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Prevalence and adult outcomes of Attention-Deficit/Hyperactivity Disorder

Evidence from a 30-year prospective longitudinal study

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The Bedford Group for Lifecourse and Statistical Studies (BG) is based in the Institute of Education, University of London. BG members share interests in the creation and analysis of longitudinal and multilevel data, which are applied to a wide range of topics in the education, social and health sciences. Further information on BG is available at www.ioe.ac.uk/bedfordgroup.

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Summary

1. Using data on over 10,000 individuals from the 1970 British Cohort Study (Butler et al, 1986), the aims of this study were to provide an estimate of the prevalence of childhood AD/HD in a birth cohort (born 5-11 April 1970), and to explore the age-30 outcomes of those with childhood AD/HD.

2. Items from the Conners rating scale (Conners, 1969), completed by parents and teachers, and the parental Rutter questionnaire (Rutter et al, 1970), were used to identify a group of cohort members with AD/HD symptoms at age 10.

3. Standard multiple regression analyses using probit specifications were used to ascertain the effect of age-10 AD/HD on twenty-four outcomes measured at age 30, whilst controlling for a large number of the cohort member's socio-economic, personal and familial characteristics measured at birth and age 5.

4. Results showed that there was a prevalence of AD/HD of 7.4%, in the upper end of the range usually reported. Although boys with AD/HD symptoms at age 10 outnumber girls, the ratio found was lower than much previous research has indicated, at 1.7:1.

5. Men and women with childhood AD/HD were significantly more likely than their unaffected counterparts to face a wide range of negative outcomes at age 30, spanning domains of education, economic status, housing, relationships, crime and health. These findings held true even when controlling for a number of background personal, familial, social and economic characteristics. Men tended to fare worse than women.

6. We conclude that AD/HD is prevalent in both male and female children and adults. The adult lives of both men and women with childhood AD/HD are typified by social deprivation and adversity.

7. Our material points the way to the need for better screening for AD/HD, perhaps in primary care, and for the wider use of sensitively designed early interventions and individually tailored treatment plans, offering both pharmacological and psychosocial elements. Ongoing treatment and support for those with AD/HD through their adolescence and into their adulthood, and raised awareness of AD/HD amongst parents, health professionals, educators and those in the criminal justice system, will help ease the negative impact that AD/HD may have on the lifecourse.

1. Introduction

Attention-deficit/hyperactivity disorder (AD/HD) is a common disorder of childhood onset, characterised by problems with concentration, impulse control and overactivity, and associated with a variety of adverse adolescent and adult outcomes[1]. The most widely accepted diagnostic criteria in use for identifying AD/HD are those in the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) (American Psychiatric Association, 1994). The DSM-IV criteria describe three major subtypes of AD/HD: (1) inattentive type; (2) hyperactive-impulsive type; and (3) combined type (inattention and hyperactivity-impulsivity). AD/HD is a disorder of heterogeneous causes that has correspondingly heterogeneous neuroanatomical underpinnings (Sowell et al, 2003). However, amongst the different pathologies, a genetic susceptibility seems to be the most common (Hill and Taylor, 2001). Identifying and diagnosing AD/HD can be a problem, not least because of the possible different sub-types of AD/HD and often comorbid conditions (Brassett-Grundy and Butler, 2004). There are those who believe that AD/HD is the single most important specific condition complicating school life, the seriousness of which is further highlighted by the fact that the cost to the national exchequer in the UK in the mid-1990s was estimated at £1 billion per annum (Knapp, 1997). However, while much is known about this condition, there are some important gaps in knowledge. More extensive literature reviews have shown that there are a number of unanswered and under-researched questions that need addressing (Brassett-Grundy and Butler, 2004). The adult outcomes of AD/HD have not been rigorously researched, partly through the lack of suitable large-scale longitudinal data. Such data are now available through the British Birth Cohorts and this paper presents analyses of one such cohort to advance our knowledge of adult outcomes from childhood AD/HD.

In this paper we first review the relevant research literature on the outcomes of AD/HD, describe the available data, and the measure of AD/HD that is available in the data. The form of multivariate analysis of the data that we undertook is then described, followed by the results and our conclusions.

1.1 Outcomes of AD/HD

Prevalence figures for childhood AD/HD, based mainly on cross-sectional research on clinical populations, range from 3-10 per cent, with a maximum age risk somewhere between 5- and 10-years-old (American Psychiatric Association, 1994; Costello, 1989; Parr et al, 2003; Szatmari et al, 1989; Taylor, 1994). Epidemiological studies have reported higher prevalence rates ranging from 9-19 per cent (Paule et al, 2000; Shekim et al, 1985; Taylor et al, 1991). Most research agrees that boys are more likely than girls to develop AD/HD but studies report varying degrees of male overrepresentation, from ratios of 1.5:1 to 12:1 (e.g. Gomez et al, 1999; Parr et al, 2003; Pineda et al, 1999; Swanson et al, 1998; Wolraich et al, 1996). These ratios may represent a real sex difference or reflect differences in the subtype of AD/HD under investigation. Some of these differences represent the inherent biases in the samples studied, e.g. the identification and referral biases on the part of parents, teachers and health professionals (e.g. Glod et al, 1996; Swanson et al, 1998; Taylor, 1994).

Numerous longitudinal follow-up studies have endeavoured to investigate the destiny of those diagnosed with childhood AD/HD. These have lasted anywhere between four and fifteen years, and the weight of the evidence they contain suggests that a number of negative adolescent and adult outcomes await those with childhood AD/HD. These include continuing problems of AD/HD symptoms and also problems in a number of life domains, including: educational

[1] Readers are directed to an earlier paper by the authors that provides an overview of the current debates surrounding the definition, measurement, identification and treatment of AD/HD. It also reviews and evaluates the recent literature pertaining to adolescent and adult outcomes of AD/HD (Brassett-Grundy and Butler, 2004).

underachievement and cognitive problems; low socio-economic status; antisocial and criminal behaviour; mood and anxiety disorders; bipolar disorder; personality disorders; tic disorders; alcohol and drug abuse; general psychiatric problems; problems with relationships and psychosocial/emotional functioning; and road traffic accidents (as reviewed in Brassett-Grundy and Butler, 2004).

Few valid longitudinal studies into the outcomes of childhood AD/HD have been carried out and those that have been conducted to date have suffered a number of problems. One problem has been that the available samples contain inherent selection biases, which means that the conclusions drawn concerning adult outcomes may be flawed. For example, existing longitudinal studies have focused almost exclusively on young men and they tell us little about the female experience of AD/HD (e.g. Mannuzza et al, 1993; Taylor et al, 1991). Very few have reported on individuals outside of North America. Samples are often selected from clinic populations (e.g. Barkley et al, 2003; Dalsgaard et al, 2002; Mannuzza et al, 1993; Mannuzza et al, 1998) and thus tend to represent individuals with more severe forms of AD/HD. Sample sizes are also small, rarely reaching 200 participants. When groups of control participants are included, these have often been smaller in number (e.g. Barkley et al, 1990; Fischer et al, 1990; Molina and Pelham, 2003) and have differed significantly from the clinical group at baseline in spite of occasional efforts to match them (e.g. Mannuzza et al, 1998). Most of the studies that used control groups compared clinic-referred cases of AD/HD to individuals selected from the general population, which can confound the causes and characteristics of AD/HD with the reason for referral (e.g. Fischer et al, 1993). There is also evidence of bias in that samples have often included those with hyperactive and combined subtypes, neglecting the inattentive subtype AD/HD. Thus, none of the longitudinal research to date has been able to investigate the outcomes of AD/HD in a nationally representative birth cohort. This presents a challenge for statistical inference and generalisability.

In addition, it is difficult to compare findings across the longitudinal studies carried out to date owing to the variation in methodologies utilised. Often, different definitions of AD/HD have been used, perhaps a product of the historical changes that have occurred to the diagnostic criteria and accepted nosology. Those with the longest follow-up were based on samples of 'hyperactives' (e.g. Barkley et al, 2003; Fischer et al, 2002; Mannuzza et al, 1998; Weiss et al, 1985). Consequently, the adult outcomes for children with symptoms primarily of inattention are unknown. Varying methods of collection of data on outcomes have also been used in earlier studies (e.g. trained undergraduate interviewers versus professional interviewers). In some cases, follow-up interviewers have not been blind to the research participants' baseline AD/HD status (e.g. Weiss et al, 1985). This could obviously introduce biases into their perceptions of the adult functioning of clinical groups compared to control groups.

The existing longitudinal research has rarely reported on individuals beyond their mid-twenties. Some studies have reported on groups of individuals with wide age ranges. Both of these factors may mask important developmental differences (e.g. Barkley et al, 2003; Dalsgaard et al, 2002; Mannuzza et al, 1997). Attrition to samples is a problem for any longitudinal research, and in some AD/HD-related research this has been high. Weiss et al's (1985) sample, for example, suffered 40 per cent attrition. It is possible that those lost to follow-up represent those who have the most negative outcomes, and this longitudinal research may therefore underestimate the outcomes of childhood AD/HD.

It is rare to find longitudinal research that has controlled for confounding factors. Mannuzza et al (1998), for example, controlled only for parental SES. Differences observed at follow-up by Mannuzza et al (1998) may be due, therefore, to some feature other than AD/HD, not measured at baseline. There may also be problems with the identification of AD/HD symptoms in adulthood. Few studies have employed the most recently developed adult diagnostic scales.

Cross-sectional research, like much longitudinal research, has been ethnocentric, geographically and sex biased, and carried out with small samples of individuals from clinic populations with

combined or hyperactive type AD/HD. Therefore, it is apparent that research is required that aims to document the experience of AD/HD in females as well as males. It should include those with inattentive type AD/HD, and be based on individuals from representative populations. Prospective designs would be better at teasing out causal pathways, which aim to isolate the long-term effects of AD/HD with the collection of a wealth of information at baseline as well as at subsequent follow-ups, enabling pre-existing characteristics to be controlled.

This study aims to address some of these criticisms by supplying empirical evidence from the 1970 British Cohort Study (BCS70) (Butler et al, 1986), a thirty-year prospective longitudinal study of a nationally representative birth cohort. Our goals are to estimate the prevalence of childhood AD/HD in this sample as a basis for examining differences in adult outcomes for this group across a wide range of measures, for men and women separately, and in comparison with those who clearly did not have symptoms of childhood AD/HD.

2. The 1970 British Cohort Study data

The data used are taken from a national birth cohort, the 1970 British Cohort Study (BCS70) (Butler et al, 1986), which followed all children born in Great Britain in the first week of April 1970, totalling approximately 18,000 children. Their parents were interviewed when the children were newborn[2], age 5, age 10 and age 16[3]. The children themselves were interviewed at ages 10, 16, 21, 26 and 30. They were given standard ability tests at ages 5, 10 and 16, and medical officers and teachers were interviewed when the children were aged 10 and 16. As a result, a considerable body of educational, medical, social, psychological and economic longitudinal information is available.

The analyses we carried out were confined to the data collected at ages 0, 5, 10 and 30. Adult outcomes were derived from the age-30 data and related to characteristics at birth and age 5, and AD/HD symptoms at age 10. Given the prospective longitudinal design of this dataset, which was not developed specifically for the purpose of AD/HD research, the interviewers gathered information 'blind' of the cohort member's AD/HD status, which we subsequently ascribed using appropriate scales.

2.1 AD/HD identification criteria

In identifying AD/HD cases in the BCS70 retrospectively, our aim was to mimic the DSM-IV diagnostic criteria as far as possible by obtaining a measure of AD/HD in both the home and at school, by the age of 7. However, given the date and design of the BCS70, no AD/HD-specific diagnostic schedule was included at the age-5 sweep to allow the identification of a group of cohort members with AD/HD before age 7. More detailed information was collected, however, at the age-10 sweep and thus it was at this age that a group of cohort members with AD/HD, as measured in two settings, was identified. This took advantage of information contained in two behavioural scales: the Conners rating scale (Conners, 1969), completed by both parents and teachers; and the modified parental Rutter A(2) questionnaire (Rutter et al, 1970). The Conners rating scale is a well-validated and reliable behavioural screening tool that has since been used widely to identify AD/HD (e.g. Farre-Riba and Narbona, 1997; Rosenbaum and Baker, 1984). Likewise, the Rutter questionnaires are long established and highly respected screening tools, which can produce a score for diagnosis of a behavioural problem. Whilst not AD/HD-specific they include questions on concentration, and factor analysis reveals a factor pertaining to hyperactivity (e.g. Berglund, 1999; Mousa Thabet and Vostanis, 2001).

Although the BCS70 included part of the modified teacher's Rutter A(2) questionnaire at the age-10 sweep, the fractional nature of its presence precluded our ability to extract an inattentive/hyperactive subscore[4]. As a result, we decided to use only the Conners scale data from teachers for our school-based rating of AD/HD symptoms. For our home-based rating of AD/HD symptoms we used information from the same Conners scale completed by parents. Where this information was missing, we used information from an inattentive/hyperactive subscale based on four statements included in the parental Rutter A(2) questionnaire (the

[2] Information was collected on those born in Northern Ireland at birth however, since they were not followed-up, data on these individuals have been excluded from this analysis.

[3] It is important to note that the age-16 data suffers from a number of problems, not least the coincidence of a teachers' strike during data collection and diminished returns, which make interpretation of results more difficult.

[4] This would follow a method recommended by Elander and Rutter (1996), where at least two out of seven statements on inattention and overactivity contained in the Teacher's Rutter questionnaire are rated 'Yes, certainly applies'.

modified version of which was included in its entirety at the age-10 sweep). Thus, the cohort member was identified as having AD/HD if they had:

(1) a 'clinical' score on the teacher-completed Conners rating scale (where cut-off scores for females is 15 and for males is 18);

and

(2) *either* (i) a clinical score on the parent-completed Conners rating scale;

or (ii) at least two of four statements from the modified parental Rutter A(2) questionnaire on inattention and hyperactivity rated as 'Yes – certainly applies'[5].

It is worth noting that the questions pertaining to AD/HD and hyperactivity in the BCS70 data referred to the cohort member's current behaviour, and thus our identification criteria at a single sweep does not correspond to the DSM-IV criteria, which requires symptoms to be present for at least 6 months. However, given that we had set the identification criteria to include only those whose scores implied clinically significant impairment in social or academic functioning, it is likely that many had experienced symptoms for at least 6 months prior to interview.

From the total sample of 14,797 children present at the age-10 sweep of the BCS70, a group of 1,101 with AD/HD was identified for analysis: 412 girls and 689 boys[6]. However, by age 30, attrition from the sample had reduced the overall sample size to 10,405, of which 721 were those identified as having AD/HD at age 10: 291 girls and 430 boys (Table 1). Women were significantly more likely to be traced at age 30 than men, as well as those without AD/HD[7]. This is the group who were available for analysis of their adult outcomes.

[5] These questions were: (1) 'Very restless, often running about or jumping up and down, hardly ever still'; (2) 'Sits still and concentrates' (the coding for this question was reversed to equate to 'Has difficulty remaining seated and concentrating'); (3) 'Is squirmy or fidgety'; and (4) 'Cannot settle to do anything for more than a few moments'. These questions are normally answered categorically 'No – does not apply', 'Yes – applies somewhat' or 'Yes – certainly applies'. However, at the age-10 sweep of the BCS70, answers to these questions were given by parents on a visual analogue scale, which at each anchor were scored 0 (No – does not apply) and 100 (Yes – certainly applies). We rated those in the top third of the analogue scale as 'Yes – certainly applies'.

[6] It should be noted that for the brevity and simplicity of this paper, and given the constraints of the AD/HD-relevant information in the BCS70, we did not try to identify AD/HD subtypes at age 10, although this offers a possibility for further research.

[7] Women present at the age-10 sweep were 9 percentage points more likely to be traced at the age-30 sweep than men ($dF/dx=0.090$; $z=11.87$; $p<0.01$; $n=14,797$). Those with age-10 AD/HD were 5.1 percentage points less likely to be traced at the age-30 sweep than those without age-10 AD/HD ($dF/dx=-0.052$; $z=-3.61$; $p<0.01$; $n=14,797$).

Table 1: The number of men and women with and without age-10 AD/HD present at the age-30 sweep of the BCS70

	Numbers present at age 30 (as % of original age-10 sample)		
	Men	Women	Total
With age-10 AD/HD	430 (62)	291 (71)	721 (65)
Without age-10 AD/HD (or unknown)	4,622 (66)	5,062 (75)	9,684 (71)
Total	5,052 (66)	5,353 (75)	10,405 (70)

2.2 Prevalence of symptoms of AD/HD

In our sample, therefore, there was a prevalence of AD/HD of 7.4%, at the upper end of the range normally reported, supporting the notion that in clinical studies underdiagnosis may be common. As previous research has reported, males with AD/HD symptoms outnumber females at the age-10 sweep of the BCS70. However, there were clearly a sizeable number of females in this survey who did have AD/HD symptoms and the ratio at age 10 of males to females was lower than the majority of previous research has indicated, at 1.7:1.

2.3 Multivariate models

Our models set out to explain the determinants of adult outcomes at age 30, examining in particular, the effects on adult outcomes of age-10 AD/HD, after controlling for other potential determinants of outcomes at age 30. We carried out standard multivariate longitudinal analyses, which conditioned for typical socio-economic risk factors that predated the identification of AD/HD, so that we could be reasonably certain that differences in outcomes for those who developed AD/HD were not due to, for example, differences in family social class or differences in levels of parental education. It was not possible to factor-in the effects of genetic risk for AD/HD in the data available. However, we have estimated the differences in outcomes for those who had childhood AD/HD and those who did not, taking into account as much prior information as possible. These are best conceptualised as indicators of the extent to which children with AD/HD are at risk of having certain outcomes, rather than the precise effects of AD/HD.

There could be a number of explanations for any observed statistical association of AD/HD status and negative adult outcomes. For example, children who develop AD/HD may already be at risk of negative adult outcomes whether or not they developed AD/HD, essentially because experiences of things such as family deprivation or low socio-economic status (SES) tends on average to lead to both negative outcomes and developing AD/HD[8]. Alternatively, it is possible that AD/HD status mediates the effect of prior aspects of psychological development that leads to AD/HD status and negative outcomes, e.g. antisocial children could be both more likely to develop AD/HD and more likely to experience negative outcomes, such as crime. In this instance, AD/HD would be merely a signal of this and may have no additional effect in itself – the negative outcome being, in fact, the product of a comorbid condition. However, it is also possible that developing AD/HD in itself does lead to negative outcomes, in addition to the processes already described. The use and analysis

[8] Peterson et al (2001) and Schachar et al (1981), for example, report a link between low SES and development of AD/HD.

of longitudinal data such as that contained in the BCS70 allows us to distinguish between these explanations and ascertain to what extent childhood AD/HD operates as an independent risk factor for negative adult outcomes independently of the other psychosocial and environmental risk factors that lead both to AD/HD and negative outcomes.

2.4 Outcome variables defined

Twenty-four age-30 adult outcomes were used in the analysis. They were constructed bearing in mind suggestions made in the literature covering a series of life domains, namely education, economic status, housing and homelessness, crime, health, parenthood and marital status, and accidents. Given the breadth of outcomes analysed, for simplicity they were coded as binary variables, enabling us to carry out logistic regression analyses that adjusted for the discrete nature of the outcomes. Thus, in the presentation of results we report the marginal effects, i.e. the change in probability of the outcome resulting from the membership of the AD/HD group (dF/dx statistics). The definitions and summary statistics for the twenty-four outcomes investigated are shown in Appendix 1, Table A1. In some cases a range of possible measures of each domain were investigated[9].

2.5 Control variables defined

A wealth of social, psychological and economic information was collected in the BCS70. This enabled the analyses to control for pre-existing circumstances, and/or background familial and personal characteristics. The summary statistics for the dummy control variables, based on information collected at birth and age 5, and used in the logistic regressions on the outcomes of AD/HD, are summarised in Appendix 2, Table A2. These control variables were devised to capture the important elements identified in previous research; they include measures of social class, parental education, ethnicity, birthweight, perinatal complications, family formation, maternal depression, parenting style and cohort member's emotional and behavioural characteristics.

[9] For example, the educational outcomes represent different ways of dissecting the overall outcome 'highest qualification attained'. We present three different ways of exploring this: (1) 'no qualifications' versus 'some qualifications'; (2) 'level 2 qualifications or less' versus 'level 3 qualifications or more'; and (3) 'less than a degree' versus 'a degree or more'. Those with no qualifications are thus included in each of the educational outcomes shown.

3. Results

A set of regressions was carried out to isolate the effects of age-10 AD/HD on age-30 adult outcomes by controlling for the socio-economic and personal characteristics of cohort members as measured at birth and age 5. This represented the 'conditional' outcomes for those with AD/HD at age 10, comparing them to individuals with similar social, economic, parental and personal characteristics in childhood. This tested whether age-10 AD/HD would still lead to negative adult outcomes independent of factors that were measured prior to this status, such as childhood deprivation and adversity. These regressions were carried out for men and women separately, and were restricted to those present at both the age-10 and age-30 sweeps of the BCS70. The results of the regressions showing the conditional outcomes for those with AD/HD at age 10 are shown in Table 2. Since the concern of this paper is outcomes of childhood AD/HD, the marginal effects of the numerous controls entered into each set of regressions are not reported in detail and instead, for simplicity and brevity, we report the p-value of the control variables as a total[10].

The results in Table 2 demonstrate that, even when controlling for a host of background characteristics, age-10 AD/HD is a significant independent risk factor for eighteen of the twenty-three adverse outcomes for men, and fifteen of the twenty-four adverse outcomes for women. These span the education, economic status, housing, relationship and family formation, crime, and health life domains. For example, men are 13.1 percentage points more likely to report minor offending by the age of 30 if they had AD/HD at age 10, and women are 16.2 percentage points more likely to be cigarette smokers at age 30 if they had AD/HD at age 10.

It should be noted that 15 of the 721 children (10 boys and 5 girls – 2%) with age-10 AD/HD did not experience any of the twenty-four age-30 adverse outcomes, compared to 477 of the 9,684 children (236 boys and 241 girls – 5%) who did not have age-10 AD/HD.

[10] The full results for each regression carried out are available from the first author.

Table 2: Conditional outcomes at age 30 for men and women in the BCS70 with AD/HD at age 10

	Men				Women			
Specific 30-year outcome	dF/dx	z-scores	p-value for age 0 and age 5 control variables	N	dF/dx	z-scores	p-value for age 0 and age 5 control variables	N
No qualifications	.113	**4.62****	.00	5,030	.088	**3.23****	.00	5,336
Level 2 qualifications or less	.166	**6.13****	.00	5,030	.107	**3.53****	.00	5,336
No Level 4 qualifications	.087	**4.32****	.00	5,030	.084	**3.58****	.00	5,336
Low income	.032	1.65	.00	3,873	.122	**3.45****	.00	3,620
Benefit claimant	.063	**4.99****	.00	5,030	.085	**4.30****	.00	5,339
Workless household	.033	**2.52***	.00	5,047	.051	**2.81***	.00	5,352
Workless household with children	-.001	0.34	.00	4,771	.036	**2.58***	.00	5,352
Temporary/social housing	.062	**4.13****	.00	4,982	.127	**5.75****	.00	5,315
Homelessness	.027	**2.50***	.00	4,325	.016	1.07	.00	5,068
Parent before age 19	-	-	-	-	.001	1.55	.00	4,774
Single parent	.000	0.38	.00	4,254	.091	**4.93****	.00	5,352
Single, separated or divorced	.061	**2.36***	.00	4,977	.106	**3.59****	.00	5,326
Minor offender	.131	**4.96****	.00	4,974	.038	**2.37***	.00	5,287
Persistent offender	.078	**5.56****	.00	4,974	.000	0.57	.00	4,832
Victim of assault	.038	**2.61***	.00	5,020	.006	0.64	.00	5,274
Smoker	.096	**3.66****	.00	5,018	.162	**5.28****	.00	5,331
Life dissatisfaction	.064	**3.40****	.00	4,972	.069	**3.11****	.00	5,286
Depressed	.095	**5.02****	.02	4,977	.054	**2.18***	.00	5,292
Alcohol problems	.024	**4.27****	.00	4,818	.000	0.60	.00	4,544
Drug problems	.000	**2.02***	.00	4,873	.004	**3.25****	.00	4,461
Bipolar disorder	.001	0.59	.00	2,946	.000	0.44	.00	3,538
OCD	.003	**3.46****	.00	4,840	.006	1.17	.00	4,923
Psychiatric disturbance	.041	**2.04***	.07	4,974	.018	0.68	.00	5,285
Road traffic accident	.032	1.45	.11	5,020	-.013	0.57	.00	5,317

* significant at 5%; ** significant at 1%

Note: For men, one outcome could not be investigated owing to insufficient response data to run a regression ('Parent before age 19').

4. Discussion

This paper has supplied empirical evidence from a thirty-year prospective longitudinal study of a nationally representative birth cohort, of both the prevalence of childhood AD/HD, and the adult outcomes of childhood AD/HD, for both males and females, whilst controlling for a large number of pre-existing characteristics.

4.1 Adult outcomes of AD/HD

Age-10 AD/HD was a significant independent risk factor for the majority of our age-30 adverse outcomes analysed for men and women, even after controlling for a wide range of background social, parental, familial and personal characteristics. When compared to BCS70 cohort members *with similar background characteristics*, men and women with age-10 AD/HD were still significantly more likely at age 30 to: have no or low level qualifications; have a low SES, as evidenced by benefit claiming status, living in a workless household and/or living in temporary or social housing; be single, separated or divorced; be cigarette smokers; be dissatisfied with their lives; be depressed; and have drug problems. Similar effects were noted in other studies (see the review in Brassett-Grundy and Butler, 2004) although in many of these, outcomes may have been the result of unmeasured pre-existing characteristics, comorbid conditions or biased sample selection.

This study also found that men and women with childhood AD/HD were at higher risk of having contact with the police as a minor offender. This concurs with other research (e.g. Hechtman et al, 1984; Rasmussen and Gillberg, 2000). However, it is at variance with the study by Babinski et al (1999) who found that the association with adult crime was only true for men. We also found that men were at greater risk of being victims of crime in the form of an assault or mugging. This is something that has not been reported before, and may be the adult expression or progression of bullying that is commonly reported in AD/HD children.

Men with childhood AD/HD were also specifically more likely at age 30 to: have experienced homelessness; have had contact with the police or courts as a persistent offender; have alcohol problems; have experienced Obsessive Compulsive Disorder (OCD) symptoms; and to have a psychiatric disturbance (as rated by a score of 4 or more on the General Health Questionnaire 12). Women with childhood AD/HD, on the other hand, were specifically more likely at age 30 to: be in poorly paid employment; be a single parent; living in a workless household with children; and be a single parent. Whilst alcohol and/or drug abuse are outcomes that have been identified for men and women in previous research (e.g. Ercan et al, 2003; Hechtman et al, 1984; Rasmussen and Gillberg, 2000), the distinction between men being more at risk of alcohol problems and women being more at risk of drug problems is one that has not previously been reported. That women were at greater risk of drug abuse may be evidence that this group of individuals had not received medical treatment for AD/HD, since research suggests treatment for childhood AD/HD with stimulants protects against illegal substance use later in life (as found by Wilens et al (2003) in a meta-analysis of six follow-up studies). This may also be evidence that girls with AD/HD are less likely to be identified and treated than boys.

It is interesting to note that only men were at risk of OCD symptoms in adulthood, and neither men nor women were at risk of bipolar disorder. This may in part be an artefact of the wording of the questions that identified this group; these relied on self-report to three questions and could not be considered a formal clinical diagnosis. In this sense, it is preferable to think of this finding in terms of men being more likely to exhibit OCD tendencies, or self-report OCD-like behaviour. Nevertheless, these are interesting sex differences.

After controlling for birth and age 5 characteristics we found evidence of general psychiatric disturbance only in males (as reported by Barkley, 2002; Fischer et al, 2002; Mannuzza et al,

1998). This contrasts with the female bias towards this outcome reported by Dalsgaard et al (2002). Earlier studies used different measures of psychiatric disturbance and different samples, however, given that our findings represent the results of a large nationally representative sample using a standard diagnostic instrument, they may be considered more reliable.

We did not find a link between childhood AD/HD and road traffic accidents, contrary to the findings of studies such as Barkley (2002) and Barkley et al (1993). This may again be an artefact of the wording of the questions. In the BCS70 questions, no distinction was made between those who were driving and those who were passengers, nor whether if, as a driver, the cohort member caused the accident. The questions also relied on self-report. This may be worth further investigation in a more detailed follow-up.

Overall, our results demonstrate that men and women who had childhood AD/HD were at a higher risk of experiencing a range of negative outcomes at age 30. The risks to men were greater than those to women.

4.2 Limitations

In interpreting these data we need to be aware of some of the limitations of research of this kind. Attrition to the cohort is always a problem of longitudinal studies such as the BCS70, and our figures show that the study was more likely to lose contact those who developed AD/HD during childhood, especially the boys. Nevertheless, the BCS70 has managed to follow-up an impressive number of cohort members over the course of three decades. A larger number of individuals were identified as having childhood AD/HD than has been identified by previous follow-up studies, affording us the opportunity of multivariate longitudinal statistical analysis to provide more robust estimates of the effects that AD/HD has on adult life. Also, given that large-scale survey research often fails to trace those who have experienced the most adverse outcomes, our findings probably underestimate the level of lifecourse adversity awaiting those with childhood AD/HD.

We do not know exactly when before age 10 cohort members developed AD/HD, nor necessarily the subtype of AD/HD developed (beyond the broad category we have identified using the Rutter and Conners scales), both of which may have an impact on outcomes. Our study reports on a global group of individuals meeting criteria for a general definition of AD/HD, and as such individuals have not been designated to one of the subtypes diagnosable using the DSM-IV criteria. It is possible that different outcomes are correlated with each of the subtypes, and a more differentiated analysis of the data is needed to explore these differences.

This study is reliant on an early version of the Conners rating scale and a subscale of the parental Rutter questionnaire for identifying those with AD/HD rather than a multi-modal diagnostic approach that is widely recommended but rarely achieved in large-scale data. However, these rating scales have been validated and are considered reliable. In addition, scores on these scales were obtained through blind interviews. Our identification criteria adhere to DSM-IV criteria in as far as clinical scores having been obtained in at least two settings. We believe that the criteria that were set for a positive diagnosis of AD/HD were stringent enough to include the vast majority of those who actually had AD/HD at age 10. It is likely that some BCS70 cohort members who had AD/HD were excluded, resulting in an underestimation of the effects of AD/HD.

Our analysis of AD/HD individuals did not differentiate between those who did or did not receive treatment for their AD/HD symptoms, be it pharmacological or otherwise. However, given that this cohort was born in Britain in 1970, we have assumed that few (if any) will have received treatment during childhood or adolescence. If our AD/HD group includes those who received treatment, our analysis will have, again, underestimated the effects of AD/HD; it is possible that if they were removed from our study the outcomes may be even worse.

The group of control variables used to isolate the effects of childhood AD/HD on adult functioning could also be criticised. In controlling for early childhood adversity we have been restricted to those variables available in the BCS70, a study that was not specifically designed to analyse the experiences of those with childhood AD/HD. Thus, our group of control variables is not an exhaustive one. However, we have selected a powerful array of many of the psychosocial and environmental control variables considered important by other researchers, covering a wide range of characteristics. We were unable, however, to control for genetic risk factors.

Finally, in reporting the outcomes of childhood AD/HD for a specific cohort of children born in 1970, our results may not necessarily be generalisable to those who are diagnosed with AD/HD today. We need to be aware of the considerable social, cultural, economic and political changes in Britain since then, which has included changes to child and adolescent mental health services, as well as other services with which AD/HD individuals come into contact, e.g. educational, police, probation and judicial services. Nevertheless, this study provides evidence from a nationally representative cohort, and it is only through longitudinal research that we can be more robust in our analysis of the causal relationships between temporal factors and 'outcomes', which by necessity take one or two decades to become apparent. This enables us to institute policies and interventions to prevent the occurrence of adverse lifecourse outcomes for younger generations.

4.3 Conclusions

This study has shown that men and women who had childhood AD/HD were significantly more likely than those without AD/HD to face a wide range of negative outcomes in adulthood, typical of social exclusion, spanning life domains of education, economic status, housing, relationships, crime and health. These findings hold even when controlling for a host of background personal, familial, social and economic characteristics. On the whole, men tended to fare worse. They were also specifically at greater risk than women of homelessness, more serious offending, being a victim of assault, alcohol problems, OCD-like behaviour and having a psychiatric disturbance by age 30. Women were specifically at greater risk than men of earning low incomes, living in workless households with children and being single parents. We do not dismiss the number of those who at age 30 did not experience adverse adult outcomes; in fact, 2% of the sample of children with age-10 AD/HD had not experienced any of the twenty-four outcomes studied at age 30. However, they have survived against the odds and the fact is that childhood AD/HD is more likely to lead to an adulthood of adversity in a number of life domains. The interesting sex differences we have discovered are worthy of replication and further investigation. This study has also found that AD/HD may be more prevalent than most previous research has indicated, and is experienced by larger numbers of females than has been considered.

Thus, AD/HD is as much a female problem as it is a male problem and the adult lives of those with childhood AD/HD are typified by social deprivation and adversity. In the light of these results there may be savings to be made, in both monetary and emotional terms, in the long-term and the short-term, through: (1) better screening for AD/HD, perhaps in primary care; (2) wider use of sensitively designed early interventions and individually tailored treatment plans; (3) ongoing treatment and support for those with AD/HD through adolescence into adulthood; and (4) raised awareness of AD/HD amongst parents, health professionals, educators and those in the criminal justice system. These measures may help to ease the negative impact that childhood AD/HD has on the lifecourse, preventing young sufferers from becoming socially excluded adults.

Using the BCS70, our future goals are to ascertain the degree to which AD/HD symptoms persevere through childhood to adolescence, to describe the psychosocial risk factors for age-10 AD/HD and to assess whether there are different age-30 outcomes for those with different AD/HD subtypes. We also plan to carry out a range of similar analyses using another UK national cohort study, the 1958 National Child Development Study (NCDS) (Shepherd, 1985).

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Appendix 1: Summary statistics for outcome variables

Table A1: Summary statistics for age-30 binary outcome variables, for those present at both the age-10 and age-30 sweeps of the BCS70

OUTCOME VARIABLE	VARIABLE DEFINITION	AGE 30 N	MEAN	STD. DEV.
1. Education				
No qualifications	Has no qualifications	10,366	0.280	0.449
Level 2 qualifications or less	Highest qualification gained is Level 2 (NVQ 1, CSE grade 2, O level grade D, or equivalent) or less	10,366	0.363	0.481
No Level 4 qualifications	Does not have a Level 4 qualification, i.e. degree or above	10,366	0.801	0.400
2. Economic situation				
Low income	In receipt of a low income (defined as 80% or less of the median of the natural logarithm of the cohort member's real gross hourly earnings)	7,493	0.186	0.389
Benefit claimant	Cohort member or his/her partner claims Job Seekers Allowance, Income Support or Housing Benefit	10,369	0.104	0.305
Workless household	Lives in a workless household	10,399	0.100	0.299
Workless household with children	Lives in a workless household with children	10,399	0.054	0.226
3. Housing				
Temporary/social housing	Lives in temporary or Local Authority/Housing Association-rented accommodation	10,297	0.145	0.352
Homelessness	Has been homeless since last interview	9,474	0.068	0.251
4. Relationships and parenting				
Parent before age 19	Had a child before age 19	10,399	0.005	0.073
Single parent	Single parent	10,399	0.057	0.231
Single, separated or divorced	Single, separated or divorced	10,303	0.333	0.471
5. Crime				
Minor offender	Arrested, cautioned or found guilty in court at least once	10,261	0.226	0.419
Persistent offender	Found guilty in court at least twice	10,261	0.046	0.211
Victim of assault	Has been a victim of violent assault, mugging or sexual assault	10,352	0.070	0.255
6. Health				
Smoker	Currently smokes one or more cigarettes daily	10,349	0.369	0.483
Life dissatisfaction	Dissatisfied with how life has turned out so far (as indicated by a response between 1 and 5 on a scale of 1 to 10, where 1 implies complete dissatisfaction and 10 implies completely satisfied)	10,258	0.149	0.356

OUTCOME VARIABLE	VARIABLE DEFINITION	AGE 30 N	MEAN	STD. DEV.
Depressed	Depressed (score of 7 or more on the 'Malaise' scale)	10,269	0.171	0.376
Alcohol problems	Has had, or still has, problems with alcohol	10,405	0.015	0.120
Drug problems	Has had, or still has, problems with drugs	10,405	0.013	0.113
Bipolar disorder	Has had, or still has, problems with overexcitement and overconfidence (as an indicator of mania or bipolar disorder)	10,405	0.003	0.059
OCD	Has had, or still has, problems with feeling compelled to repeat certain activities (as an indicator of obsessive compulsive disorder (OCD))	10,405	0.010	0.099
Psychiatric disturbance	Psychiatric disturbance (score of 4 or more on the 'General Health Questionnaire 12')	10,259	0.204	0.403
7. Accidents				
Road traffic accident	Has had a road accident as a driver or passenger in a vehicle	10,352	0.203	0.402

Note: For men, one outcome could not be investigated owing to insufficient response data to run a regression ('Parent before age 19').

It is worth noting that although the total possible sample size was 10,405, the number of observations ('Age-30 N') for each outcome was often lower than this, owing to information missing in the variables used to create them (i.e. where respondents chose, or failed, to supply an answer to a question). The statistics presented are therefore based on known information. In a few cases, specifically the variables relating to alcohol, drugs, bipolar disorder and obsessive compulsive disorder (OCD), only positive answers were recorded on the database. Thus, all missing information was coded, conservatively, as negative. This probably underestimates the true means for these outcomes.

Appendix 2: Summary statistics for control variables

The statistics presented are based on all those cohort members who were present at both the age-10 and age-30 sweeps of the BCS70, and for whom information on sex was available (a total of 10,405 individuals).

The control variables listed below relate to information recorded at birth and age 5. These were all dummy variables (i.e. assigned a value of '0' for non-group membership and '1' for group membership). Missing values were replaced by a missing value dummy variable for each construct. The dummy variables within each category were mutually exclusive, and thus the sum of the means for each category is 1. For example, the breast-fed category has three dummy variables: one for 'breast-fed' group membership; one for 'never breast-fed' group membership; and one for 'unknown whether breast-fed' group membership. The mean for the breast-fed group is 0.320, showing that 32.0% of our sample were breast-fed. The mean for the never breast-fed group is 0.516, showing that 51.6% of our sample were never breast-fed. The mean for the unknown whether breast-fed group is 0.164, showing that information is missing on breast-feeding status for 16.4% of our sample. The sum of 0.320, 0.516 and 0.164 equals 1, i.e. 100% of our sample.

Table A2: Independent control variables measured at the birth and age-5 sweeps of the BCS70

Age-0 control dummy variables

	N	Mean	Std. Dev.
Birthweight in bottom quartile	10,405	0.219	0.414
Birthweight in second quartile	10,405	0.246	0.431
Birthweight in third quartile	10,405	0.257	0.437
Birthweight in top quartile	10,405	0.234	0.423
Birthweight unknown	10,405	0.044	0.206
Breast-fed	10,405	0.320	0.466
Never breast-fed	10,405	0.516	0.500
Unknown whether breast-fed	10,405	0.164	0.370
Mother's marital status married	10,405	0.887	0.317
Mother's marital status single/divorced/ separated/ widowed/other	10,405	0.048	0.213
Mother's marital status unknown	10,405	0.065	0.247
No father figure (or unknown)	10,405	0.030	0.172
Father figure present	10,405	0.970	0.172

	N	Mean	Std. Dev.
No siblings	10,405	0.410	0.492
One older sibling	10,405	0.317	0.465
Two older siblings	10,405	0.141	0.348
Three or more older siblings	10,405	0.088	0.283
Unknown number of older siblings	10,405	0.044	0.204
Ethnic group is European/British/Irish/other White	10,405	0.945	0.228
Ethnic group is West Indian/African/other Black	10,405	0.009	0.093
Ethnic group is Asian	10,405	0.020	0.140
Ethnic group is other	10,405	0.026	0.160
Ethnic group is unknown	10,405	0	0
Mother's age at cohort member's (cm's) birth 18 years or under	10,405	0.047	0.212
Mother's age at cm's birth 19 years or over	10,405	0.883	0.321
Mother's age at cm's birth unknown	10,405	0.070	0.255
Father's age at cm's birth 18 years or under	10,405	0.149	0.356
Father's age at cm's birth 19 years or over	10,405	0.631	0.483
Father's age at cm's birth unknown	10,405	0.220	0.414
Mother's region of origin is South East England	10,405	0.219	0.414
Mother's region of origin is elsewhere in England	10,405	0.491	0.500
Mother's region of origin is Wales (inc. Monmouth)	10,405	0.057	0.233
Mother's region of origin is Scotland	10,405	0.098	0.297
Mother's region of origin is Northern Ireland	10,405	0.005	0.068
Mother's region of origin is Eire	10,405	0.018	0.132
Mother's region of origin is other country	10,405	0.046	0.211
Mother's region of origin is unknown	10,405	0.066	0.249
Mother's occupational class I or II	10,405	0.081	0.273
Mother's occupational class IIINM or IIIM	10,405	0.312	0.464
Mother's occupational class IV	10,405	0.172	0.377
Mother's occupational class V	10,405	0.008	0.089
Mother's occupational class other/unknown	10,405	0.427	0.495

	N	Mean	Std. Dev.
Father's occupational class I or II	10,405	0.159	0.366
Father's occupational class IIINM or IIIM	10,405	0.542	0.498
Father's occupational class IV	10,405	0.127	0.332
Father's occupational class V	10,405	0.049	0.217
Father's occupational class other/unknown/NA	10,405	0.123	0.328
Mother has no qualifications	10,405	0.431	0.495
Mother's highest qualification: O Level or equivalent, Vocational or other	10,405	0.282	0.450
Mother's highest qualification: A Level or equivalent, SRN, Certificate of Education, Degree or above	10,405	0.094	0.292
Mother's highest qualification: unknown	10,405	0.193	0.394
Father has no qualifications	10,405	0.340	0.474
Father's highest qualification: O Level or equivalent, Vocational or other	10,405	0.228	0.420
Father's highest qualification: A Level or equivalent, SRN, Certificate of Education, Degree or above	10,405	0.179	0.384
Father's highest qualification: unknown	10,405	0.253	0.434
No pregnancy and/or birth complications (or unknown)	10,405	0.249	0.432
One pregnancy or birth complication	10,405	0.308	0.462
Two pregnancy and/or birth complications	10,405	0.233	0.423
Three or more pregnancy and/or birth complications	10,405	0.210	0.407

Note: Pregnancy and birth complications were based upon variables relating to: mother smoking during pregnancy; pitting oedema in pregnancy; eclamptic fits pre-labour; bleeding during pregnancy; mother was an inpatient during pregnancy; mother had a pelvic x-ray during pregnancy; mother was in labour for 24 hours or more; foetal heartbeat was abnormal; eclampsia during labour; baby's respiration was achieved after 1 minute after birth; baby was jaundiced; baby experienced breathing problems; baby had a cyanotic attack; baby had fits/convulsions; baby had abnormal cerebral signs; baby had fractures; baby had cephalhaematoma; baby had sticky eyes; baby had umbilical discharge; baby had blood transfusions; baby had operations (excluding circumcision); baby had some other illness/condition.

Age-5 control dummy variables

	N	Mean	Std. Dev.
Has been to Child Guidance	10,405	0.005	0.068
Has not been to Child Guidance	10,405	0.781	0.414
Unknown if ever been to Child Guidance	10,405	0.214	0.410

	N	Mean	Std. Dev.
Never had headaches in last year	10,405	0.516	0.500
Has less than one headache per month	10,405	0.236	0.425
Has headaches monthly or weekly	10,405	0.049	0.215
Unknown whether has headaches	10,405	0.199	0.400
Never had stomach-aches or vomiting in last year	10,405	0.326	0.469
Has less than one stomach-ache or vomiting per month	10,405	0.398	0.489
Has stomach-aches or vomiting monthly or weekly	10,405	0.081	0.272
Unknown whether has stomach-aches or vomiting	10,405	0.195	0.396
Never been bilious in last year	10,405	0.625	0.484
Bilious less than once per month	10,405	0.128	0.334
Bilious monthly or weekly	10,405	0.014	0.116
Unknown whether bilious	10,405	0.233	0.422
Never had temper tantrums in last year	10,405	0.474	0.499
Has less than one temper tantrums per month	10,405	0.145	0.352
Temper tantrums monthly or weekly	10,405	0.174	0.379
Unknown whether has temper tantrums	10,405	0.207	0.405
Has sleeping difficulty	10,405	0.214	0.410
Has no sleeping difficulty	10,405	0.623	0.485
Unknown if cohort member has sleeping difficulty	10,405	0.163	0.369
Wets self during day	10,405	0.168	0.374
Does not wet self during day	10,405	0.669	0.470
Unknown if cohort member wets self during day	10,405	0.163	0.369
Wets self during night	10,405	0.083	0.275
Does not wet self during night	10,405	0.755	0.430
Unknown if cohort member wets self during night	10,405	0.162	0.369
Soils self	10,405	0.034	0.182
Does not soil self	10,405	0.804	0.397
Unknown if cohort member soils self	10,405	0.162	0.369

	N	Mean	Std. Dev.
Has eating/appetite difficulty	10,405	0.314	0.464
Has no eating/appetite difficulty	10,405	0.523	0.499
Unknown if cohort member has eating/appetite difficulty	10,405	0.163	0.369
Rutter score in non-clinical range	10,405	0.700	0.458
Rutter score in clinical range	10,405	0.142	0.350
Rutter score unknown	10,405	0.158	0.365
Change since birth - father figure now absent	10,405	0.029	0.169
No change since birth in father figure becoming absent	10,405	0.971	0.169
Change in father's occupational class since birth down into classes IV or V	10,405	0.057	0.233
No change (or unknown) in father's occupational class since birth down into classes IV or V	10,405	0.943	0.233
Change in mother's occupational class since birth down into classes IV or V	10,405	0.040	0.196
No change (or unknown) in mother's occupational class since birth down into classes IV or V	10,405	0.960	0.196
Housing tenure: owned outright	10,405	0.110	0.312
Housing tenure: being bought	10,405	0.386	0.487
Housing tenure: Council rented	10,405	0.254	0.436
Housing tenure: other	10,405	0.089	0.285
Housing tenure: unknown	10,405	0.161	0.367
Additional siblings since birth	10,405	0.373	0.484
No (or unknown) additional siblings since birth	10,405	0.627	0.484
Housing density bottom quartile	10,405	0.142	0.349
Housing density second quartile	10,405	0.416	0.493
Housing density third quartile	10,405	0.114	0.318
Housing density top quartile	10,405	0.159	0.366
Housing density unknown	10,405	0.169	0.375
One or less household moves since birth	10,405	0.668	0.471
Two or more household moves since birth	10,405	0.165	0.371
Household moves since birth unknown	10,405	0.167	0.373

	N	Mean	Std. Dev.
Parent(s) died, divorced or separated since birth	10,405	0.051	0.219
Parent(s) not known to have died, divorced or separated since birth	10,405	0.949	0.219
Mother not depressed (Malaise score under 7)	10,405	0.601	0.490
Mother depressed (Malaise score 7 or over)	10,405	0.174	0.379
Mother's Malaise score unknown	10,405	0.225	0.418
Parent does not hold authoritarian views towards parenting	10,405	0.389	0.488
Parent does hold authoritarian views towards parenting	10,405	0.432	0.495
Unknown if parent holds authoritarian views towards parenting	10,405	0.179	0.383
Has no special educational needs or is not educationally subnormal	10,405	0.741	0.488
Has special educational needs or is educationally subnormal	10,405	0.013	0.114
Unknown if has special educational needs or is educationally subnormal	10,405	0.246	0.430
Has never been in care	10,405	0.830	0.375
Has been, or is, in care	10,405	0.012	0.107
Unknown if has ever been in care	10,405	0.158	0.365
Parental rating - not hyperactive	10,405	0.579	0.494
Parental rating - mildly hyperactive	10,405	0.162	0.369
Parental rating - hyperactive	10,405	0.074	0.263
Parental rating - very hyperactive	10,405	0.022	0.147
Parental rating - unknown if hyperactive	10,405	0.163	0.369

Prevalence and adult outcomes of Attention-Deficit/Hyperactivity Disorder

Evidence from a 30-year prospective longitudinal study

Attention-deficit/hyperactivity disorder (AD/HD) is a common disorder of childhood onset, characterised by problems with concentration, impulse control and overactivity, and associated with a variety of adverse adolescent and adult outcomes. However, whilst much is known about this condition, there are some important gaps in knowledge. The adult outcomes of AD/HD have not been rigorously researched, partly through the lack of suitable large-scale longitudinal data. Such data are now available through the British Birth Cohorts and this report presents analyses of one such cohort, the 1970 British Cohort Study, to advance our knowledge of adult outcomes from childhood AD/HD. Using data on over 10,000 individuals, the aims of this study were to provide an estimate of the prevalence of childhood AD/HD in a birth cohort, and to explore the age-30 outcomes of those with childhood AD/HD.

In this paper the authors review the relevant research literature on the outcomes of AD/HD, describe the available data, and the measure of AD/HD that is available in the data. The form of multivariate analysis of the data that was undertaken is then described, followed by the results and the authors' conclusions.

This study follows on from an earlier paper written by Brassett-Grundy and Butler, ***Attention-Deficit/Hyperactivity Disorder: an overview and review of the literature relating to the correlates and lifecourse outcomes for males and females*** (2004), which provides an overview of the current debates surrounding the definition, measurement, identification and treatment of AD/HD as well as evaluating the recent literature pertaining to adolescent and adult outcomes of AD/HD.

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