test | Cohen’s d (effect size) |
|  |  | |  |  |
| No  (n=3113) | Yes  (n=849) |
| Covariates |  |  |  |  |
| Mean Birthweight | 3366.83 SD=529.16 | 3403.82 SD=512.92 | t(df=3666)=-1.76, p=0.079 | 0.08 |
| Mean Maternal BMI | 22.94 SD=3.35 | 24.89 SD=4.64 | t(df=3798)=-13.49, p=0.000 | 0.48 |
| Mean Paternal BMI | 24.18 SD=2.81 | 25.29 SD=3.37 | t(df=3615)=-9.33, p=0.000 | 0.36 |
|  |  |  |  |  |
|  |  |  | OR (95% CI) |  |
|  |  |  |  |  |
| % Low family social class | 13.1 | 16.4 | 1.31 (1.05, 1.63), p=0.018 |  |
| % Conduct problems | 10.5 | 12.5 | 1.22 (0.97, 1.55), p=0.095 |  |

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | | Females (n=4320) | | | | |
|  | |  | | | | |
| Obesity | | | T test | Cohen’s d (effect size) |
|  | |  | | |  |  |
| No  (n=3556) | | Yes  (n=764) |
| Covariates | |  | |  |  |  |
| Mean Birthweight | | 3260.52 SD=494.10 | | 3253.76 SD=512.8 | t(df=4022)=0.33, p=0.743 | 0.01 |
| Mean Maternal BMI | | 23.06 SD=3.57 | | 24.98 SD=4.61 | t(df=4165)=-12.48, p=0.000 | 0.47 |
| Mean Paternal BMI | | 24.23 SD=2.86 | | 25.43 SD=3.24 | t(df=3951)=-9.81, p=0.000 | 0.39 |
|  |  | |  |  |  |
|  | | |  |  | OR (95% CI) | |
|  | |  | |  |  | |
| % Low Family Social Class | | 14.0 | | 19.7 | 1.50 (1.21, 1.86), p=0.000 | |
| % Conduct Problems | | 6.0 | | 8.4 | 1.44(1.07, 1.93), p=0.015 | |

These associations with both ADHD problems and obesity demonstrate which variables could be potential confounders in the association between childhood ADHD problems and obesity. For males, consistently with previous literature (Nigg, 2016) social class is the only covariate associated with both ADHD problems and obesity and therefore will be included in the logistics regression as a confounding factor as seen in Table 4.

**Table 4: Associations between childhood ADHD problems and age 42 obesity: unadjusted and adjusted for confounding factors in males.**

|  |
| --- |
| ADHD problems |
|  | Male  (OR 95% CI) |
|  |  |
| 1. Unadjusted model | 1.53 (1.16, 2.01), p=0.002 |
| 2. Adjusted for family social class | 1.40 (1.04, 1.87), p=0.026 |

Table 4 shows that there is a significant association between childhood ADHD problems and obesity at age 42, even after adjusting for social class. Adjusting for social class slightly attenuates the association, however the association is still statistically significant (OR=1.40 (1.04, 1.87), p<0.05 (p=0.026)).

Tables 2 and 3 show that for females, social class and conduct problems are associated with both ADHD problems and obesity and so will be included in the logistics regression described in table 5.

**Table 5: Associations between childhood ADHD problems and age 42 obesity: unadjusted and adjusted for confounding factors in females.**

|  |
| --- |
| ADHD problems |
|  | Female  (OR 95% CI) |
|  |  |
| 1. Unadjusted model | 1.56 (1.11, 2.20), p=0.011 |
| 2. Adjusted for family social class | 1.49 (1.03, 2.14), p=0.034 |
| 3. Adjusted for age 10 conduct problems | 1.35 (0.93, 1.95), p=0.113 |
| 4. Adjusted for all significant covariates | 1.33 (0.90, 1.97), p=0.150 |

Table 5 shows a significant association between childhood ADHD and obesity at age 42 in females before adjusting for confounding factors. Social class slightly attenuates the affect but it is still significant. Conduct problems attenuate the association to the extent that it is no longer significant (OR=1.35 (0.93, 1.95, p=0.113) and this is enhanced by covarying for both social class and conduct problems. This shows that there is no independent effect of childhood ADHD problems on obesity at age 42 in females and the association is influenced by childhood conduct problems.

As shown in tables 2 and 3, the familial variables of maternal BMI and paternal BMI in both genders are associated with obesity at age 42, but not with ADHD problems. They therefore were not considered as official confounding factors. However, their strong association with obesity creates the question of whether they contribute to the association between childhood ADHD problems and obesity. Logistics regression was run adjusting for these covariates, as seen in table 6.

**Table 6: Associations between ADHD and obesity at age 42, adjusting for familial covariates**

|  |  |  |
| --- | --- | --- |
| ADHD problems | Obesity Age 42 years | |
|  | Male  (OR 95% CI) | Female  (OR 95% CI) |
|  |  |  |
| 1. Unadjusted model | 1.53 (1.16, 2.01), p=0.002 | 1.56 (1.11, 2.20), p=0.011 |
|  |  |  |
| 2a. Adjusted for maternal BMI | 1.47 (1.10, 1.96), p=0.009 | 1.74 (1.22, 2.48), p=0.002 |
| 2b. Adjusted for paternal BMI | 1.46 (1.10,1.95), p=0.010 | 1.61 (1.12, 2.30), p=0.009 |
| 3. Adjusted all above covariates | 1.46 (1.08, 1.98), p=0.015 | 1.63 (1.12, 2.38), p=0.011 |

Table 6 shows that for males and females, the association between ADHD problems and obesity is significant, regardless of maternal BMI or paternal BMI. There is strong overlap of the confidence intervals between the unadjusted and fully adjusted model (model 3), showing that the familial variables do not explain the association between childhood ADHD and obesity.

The secondary aim of the study was to identify whether the inattentive or hyperactive/ impulsive component of ADHD contributes to the association with obesity. Logistic regression between inattention only and hyperactivity only and obesity at age 42 was done for the whole cohort (men and women together) as seen in Table 7. In this table, all factors which may potentially contribute to the association were included individually and variables which themselves were significantly associated were then included in the model adjusting for all significant covariates.

**Table 7: Associations between Inattention only and Hyperactivity/Impulsivity only and age 42 obesity: unadjusted and adjusted for covariates.**

|  |  |  |
| --- | --- | --- |
| ADHD problems | Obesity Age 42 years | |
|  | Inattention  (OR 95% CI) | Hyperactivity/Impulsivity  (OR 95% CI) |
|  |  |  |
| 1. Unadjusted model | 1.41 (1.17, 1.69), p=0.000 | 1.09 (0.90, 1.33), p=0.365 |
| 2a. Adjusted for birthweight | 1.50 (1.24, 1.81), p=0.000 | 1.12 (0.91, 1.37), p=0.290 |
| 2b. Adjusted for maternal BMI | 1.41 (1.17, 1.71), p=0.000 | 0.99 (0.81, 1.22), p=0.925 |
| 2c. Adjusted for paternal BMI | 1.40 (1.15,1.71), p=0.001 | 1.01 (0.87, 1.33), p=0.491 |
| 2d. Adjusted for parental obesity | 1.43 (1.18, 1.75), p=0.000 | 1.05 (0.85, 1.29), p=0.676 |
| 2e. Adjusted for family social class | 1.42 (1.17, 1.73), p=0.000 | 1.05 (0.85, 1.29), p=0.659 |
| 2f. Adjusted for age 10 conduct problems | 1.38(1.15, 1.67), p=0.001 | 1.08 (0.89, 1.32), p=0.445 |
| 3. Adjusted for all significant covariates | 1.36 (1.10, 1.68), p=0.005 | 0.95 (0.75, 1.19), p=0.646 |

For childhood inattention only, all covariates apart from birthweight were themselves significant (birthweight OR=1.01 (0.99, 1.03) p=0.23), therefore, model 3 includes all other covariates. Childhood Inattention only was significantly associated with obesity at age 42 despite adjusting for covariates (OR=1.36 (1.10, 1.68) p=0.005). The relative risk did not reduce dramatically from the unadjusted model (OR=1.41(1.17, 1.69), p=0.000). For childhood hyperactivity/impulsivity only, the unadjusted model did not show a significant association between childhood hyperactivity/impulsivity and obesity at age 42.

Discussion

This study has found no association in males or females between ADHD problems and obesity at age 10. In males and females, there was an increased risk of developing obesity at age 42 with childhood ADHD problems, (before adjusting for confounders), which suggests that the association manifests not in childhood but in later life. In contrast to the primary hypothesis, our study found a significant independent association between ADHD problems at age 10 and adulthood obesity at age 42 in males but not in females, after adjusting for confounders. These findings contrast to previous research (Castaneda et al, 2016, Nigg, 2016) which found an association in females but not males. The study by Castaneda et al (2016) only followed patients into early adulthood and it is possible that the association changes later in adulthood, explaining the difference with our results. Our study furthers the knowledge around the association between childhood ADHD and obesity, as it is the first study to investigate the association between childhood ADHD problems and obesity as late into adulthood for both males and females. Our study shows us that childhood ADHD can increase the risk of obesity into the fourth decade of life in males, but for females there is not an independent association.

For females, there was a significant association between childhood ADHD and obesity in the unadjusted model and after adjusting for social class. It was adjusting for childhood conduct problems which critically attenuated the association. It is well established that there is a strong comorbidity between ADHD and conduct disorder (Nigg, 2013) and our initial analysis shows that there was a strong association between childhood ADHD and childhood conduct problems in this cohort (for females OR=10.78, (7.73,15.05)). Therefore, although the results do not show an independent association between childhood ADHD and adulthood obesity, the results suggest that in females, the group of children that are at high risk of adulthood obesity are children with ADHD or conduct problems, or indeed both. In males, conduct problems were not found to be associated with obesity, and an independent association was found between ADHD and obesity at age 42, suggesting the high-risk group in males is children with ADHD, regardless of conduct problems.

Many previous studies investigating the association of ADHD with obesity have either included conduct disorder and/or ADHD as the independent variable, or excluded participants with conduct disorder. It has been shown that there are common genetic and environmental factors underpinning the aetiology of the two conditions (Thapar et al, 2001). A strength of the study was being able to covary for conduct problems as it was important to establish whether the association between ADHD problems and obesity is unique to ADHD or common to both disorders, which may suggest the underlying causal factor is shared by the two disorders. The fact that the association between conduct problems and obesity was found in females but not males, and an independent association between ADHD and obesity was found in males but not females, suggests that there may be different causal mechanisms for the association between genders. In males, the association may be due to a factor not shared with conduct disorder, however in females it may be due to a shared factor.

The secondary aim of the study was to assess whether it is the inattentive or hyperactive/impulsive component of ADHD that is more strongly associated with obesity at age 42. Results showed that childhood inattention only was significantly associated with adulthood obesity, even after adjusting for multiple covariates. However, childhood hyperactivity/impulsivity only was not significantly associated, even before adjusting for covariates. These findings are consistent with previous research from clinical populations, for example Holtkamp et al (2004), who found the inattentive subtype of ADHD was linked to obesity in adulthood.

*Strengths*

The study used a large population based sample in the UK, which makes it generalisable to this population. This is a strength since many previous studies were conducted in the USA (Cortese et al, 2013, Anderson, 2006). A limitation of clinical samples is that often the sample contains more severe cases of ADHD; this population based study will include cases of ADHD of varying severities, improving the generalisability of the results. Furthermore, many previous studies in this area have been cross-sectional and the longitudinal nature of this study allows us to see the direction of association more clearly. Most previous longitudinal studies have investigated the association between childhood ADHD and obesity in childhood, adolescence or early adulthood (Castaneda et al, 2016, Nigg, 2016). Other studies following participants later into adulthood have been mainly clinical samples and of males (Cortese et al, 2013). To the best of our knowledge this is the first longitudinal population based study investigating the association of childhood ADHD problems into the fourth decade of life for both males and females.

A strength of using the 1970’s British Cohort study was that there was data available which allowed calculation of maternal and paternal BMI, which is rarely available when carrying out clinical samples. Both maternal and paternal BMI had significant association with obesity in males and females and so the ability to covary for these two factors was another strength of the study. Heritability of obesity is well established (Cortese and Vincenzi, 2011) and so it was useful to use these variables to represent the familial influence of obesity. The results showed that familial influence did not explain the association between ADHD problems and obesity, which is important to know in terms of further research into the causal mechanisms of the association. The fact that there seems to be little familial influence, and that there was no association between ADHD problems and obesity at age 10, suggests that the causal mechanism is something that occurs or manifests later in life, which is important to consider when trying to identify this.

*Limitations*

A limitation, as previously mentioned, was that of attrition. It was confirmed through logistic regression that there were statistically significant associations between the male gender, childhood ADHD problems, conduct problems and lower social class and non-response at age 42. As shown in Table 1, the percentage of children with ADHD problems fell from 6.5% to 5.6%, which is still within the normal range of estimated ADHD prevalence (Nigg, 2013). However, it is possible that having a smaller number of participants with childhood ADHD could make an association with obesity at age 42 harder to detect, leading to underestimation of our results.

Attrition may have been an issue for social class, since for both males and females, this attenuated the association of childhood ADHD problems and obesity at age 42 to the point where it was still significant, but only just (for males, OR= 1.40 (1.04, 1.87) p=0.026 and for females, OR=1.49 (1.03, 2.14) p=0.034); the lower confidence interval is close to 1 in both cases. It is possible that if the analytic sample contained the same distribution of lower social class participants as the initial sample, this may have further attenuated the results. Nevertheless, a benefit of our large cohort was that the remaining sample size was still large, which reduces the effect of non-response on the validity and reliability of the findings.

Another limitation is that mothers’ self-reports for inattention and hyperactivity/impulsivity were used and so a clinical diagnosis of ADHD could not be considered. It could be argued that mothers’ self-reports may be unreliable and subjective as it may be difficult for a mother to detect or admit these symptoms which could lead to an underestimation. However, the percentages of children with ADHD problems (5.57%) is similar to that reported by other studies using a clinical diagnosis (Nigg, 2013) and self-reports were based on the Rutter and Conners questionnaire, which are both reliable and validated (Murray et al, 2010). Furthermore, evidence shows that mothers’ self-report of ADHD problems are both reliable and valid (Faraone et al, 1995). Using mothers’ self-report could be considered a strength as this may have detected subthreshold cases, which have been found to be at risk of some adverse outcomes in adulthood (Thapar et al, 2012). It could also be argued that both height and weight may not be reliable measures as they are self-reported. These were used to compute the BMI variables and it is again possible that these values were underreported or inaccurate, especially since participants may not have all had access to scales. However, studies have shown that self-reported height and weight are mostly accurate (Stewart, 1982) so the self-reported variables should not have affected the reliability and validity of the results.

It was previously thought that ADHD may reduce the risk of obesity, due to hyperactivity causing expenditure of energy (Holtkamp et al, 2004). It was then thought that, for the many children who did not retain hyperactivity into adulthood, the reduction in energy expenditure could lead to weight gain in adulthood. However, as found in previous studies and as hypothesised, it was the inattention only group that showed a significant association with obesity at age 42. The children with hyperactivity/impulsivity only did not. Therefore, our results suggest it is the inattentive component of ADHD that is associated with obesity at age 42, which is important to know in terms of targeting those at risk.

There are many possible pathophysiological reasons for the association. Firstly, there could be a common gene mutation which causes the presentation of both ADHD and obesity, manifesting in different periods of life. Cortese and Vincenzi, (2011), described the genetic mutation may lead to a common neurobiological dysfunction in the dopaminergic system, in which there is reduced dopamine-related reward, which is thought to be involved with both ADHD and obesity. Secondly, the inattention in childhood ADHD may predispose people to unhealthy lifestyle behaviours such as poor diet, poor exercise and increased alcohol consumption, which could increase the risk of obesity. In terms of diet, inattention may make someone less likely to sustain a regular eating pattern, leading to dysfunctional eating habits such as binge eating. On a neurological level, inattention may reduce sensitivity to internal satiation cues (Nigg, 2016), leading to excessive food intake. Thirdly, some of the children with ADHD problems may have been taking a stimulant medication (methylphenidate), which is known to suppress appetite. It is uncommon for patients to be taking this in adulthood, therefore, once appetite is no longer suppressed, these participants may eat excessively, causing weight gain. Finally, there has been evidence that both childhood ADHD and obesity are associated with depression in adulthood (Stuart-Smith, 2017). Although depression is typically associated with weight loss, some patients experience excessive eating with depression (Rofey et al, 2009) and so depression could be influencing the association between childhood ADHD and obesity. In future research, it will be important to covary for medication such as methylphenidate and depression.

*Implications*

Our study has been important in clarifying an increased risk of obesity in adulthood (into the fourth decade) for children with ADHD. To extend this study, similar studies could be done with different population based cohorts, such as beginning data collection in a different year or using data from different countries. The average BMI of this cohort at age 42 was 26.85 which is overweight and so it is possible that this cohort was more predisposed to obesity than other cohorts. Repeating the study with a different cohort would show whether our results are reliable, or whether it was something about this cohort which made the association easier to find.

Further research should be done to identify the mechanisms involved in the association between ADHD problems and obesity, to target these specific causal factors. This should be done separately in males and females because, as previously mentioned, gender may affect the causal mechanism. ADHD is a common disorder in children and so our study identifies an at-risk group which will include a significant proportion of the population. It is important to identify this group to think about early-prevention and care. It may be possible to increase education about the importance of regular eating patterns and exercise amongst this group, in schools, to parents and in a clinical psychiatric setting, when the child is first diagnosed and in their subsequent care. It could be possible to take a multidisciplinary approach and involve dieticians to monitor and help create appropriate eating patterns to reduce the likelihood of developing adulthood obesity.

Conclusion

Although no significant association was found between childhood ADHD problems and obesity at age 10, a significant association between childhood ADHD problems and obesity in adults at age 42 was found in males. In females, the association between ADHD problems and obesity was attenuated by conduct problems, suggesting females with conduct problems and ADHD are a high-risk group for obesity. Our study suggests it is the inattentive subtype of ADHD involved in the association with obesity, not hyperactivity/impulsivity and there are many potential causes for this which should be further researched. Since obesity is an increasing problem in the UK population, identifying children with ADHD problems as a high-risk group can be useful for early education and prevention.

References

Allender, S. and Rayner, M. (2007) “The Burden of Overweight and Obesity-Related Ill Health in the UK”, *Obesity Reviews,* 8(5), pp. 467-473.

American Psychiatric Association. (2013). “Diagnostic and Statistical Manual of Mental Disorders”, (5th ed.).

Anderson, S., Cohen, P., Naumova, E. and Must, A. (2006) “Relationship of Childhood Behaviour Disorders to Weight Gain from Childhood into Adulthood”, *Ambulatory Pediatrics,* 6(5), pp.297-301.

Castaneda, R., Kumar, S., Voigt, R., Leibson, C., Barbaresia, W., Weaver, A., Killian, J. and Katusic, S. (2016) “Childhood Attention-Deficit/Hyperactivity Disorder, Sex, and Obesity: A Longitudinal Population-Based Study”, *Mayo Clinic,* 91(3), pp. 352-361.

"CDC - Recommended BMI-For-Age Cutoffs - BMI For Age Training Course - DNPAO". *Cdc.gov*. N.p., 2017. Web. 16 Mar. 2017.

Chandola, T. and Jenkinson, C. (2000) “The New UK National Statistics Socio-Economic Classification (NS-SEC); Investigating Social Class Differences in Self-Reported Health Status”, *Journal of Public Health Medicine,* 22(2), pp. 182-190.

Chen, A., Kim, S., Houtrow, A., and Newacheck, P. (2010) “Prevalence of Obesity Among Children with Chronic Conditions,” *Obesity: A Research Journal,* 18(1), pp. 210-213

Cortese, S and Vincenzi, B (2011) “Obesity and ADHD: Clinical and Neurobiological Implications”, *Behavioural Neuroscience of Attention Deficit Hyperactivity Disorder and its Treatment,* 9, pp. 199-218.

Cortese, S., Olazagasti, M.A.R., Klein, R., Castellanos, F.X., Proal, E. and Mannuzza, S. (2013) “Obesity in Men with Childhood ADHD: A 33-Year Controlled, Prospective, Follow-up Study”, *Pediatrics,* 131(6), pp.1731-1738.

Cortese, S., Moreira-Maia, C.R., Fleur, D.S., Morcillo-Penalver, C., Rohde, L.A. and Faraone, S.V. (2016) “Association Between ADHD and Obesity: A Systematic Review and Meta-Analysis”, The *American Journal of Psychiatry,* 173(1), pp. 34-43.

Curtin, C., Bandini, L.G., Perrin, E.C., Tybor, D.J. and Must, A. (2005) “Prevalence of Overweight in Children and Adolescents with Attention Deficit Hyperactivity Disorder and Autism Spectrum Disorders: A Chart Review”, *BMC Pediatrics,* 5(48).

Durlak, J. (2009) “How to Select, Calculate and Interpret Effect Sizes “, *Journal of Pediatric Psychology,* 34(9), pp. 917-928.

Elliot, J. and Shepherd. P. (2006) “Cohort Profile: 1970 British Birth Cohort (BSC70)”, *International Journal of Epidemiology,* 35(4), pp. 836-843.

Erskine, H., Norman, R., Ferrari, A., Chan, G., Copeland, w., Whiteford, H. and Scott, J. (2016) “Long-Term Outcomes of Attention-Deficit/ Hyperactivity Disorder and Conduct Disorder: A Systematic Review and Meta-Analysis”, *Journal of the American Academy of Child and Adolescent Psychiatry,* 55(10), pp. 841-850.

Faraone, S., Biederman, J. and Milberger, S. (1995) “ How Reliable Are Maternal Reports of Their Children’s Psychopathology? One-Year Recall of Psychiatric Diagnoses of ADHD Children”, *Journal of the American Academy of Child and Adolescent Psychiatry,* 34(8), pp. 1001-1008.

Fuemmeler, BB., Ostbye, T., Yang, C., McClernon, F. and Kollins, S. (2011) “Association between Attention-Deficit/ Hyperactivity Disorder (ADHD) Symptoms and Obesity and Hypertension in Adulthood: A Population-based Study”, *International Journal of Obesity,* 35(6), pp. 852-862.

Gaub, M. and Carlson, C. (1997) “Gender Differences in ADHD: A Meta-Analysis and Critical Review”, *Journal of the American Academy of Child and Adolescent Psychiatry,* 36(8), pp. 1036-1045.

Holtkamp, K., Konrad, K., Muller, B., Heussen, N., Herpetz, S., Herpetz-Dahlmann, B. and Hebebrand, J. (2004) “Overweight and Obesity in Children with Attention Deficit/ Hyperactivity Disorder”, *International Journal of Obesity,* 28, pp. 685-689.

Klein, R., Mannuzza, S., Olazagasti, M., Rozien, E., Hutchinson, J., Lashua, E. and Castellanos, X. (2012) “Clinical and Functional Outcome of Childhood Attention-Deficit/Hyperactivity Disorder 33 Years Later”, *Archives of General Psychiatry,* 69(12), pp. 1295-1303.

Kopelman, P.G. (2000) “Obesity as a Medical Problem”, *Nature,* 404, pp. 635-643.

Mansournia, M.A. (2016) “Inverse Probability Weighting”, *The British Medical Journal,* 352(189).

Marcus, M. and Wildes, J (2009) “Obesity: Is it a mental disorder?”, *International Journal of Eating Disorders,* 42(8), pp. 739-753.

Murray, J., Irving, B., Farrington, D.P., Colman, I. and Bloxson, C.A.J. (2010) “Very Early Predictors of Conduct Problems and Crime: Results from a National Cohort Study”, *The Journal of Child Psychology and Psychiatry,* 51(11), pp. 1198-1207.

Must, A., Spadano, J. and Coakley, E. (1999) “The Disease Burden Associated with Overweight and Obesity”. *JAMA,* 282(16), pp. 1523-1529.

Nigg, J. (2013) “Attention-Deficit/ Hyperactivity Disorder and Adverse Health Outcomes”, *Clinical Psychology Review,* 33(2), pp. 215-228.

Nigg, J., Johnstone, J.M., Musser, E.D., Long, H.G., Willoughby, M.T. and Shannon, j. (2016) “Attention-deficit/hyperactivity disorder (ADHD) and being overweight/obesity: New data and meta-analysis”, *Clinical Psychology Review,* 43, pp. 67-69.

“Non-Communicable Diseases”. *World Health Organization.* N.p., 2015. Web. 15 Mar. 2017.

Pagoto, S. L., Curtin, C., Lemon, S. C., Bandini, L. G., Schneider, K. L., Bodenlos, J. S. and Ma, Y. (2009), Association Between Adult Attention Deficit/Hyperactivity Disorder and Obesity in the US Population. *Obesity*, 17: 539–544.

Public Health England. (2017) *UK and Ireland prevalence and trends: Public health England obesity knowledge and intelligence team.* Available at:

<https://www.noo.org.uk/NOO_about_obesity/adult_obesity/UK_prevalence_and_trends> (Accessed: 7 January 2017)

Rofey, D.L., Kolko, R.P., Iosif, A., Silk, J., Bost, J., Feng, W., Szigethy, E.M., Noll, R.B., Ryan, N.D. and Dahl, R. (2009) “A Longitudinal Study of Childhood Depression and Anxiety in Relation to Weight Gain”, *Child Psychiatry and Human Development,* 40 (4), PP. 517-526.

Rosenthal, R. (1994) “Parametric Measures of Effect Size”,1ST ed. New York: *Russell Sage Foundation.*

Rutter, M., Tizard, J. and Whitmore, K. (1970) “Education, Health and Behaviour”, *Longman Publishing Group.*

Stewart, A. (1982) “The Reliability and Validity of Self-Reported Weight and Height”, *Journal of Chronic Diseases,* 35(4), pp. 295-309.

Thapar, A., Harrington, R., McGuffin, P. (2001) “Examining the Comorbidity of ADHD-Related Behaviours and Conduct Problems using a Twin Study Design”, *The British Journal of Psychiatry,* 179(3), pp. 224-229.

Thapar, A., Cooper, M., Jefferies, R. and Stergiakouli, E. (2012) “What Causes Attention Deficit Hyperactivity Disorder”, *Archives of Disease in Childhood,* 97, pp. 260-265.

"UK Data Service". *Ukdataservice.ac.uk*. N.p., 2017. Web. 16 Mar. 2017.

Appendix

**Appendix A1 – Distributions of continuous variables**









**Appendix A2: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Social class in males**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .334 .150 4.985 1 .026 1.397 1.042 1.873

socialclass2 .255 .113 5.088 1 .024 1.290 1.034 1.609

Constant -1.375 .046 896.369 1 .000 .253

a Variable(s) entered on step 1: MUMADHD, socialclass2.

**Appendix A3: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Social class in females**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .395 .186 4.508 1 .034 1.485 1.031 2.138

socialclass2 .393 .109 13.072 1 .000 1.482 1.197 1.834

Constant -1.604 .047 1142.560 1 .000 .201

a Variable(s) entered on step 1: MUMADHD, socialclass2.

**Appendix A4: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Conduct disorder in females**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .297 .187 2.514 1 .113 1.346 .932 1.944

MCOND10 .297 .156 3.605 1 .058 1.345 .990 1.827

Constant -1.577 .042 1388.253 1 .000 .207

a Variable(s) entered on step 1: MUMADHD, MCOND10.

**Appendix A5: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Maternal BMI in males**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .380 .147 6.704 1 .010 1.463 1.097 1.951

mBMI .126 .010 149.771 1 .000 1.134 1.111 1.157

Constant -4.326 .251 295.94 1 .000 .013

a Variable(s) entered on step 1: MUMADHD, mBMI.

**Appendix A6: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Maternal BMI in females**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .474 .182 6.739 1 .009 1.606 1.123 2.296

mBMI .112 .010 133.761 1 .000 1.119 1.098 1.140

Constant -4.243 .240 312.877 1 .000 .014

a Variable(s) entered on step 1: MUMADHD, mBMI.

**Appendix A7: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Paternal BMI in males**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .397 .151 6.968 1 .008 1.488 1.108 1.998

fBMI .119 .013 81.031 1 .000 1.126 1.098 1.156

Constant -4.267 .333 164.176 1 .000 .014

a Variable(s) entered on step 1: MUMADHD, fBMI.

**Appendix A8: Logistics regression of childhood ADHD and obesity at age 42, showing covariate scores: Paternal BMI in females**

Variables in the Equation

B S.E. Wald df Sig. Exp(B) 95% C.I.for EXP(B)

Lower Upper

MUMADHD .458 .188 5.956 1 .015 1.581 1.094 2.284

fBMI .128 .014 89.420 1 .000 1.137 1.107 1.167

Constant -4.751 .343 191.466 1 .000 .009

a Variable(s) entered on step 1: MUMADHD, fBMI.