Adolescent clinical outcomes for young people with attention-deficit hyperactivity disorder

Kate Langley, Tom Fowler, Tamsin Ford, Ajay K. Thapar, Marianne van den Bree, Gordon Harold, Michael J. Owen, Michael C. O'Donovan and Anita Thapar

Background
Attention-deficit hyperactivity disorder (ADHD) is recognised as a common, disabling condition. Little information is available regarding the long-term outcomes for individuals with ADHD in the UK.

Aims
To examine the 5-year outcome for a UK cohort of children with diagnosed, treated ADHD and identify whether maternal and social factors predict key outcomes.

Method
One hundred and twenty-six school-aged children (mean age 9.4 years, s.d. = 1.7) diagnosed with ADHD were reassessed 5 years later during adolescence (mean age 14.5 years, s.d. = 1.7) for ADHD, conduct disorder and other antisocial behaviours.

Results
Most adolescents (69.8%) continued to meet full criteria for ADHD, were known to specialist services and exhibited high levels of antisocial behaviour, criminal activity and substance use problems. Maternal childhood conduct disorder predicted offspring ADHD continuity; maternal childhood conduct disorder, lower child IQ and social class predicted offspring conduct disorder symptoms.

Conclusions
The treatment and monitoring of ADHD need to be intensified as outcomes are poor especially in offspring of mothers with childhood conduct disorder symptoms.

Declaration of interest
None.

Attention-deficit hyperactivity disorder (ADHD) is the most common reason for follow-up in UK child and adolescent mental health services (CAMHS).[1] Recognition and treatment are considered important.[2] However, little is known regarding long-term outcomes of children receiving ADHD interventions in UK CAMHS. The clinical outcomes of ADHD in other countries are poor and the MTA trial[3] found treatment benefits reduced over time. Utilising a sample of UK individuals diagnosed with ADHD in childhood reassessed in adolescence, we investigated the prevalence of key adolescent outcomes examining whether maternal and social factors influence the course of ADHD.

Method

Sample

The Cardiff Longitudinal ADHD Sample Study (CLASS) recruited children between 1998 and 2002 from child and adolescent psychiatry and paediatric out-patient clinics across north-west and south-west England and South Wales, UK.[4] Initially, 98% of clinic families agreed to participate. Five years later, we invited individuals who had reached adolescence (aged over 12 years) to participate in a follow-up study. Those eligible for follow-up were 157 British White children, aged 6–13 years (mean age 9.4 years, s.d. = 1.7), who met diagnostic criteria for DSM–IV[5] ADHD at baseline (time 1 (T_1)) and were currently (time 2 (T_2)) aged over 12 years. These children were newly referred at T_1, had IQ test scores above 70, no diagnosis of autism or related disorders, Tourette syndrome or any neurological conditions. At initial assessment, all clinicians responsible for the care of the children were provided with a written feedback summary of the research diagnostic assessment information confirming the ADHD diagnosis and any comorbid conditions such as conduct disorder.

At follow-up, participants were aged 12–18 years (mean age 14.5 years, s.d. = 1.7). Contact had been maintained with families and 95% of the original sample was traced; 80% ($n = 126$)

participated in the follow-up. We obtained informed consent and assent from parents and children at both time points. The North-West England Multi-Centre Research Ethics Committee, UK, approved the study.

Comparison data, using identical questionnaire measures, on substance use were available for 121 age- and gender-matched controls (7 females, 114 males; mean age 14.5 years, s.d. = 1.5) from a general population-based sample, the Cardiff Study of All Wales and North West of England Twins (CaStANET)[6] from the same geographical location as CLASS. Only one twin from a pair was included. Comparison data were obtained concurrently to CLASS T_2 assessments. The CaStANET prevalence rates for adolescent cigarette smoking and alcohol use were comparable with other UK studies, and equal prevalence of questionnaire-assessed ADHD and conduct disorder behaviours were observed.[7,8]

Clinical assessments and outcome measures

ADHD

At baseline and follow-up, the parent version of the Child and Adolescent Psychiatric Assessment (CAPA),[9] a semi-structured research diagnostic interview, was used to assess DSM–IV and ICD–10[10] ADHD symptoms. The Child ADHD Teacher Telephone Interview (ChATTI)[11] was used to assess symptoms and impairment at school. Teacher reports were unavailable for those who had left school at T_2. To avoid excluding data from older individuals, in these cases parental report of impairment in school or community settings was used to assess symptom pervasiveness. Interviews were conducted by trained, supervised psychology graduates and child psychiatrists, and perfect interrater diagnostic reliability was found; $\kappa = 1.00$ for DSM–IV ADHD and ICD–10 hyperkinetic disorder. Because all individuals had baseline ADHD diagnoses and we were interested in predicting the change in these symptoms over time, we calculated an ADHD symptom change score (DSM–IV total ADHD symptom score at

T_2 minus ADHD total symptom score at T_1). For ease of interpretation, this score was transformed to reverse the outcome (so a greater decrease in ADHD symptoms – more change – resulted in a higher score).

Antisocial behaviour

The CAPA–parent version[9] was used at both time points to assess clinical symptoms of antisocial behaviours characterising DSM–IV oppositional defiant disorder and conduct disorder.

At follow-up, adolescents were also interviewed using the CAPA–child version.[12] Adolescent antisocial symptoms were considered present if reported by either the parent or adolescent within the past 12 months.[13] For our adolescent conduct disorder outcome measure, we investigated T_2 total conduct disorder scores. Where significant association was observed between adolescent conduct disorder scores and our putative predictor variables, we controlled for baseline conduct disorder symptoms. At T_1, 18 individuals (14.5%) met diagnostic criteria for conduct disorder.

Impairment of functioning

The effect of symptoms on family, school or peer relations was assessed using the CAPA.[9,12] Impairment was assessed for each type of disorder described by interviewees as interfering 'never', 'rarely', 'sometimes' or 'often'. Ratings of 'sometimes' and 'often' were taken as indicating significant impairment.

Adolescent substance use

Adolescent lifetime and current use of alcohol, cigarettes and cannabis was assessed using self-reported questionnaire measures from the 'Add Health' study.[14]

Other measures

Child and parent CAPA interviews provided information on police contact resulting from the child's behaviour (as opposed to being the victim of, or reporting, a crime) as lifetime ever and during the previous 3 months (endorsed if reported by either informant). Full-scale IQ scores were obtained at T_1 using the Wechsler Intelligence Scale for Children (WISC–III) assessment.[15] The mean full-scale IQ score was 91.8 (s.d. = 12.6).

Data on social class were obtained at T_1 using standard parental occupation measures.[16] Families were split into those from high (classes 1 and 2), $n = 32$, 26.7%; medium (classes 3 skilled and non-skilled, and class 4), $n = 42$, 35.0%; and low social class (class 5 and unemployed or unclassified individuals), $n = 46$, 38.3%. Two individuals had missing data and four stated the armed forces as their occupation and, due to difficulties in classification, were not included in these analyses. Parents also reported on their children's birth weight and friendships. Responses could be classed as 'being normal' (21.7%), 'having some friends with some problems with friendships' (63.5%) or 'having no friends' (14.8%).

Finally, mothers reported on their own childhood ADHD symptoms aged 7–12 years using the Barkley Adult ADHD scale[17] and on their childhood conduct disorder symptoms (before age 18 years) using a presence/absence checklist of DSM–IV conduct disorder symptoms. Over half the sample had missing data on unavailable fathers, thus father symptoms were not examined.

Medication and service use

Service use related to mental health and medication was measured using the Children's Service Interview with the parent, asking about service use within the past year.[18] No information regarding medication adherence was obtained.

Statistical analysis

Chi-squared and t-test analyses were used to compare T_1 characteristics between those who did and did not participate at T_2 and to compare prevalence of substance use between the CLASS and CaStANET samples. Linear regression was used to investigate predictors of outcome. All analyses were performed using SPSS (Version 12) for Windows. The predictors investigated reflected maternal factors (childhood ADHD and conduct disorder symptoms) and social factors (friendships and social class) previously implicated as potentially important.[19] We additionally examined child IQ and birth weight, found to predict favourable outcomes in the largest treatment trial of ADHD (the MTA trial)[20] as well as child gender. Finally, we adjusted for age and prescribed medication.

Results

Participation rates at follow-up were 80.3%. No significant baseline differences were found between those who did and did not participate (Table 1). Some T_2 items were not completed by interviewees. Data on ADHD symptoms, diagnoses and impairment were available for 126 individuals, antisocial behaviour for 124 individuals and substance use for 117. There were no baseline differences between those with complete and incomplete T_2 data, apart from an older age at time of assessment in those without teacher information (data available from author on request).

ADHD continuity and associated impairment

The mean ADHD symptom change score was 2.91 (s.d. = 3.54) indicating symptom reduction over time. At T_2, the majority of adolescents ($n = 88$, 69.8%) continued to meet DSM–IV ADHD diagnostic criteria (combined, inattentive and hyperactive–impulsive subtypes) and ICD–10 criteria for hyperkinetic disorder ($n = 63$, 51%). A total of 70 individuals (68.9%) had the same DSM–IV subtype diagnosis as at T_1. Even among those not meeting full diagnostic criteria ($n = 33$), the majority ($n = 20$) still reported significant impairment from their remaining ADHD symptoms. Twelve (10%) of the sample showed insufficient symptoms and impairment for an ADHD diagnosis. All of these individuals had been prescribed stimulant medication at some point and four continued to take it at follow-up. The remaining eight (6% of whole sample) seem to have recovered both symptomatically and functionally. Finally, one individual reported symptoms that would meet diagnostic criteria, but without any impairment. Attention-deficit hyperactivity disorder symptom score change was not associated with the time between assessments ($\beta = 0.07$, $t = 0.81$, $P = 0.42$), suggesting symptom change is not just because of the length of time since diagnosis.

Antisocial outcomes

Conduct disorder and associated impairment

At T_2, 39 individuals (31.5%) met DSM–IV conduct disorder diagnostic criteria, a significant increase in those diagnosed at T_1 ($\chi^2 = 15.94$, $P < 0.001$). A total of 27 (21.8%) individuals developed conduct disorder between T_1 and T_2 (emergent conduct disorder) with 12 (66.7%) showing conduct disorder persisting between time points. This showed high continuity of conduct disorder diagnosis, although conduct disorder did desist for 6 (33.3%) individuals. A total of 79 (63.7%) individuals

Table 1 Descriptive statistics for time 1 (T_1) and time 2 (T_2) characteristics with comparison of T_1 data between those participating and not participating at T_2

	Participants (n = 126)	Non-participants (n = 31)	Participant/non-participant comparisons	
			Test statistic	P
T_1 data				
Male, n (%)	118 (94)	28 (87.5)	$\chi^2 = 0.12$ (d.f. = 1)	0.73
Socioeconomic status, low: n (%)	46 (38)	10 (32)	$\chi^2 = 0.12$ (d.f. = 2)	0.94
Conduct disorder diagnosis, n (%)	18 (14.5)	4 (12.5)	$\chi^2 = 0.20$ (d.f. = 1)	0.66
Oppositional defiant disorder diagnosis, n (%)	61 (48.4)	19 (61.3)	$\chi^2 = 1.41$ (d.f. = 1)	0.24
ADHD symptoms, mean (s.d.)	14.56 (2.35)	14.77 (2.69)	$t = 0.39$	0.70
T_2 data				
Age, years: mean (s.d.)	14.7 (1.7)	–	–	–
ADHD symptoms, mean (s.d.)	12.07 (4.94)	–	–	–
Conduct disorder symptoms, mean (s.d.)	1.99 (1.87)	–	–	–
Stimulant medication, n (%)				
Ever	117 (93)	–	–	–
Current	83 (66)	–	–	–
Currently in contact with CAMHS, n (%)	88 (70)	–	–	–

ADHD, attention-deficit hyperactivity disorder; CAMHS, child and adolescent mental health services.

showed no diagnosis of conduct disorder at either time point. The majority of individuals (62/85, 72.9%) not reaching the diagnostic threshold for conduct disorder at T_2 reported impairment due to conduct disorder symptoms. For those with a T_2 conduct disorder diagnosis, 31 (79.5%) met diagnostic criteria for childhood-onset conduct disorder, presenting with one or more symptoms before age 10 years. The mean number of conduct disorder symptoms at T_2 was 1.99 (s.d. = 1.87).

Specific antisocial behaviours

A substantial minority of individuals at follow-up engaged in serious antisocial acts, including using a weapon (15/124, 12%), committing a crime involving confronting the victim (9/124, 7%), breaking and entering (14/124, 11%), stealing (25/124, 20%), fire-setting (16/124, 13%) and vandalism (27/124, 22%), with 47% (59/124) reporting at least one of these behaviours. Over half reported at least one police contact (75/124, 61%) with first police contact prior to adolescence (mean age 10 years, s.d. = 2.6). Police contact in the past 3 months was reported by 33/124 (27%).

Adolescent alcohol, cigarette and cannabis use

The rates of substance use and comparisons with the CaStANET sample are provided in Table 2. The ADHD sample were more

likely to report heavier and more hazardous use of alcohol, more recent (within previous 30 days) use of cannabis and regular recent use of cigarettes. They were also significantly younger when they first tried cigarettes (11.0 v. 12.7 years, $t = -3.2$, $P = 0.002$) and cannabis (12.6 v. 13.8 years, $t = -2.4$, $P = 0.018$) but not alcohol (12.7 years v. 12.9, $t = -1.1$, $P = 0.3$). There was no association between any measure of substance use and baseline ADHD ($P > 0.1$) or conduct disorder severity ($P > 0.07$), with the exception of heavy recent use of cigarettes, which was more likely in individuals with greater baseline conduct disorder severity (odds ratio (OR) = 1.42, 95% CI 1.01–1.99, $P = 0.046$). Cannabis and cigarette use were associated with greater concurrent conduct disorder severity ($P < 0.018$).

Predictors of ADHD and antisocial behaviour at follow-up

At T_2, 63% of the sample reported being currently prescribed stimulant medication. Those prescribed stimulant medication had significantly more ADHD symptoms and rates of ADHD diagnosis than those no longer using medication.[21] Medication use was not significantly associated with T_2 conduct disorder diagnosis ($P = 0.22$) or conduct disorder symptoms ($P = 0.08$), binge drinking, cigarette or cannabis use ($P = 0.16$ to $P = 0.83$).

Mothers' mean number of childhood ADHD symptoms was 5.13 (s.d. = 4.88) and conduct disorder symptoms 2.91

Table 2 Rates of adolescent substance use and comparison data[a]

	CLASS sample (n = 117)	CaStANET sample (control) (n = 117)	χ^2	P
Alcohol use, n (%)				
Ever without parents knowledge	61 (52.1)	63 (53.8)	0.1	0.79
At least once a week	23 (19.7)	13 (11.1)	3.3	0.07
Binge drinking[b]	16 (13.7)	6 (5.1)	5.0	0.025
Cigarette use (whole cigarette), n (%)				
Ever	50 (42.7)	26 (22.2)	11.2	0.001
In the past 30 days	40 (34.2)	9 (18.4)	24.5	<0.001
Regularly (at least one a day)	29 (24.8)	5 (7.7)	19.6	<0.001
Cannabis use, n (%)				
Ever	30 (25.6)	20 (17.1)	2.5	0.11
In the past 30 days	19 (16.2)	8 (6.8)	5.1	0.024
Regularly (at least one a day)	11 (9.4)	6 (5.1)	1.6	0.20

CLASS, Cardiff Longitudinal ADHD Sample Study; CaStANET, Cardiff Study of All Wales and North West of England Twins.
a. Self-report information on substance use was not available for nine individuals.
b. More than five drinks in a row at least once a week.

(s.d. = 3.54). Child IQ, birth weight, friendship problems, social class and maternal childhood ADHD symptoms were not significantly associated with adolescent ADHD scores or diagnosis (Table 3). However, mother's childhood conduct disorder symptoms predicted adolescent ADHD scores (Table 3) even when additionally controlling for the child's IQ and baseline conduct disorder symptoms ($\beta = -0.21$, $t = -2.18$, $P = 0.03$).

Children's IQ and social class, but not birth weight, friendship problems or maternal childhood ADHD symptoms, were predictive of conduct disorder outcomes. Those with a higher social class and IQ had fewer conduct disorder symptoms (Table 4), an effect strongest for performance IQ (results available from the author on request). Maternal childhood conduct disorder symptoms also significantly predicted adolescent conduct disorder symptoms (Table 4). These findings remained significant controlling for baseline ADHD and conduct disorder symptoms (full-scale IQ $\beta = -0.17$, $t = -2.14$, $P = 0.05$; social class $\beta = -0.19$, $t = -2.21$, $P = 0.03$; maternal conduct disorder symptoms $\beta = 0.20$, $t = 2.14$, $P = 0.04$). These same factors also significantly predicted diagnosis of conduct disorder at follow-up (Table 4). All findings remained when multiple regression analysis including all significant variables concurrently was undertaken (details available from author on request).

Discussion

Continued ADHD and conduct disorder

The results of this 5-year prospective study show that despite clinical recognition, confirmation of diagnosis and freely available treatment, children with ADHD experience ongoing difficulties, particularly the persistence of ADHD symptoms and diagnoses,

serious antisocial behaviour, police contact and high-risk health-related behaviours. Medication use for the treatment of ADHD was associated with increased ADHD severity, likely as a result of greater severity leading to continued treatment, but was not associated with other outcomes.

Even those not meeting full diagnostic criteria for ADHD at follow-up still showed substantial impairment due to their remaining ADHD symptoms. Only 10% of the sample appeared to have functionally and symptomatically recovered, some of whom continued to be prescribed medication. This is comparable with the low levels of functional remission reported in one US sample of children with ADHD[22] and in UK findings that hyperactivity in childhood predicts hyperactivity in adolescence.[23]

There are various potential explanations why ADHD and associated impairments persisted. First, ADHD is a chronic disorder and like other chronic conditions there may simply be a high level of stability over time, with affected individuals requiring ongoing monitoring and treatment. Second, inadequate treatment may be a contributory factor. Medication is known to be initially effective[24] but treatment requirements alter over time. Moreover, the treatment effect in those receiving medication through routine clinical care (compared with treatment trial medication provision) is known to be relatively poor[25] and treatment benefits appear to diminish or disappear over time.[3] This could also be the case in the UK. Third, participants may not have been taking their medication. Overall, the low level of remission and high continuing impairment as a result of ADHD in this study raise concerns, suggesting that long-term approaches to the management of ADHD need review by both clinicians and researchers.

Despite recognition of ADHD by specialist services and treatment, we also found a high percentage of individuals,

Table 3 Predictors of attention-deficit hyperactivity disorder (ADHD) symptom change over time (time 2 (T_2) – time 1 (T_1)) and DSM–IV ADHD diagnosis at follow-up

	Symptom change over time				T_2 DSM–IV ADHD diagnosis			
	R^2	β	t	P	Wald	OR	95% CI	P
Age (T_2)[a]	**0.14**	**0.38**	**4.41**	**<0.001**	2.70	0.80	0.62–1.04	0.10
Gender	0.01	0.11	1.27	0.21	1.17	0.40	0.07–2.13	0.28
Full-scale IQ	<0.001	−0.01	−0.14	0.89	1.46	0.98	0.95–1.01	0.23
Birth weight	0.003	−0.06	−0.65	0.52	1.80	1.00	1.00–1.01	0.18
Social class	<0.001	<0.001	<0.001	1.00	1.40	1.39	0.80–2.41	0.24
Friendship problems	0.006	−0.08	−0.85	0.40	0.07	1.10	0.54–2.23	0.80
Mother's childhood ADHD	0.07	−0.26	−1.63	0.11	0.44	1.04	0.93–1.15	0.51
Mother's childhood conduct disorder	**0.04**	**−0.21**	**−2.26**	**0.03**	**5.48**	**1.31**	**1.05–1.64**	**0.02**

Values in bold are significant at $P < 0.05$ level.
a. Adjusted for medication only; all others adjusted for medication and age at T_2.

Table 4 Predictors of time 2 (T_2) DSM–IV conduct disorder symptoms and diagnosis

	T_2 conduct disorder symptoms				T_2 DSM–IV conduct disorder diagnosis			
	R^2	β	t	P	Wald	OR	95% CI	P
Age (T_2)[a]	0.002	0.04	0.48	0.64	0.21	1.06	0.82–1.37	0.65
Gender	0.02	−0.13	−1.38	0.17	0.90	0.35	0.04–3.09	0.34
Full-scale IQ	**0.04**	**−0.21**	**−2.34**	**0.02**	**5.35**	**0.95**	**0.92–0.99**	**0.02**
Birth weight	0.01	−0.11	−1.21	0.28	1.04	1.00	0.99–1.00	0.31
Social class	**0.06**	**−0.24**	**−2.63**	**0.01**	**4.74**	**0.56**	**0.33–0.94**	**0.03**
Friendship problems	0.005	0.07	0.75	0.46	0.10	1.12	0.57–2.17	0.75
Mother's childhood ADHD	0.004	0.07	0.26	0.72	0.02	0.99	0.91–1.09	0.90
Mother's childhood conduct disorder	**0.08**	**0.29**	**2.99**	**0.004**	3.22	1.12	0.99–1.27	0.07

Values in bold are significant at $P < 0.05$ level.
a. Adjusted for medication only; all others adjusted for medication and age at T_2.

72.9%, impaired because of antisocial behaviour and a significant increase in the diagnosis of conduct disorder at follow-up (25.5%). A substantial number of serious antisocial acts were also committed. In UK community-based studies, 23% of adolescents (11–16 years) report ever being in trouble with the police[26] (61% in this study) and parental report of police contact in the past year is 6.6% for adolescent males[21] (v. 21% in 3 months for this study). This suggests that individuals with ADHD represent a high-risk group for serious antisocial behaviour, with police contact, and represent a significant cost to society as well as to the individual. Thus, any long-term clinical treatment of individuals with ADHD should include monitoring and interventions for antisocial behaviour and its impact, even at diagnostic subthreshold levels.

Substance use

There is growing evidence suggesting that childhood ADHD is a risk factor or precursor for later substance use, particularly cigarette use,[28] although not universally.[29] In our sample, adolescents were more likely to have tried cigarettes, started smoking at a younger age and become regular smokers than a community sample. Given that drinking alcohol in early adolescence is now considered normative in the UK,[30] it is perhaps unsurprising that there were no observed differences in the age of alcohol initiation. However, adolescents with childhood ADHD were more likely to report binge drinking[30] at least once a week over the past year and are therefore at high risk of hazardous alcohol use. Adolescents with ADHD were no more likely to have ever tried cannabis but indicated more recent use, as well as starting experimenting at an earlier age (associated with greater liability for misuse and psychosis).[31,32] Adolescent cigarette smoking, heavy alcohol and cannabis use have established risk effects on physical and mental health.[33] Our findings suggest the need for intervention regarding these behaviours in young people with ADHD.

These findings paint a bleak picture for those diagnosed with ADHD in childhood, especially as we have also shown that a substantial proportion of these children were recognised as having special educational needs and needed additional school support.[21] Future studies investigating the outcome of individuals with ADHD might wish to take into account additional outcomes such as quality of life or coping, which might provide additional information on this group.

Predictors of outcome

In line with previous studies and expectations, symptoms significantly decreased with age. We also examined child IQ and birth weight given a previous report of their importance.[20] Overall, there were no strong predictors of ADHD behaviour. The exception was maternal childhood conduct disorder symptoms, although maternal ADHD symptoms in childhood were not significant. The latter lack of association might be because of the use of retrospective self-reports of ADHD, which may be less reliable than reports from informants who knew the adult as a child.[34] These findings indicate that obtaining a history of the mother's own childhood conduct symptoms may be one useful marker of poorer outcome in addition to the child's own clinical presentation.[35]

In the MTA trial, individuals with the most consistent pattern of ADHD symptoms decline over 36 months[20] had higher IQ, were heavier at birth, showed higher baseline social functioning and lower social adversity. None of these factors were associated with a decline in ADHD symptoms in our sample. However, in the MTA trial, these factors were predictive of membership of a latent class with consistent decline in symptoms, whereas other latent classes had different patterns of decline. As we did not examine latent classes within our sample, we may have missed these subtle differences. Moreover, in our UK sample, there were few individuals with higher IQ and from higher social classes.

In the MTA trial, the time between initial and follow-up assessments was fixed, whereas in this study there was variation in the length of follow-up. The MTA trial can therefore be more exact regarding the effects of time. However, in our study, time between assessments was not associated with ADHD symptom change.

It is possible that factors not investigated here may be predictive of adolescent outcome. For example, negative parenting factors influence outcomes of other disorders, but for ADHD these appear to be a consequence of the disorder.[36] Research on other disorders suggests that a positive family history may also indicate those with poorer outcomes.[37]

In contrast to our lack of predictors for ADHD persistence, we did find predictors of adolescent conduct disorder symptoms, specifically maternal childhood conduct disorder, social class and higher IQ, the latter finding previously reported in those with and without a childhood diagnosis of ADHD.[38,39]

Limitations

As with nearly all longitudinal studies there was sample attrition. However, the rates of successful tracing (95%) and engagement in the second assessment were good and there were no baseline differences between those followed up and those lost to the study. Our study design did not include any measures of individual's medication adherence, therefore we can only comment on the prescription, rather than actual taking, of stimulant medication.

We were also unable to examine paternal externalising symptoms. We had no measure of current maternal conduct disorder symptoms. However, a proxy measure of current anti-social behaviour (mother reports of being in trouble with the law in the past 5 years) was not found to be associated with child outcomes, although few mothers endorsed this item ($n = 9$).

As with any study, these finding require replication, and multiple testing could give rise to false positive results. Finally, this sample represents children with ADHD that has been recognised and diagnosed by specialist clinical services in the UK. Our findings, therefore, cannot be generalised to those in the community whose ADHD has not been identified. However, as the aims of this study were to investigate the outcomes of children with diagnosed ADHD, our findings remain clinically informative.

The results of this UK prospective study suggest adolescents who were clinically recognised, treated and met diagnostic criteria for ADHD in childhood show poor adolescent ADHD and anti-social outcomes. The findings are consistent with some US studies (e.g. Barkley et al, Biederman et al and Mannuzza et al)[40–42] and demonstrate that the earlier findings generalise to another country and a different healthcare context. Even after widespread publication and dissemination of treatment guidelines, ADHD leads to a range of poor clinical outcomes. Moreover, there is a wider health risk in terms of high-risk patterns of cigarette smoking, alcohol and drug use.

There was some evidence that maternal childhood conduct disorder symptoms were associated with child ADHD and conduct disorder symptom outcomes, although the nature of this relationship requires further elucidation. However, we found no other significant predictors of ADHD continuity from baseline characteristics. This paints a bleak picture for clinicians in the field indicating that more research into other possible predictors and interventions effective in the long term is needed. However, social

and maternal factors, as well as child IQ may be important in protecting children with ADHD from developing adolescent antisocial behaviour.

Kate Langley, PhD, **Tom Fowler**, PhD, Department of Psychological Medicine and Neurology, School of Medicine, Cardiff University, UK; **Tamsin Ford**, MRCPsych, PhD, Child Health Group, Peninsula Medical School, University of Exeter, UK; **Ajay K. Thapar**, MD, MRCGP, **Marianne van den Bree**, PhD, Department of Psychological Medicine and Neurology, School of Medicine, Cardiff University, UK; **Gordon Harold**, PhD, University of Otago, Dunedin, New Zealand; **Michael J. Owen**, FRCPsych, FMedSci, PhD, **Michael C. O'Donovan**, FRCPsych, PhD, **Anita Thapar**, FRCPsych, PhD, Department of Psychological Medicine and Neurology, School of Medicine, Cardiff University, UK

Correspondence: Kate Langley, Department of Psychological Medicine and Neurology, School of Medicine, Cardiff University, Heath Park, Cardiff, CF14 4XN, UK. Email: langleyk@cardiff.ac.uk

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