

Hyperactivity and conduct disorder: exploring origins

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Abstract

Objectives: This paper explores the relationship of hyperactivity (HA), conduct disorder (CD) and combined hyperactivity and conduct disorder (HACD) with certain environmental and biological stresses and vulnerabilities.

Method: It is based upon a large epidemiological database from the North of England.

Results: The findings suggest that CD is uncommon and strongly related to environmental stresses. This is true to a lesser extent of HACD. While both CD and HACD were related to family adversity and adverse styles of parental discipline, subtly different patterns of associations are also evident. In particular, CD is linked with poverty, parental violence and contact with child care social agencies. These findings are consistent with the hypothesis that HA contributes to a pattern of confrontation and punishment associated, in some cases, with the emergence of a more complex combination disturbance. However, CD occurs against a background of family conflict and poor child-care.

Conclusions: Most apparent cases of conduct disorder are in fact hybrid conditions including symptoms of HA and CD. True CD should be diagnosed not only by positive symptomatology but also by the absence of hyperactivity symptoms.

Keywords: Conduct disorder; Hyperactivity; Stress; Environmental; Biological; Childhood.

Problem behaviour in childhood has been always a perturbing issue in organised human society.¹ However, attempts to use systematic classification and formal terminology to describe problem childhood behaviour are of relatively recent origin. For instance, the term conduct disorder appears to date only from early last century.² Hyperactivity is often regarded as a distinct form of troubled childhood behaviour³ and as a clinical concept may be of even more recent origin.⁴

The meaning of conduct disorder has evolved since its early description as a disorder of instinctive behaviours.² In an abbreviated form of the current WHO definition, it is now "a repetitive and persistent pattern of dissocial, aggressive, or defiant conduct".⁵ Hyperactivity is superficially similar but refers to a dimension or a category of psychopathology characterised by both restlessness and

inattention.³ As a category it is central to the modern hyperkinetic disorder of ICD-10² and attention-deficit/hyperactivity disorder in DSM-IV.⁶ Together with conduct disorder, it represents one of the most widely diagnosed disruptive behaviour disorders of childhood.

Within the recent past, some authorities doubted the justification for separating hyperactivity and conduct disorder.⁷⁻⁹ However, subsequent data mainly from clinical studies demonstrated associations between hyperactivity and clumsiness, accidents and language delays and between conduct disorder and problems of parenting.¹⁰⁻¹³ These findings gave rise to the view that hyperactivity is rooted in problems of maturation and development and conduct disorder related to social adversity.¹⁴ There is some corroboration for this view from non-clinical population studies.¹⁵ However, others have reported no differences between conduct disordered and hyperactive schoolchildren.¹⁶ Indeed, one major study showed few developmental, family relationship or social background differences between six and seven year old boys with symptoms of hyperactivity, conduct disorder, or symptoms of both combined.¹⁷

Early research proposed that behavioural syndromes akin to hyperactivity were characterised by 'minimal brain damage', often linked to perinatal adversity.¹⁸⁻²⁰ However, later work failed to demonstrate links between minimal brain damage and hyperactivity.²¹ More recently, brain-imaging studies have shown evidence of reduced right prefrontal cortical activation in hyperactive children and adolescents.²² Consistent with the timing and sequence of brain maturation²³ the authors argue that this represents a frontal dysmaturation in hyperactivity. However, there is also evidence of abnormal frontal cortical function among adults with antisocial personality disorder.²⁴ Since the latter is said to be the developmental sequel of conduct disorder rather than hyperactivity, these findings give little support to the unequivocal differentiation of the two conditions. In addition, there is evidence that conduct disorder as well as hyperactivity respond to stimulant medication.²⁵ Finally, conduct disorder without co-existing hyperactivity ('pure' conduct disorder) is rare in childhood, adding to the difficulty of investigating these disorders.¹²

Hence, despite considerable research, the differences between hyperactivity and conduct disorder are not necessarily robust and the validity of these distinctions requires continuing review and investigation. In the current study, we examine the hypothesis that hyperactivity shows an association with developmental adversity and conduct disorder with social and family adversity.

Method

In examining this question we made use of a large Newcastle data base obtained from screening

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representative samples of Newcastle children.²⁶

Multicriterion screen

The screen included, for all the children: the Rutter teacher scales²⁷ and sociometric indices;²⁸ for the older children, the neuroticism subscale of the Junior Eysenck Personality Inventory²⁹ and for the younger children the Young Group Reading Test.³⁰ Extreme scores on each measure were used as an indicator of deviance.

For the senior children, in order to maximise sensitivity, and to avoid excluding children with extreme scores on individual measures because of an insufficiently high summed score, weighting formulae were adopted which allowed children with markedly deviant teacher- or self-ratings to be selected on that basis alone.³¹ With the younger children the system was less complex. Identification by any one or more criteria was taken as indicating that the child might be 'at risk'.³² The original screening procedure for the older children had been aimed at identifying children who were actually maladjusted and that in the juniors, those 'at risk' as well as maladjusted.

Population studied

This consisted of approximately 3,300 11 and 12 year old senior school children and 1,040 seven and eight year old children. Screening yielded 322 screen-positive or 'maladjusted' senior school children (9.8% of the total population), 309 of whom had entered the original study;²⁶ these were considered to have a high probability of clinical disturbance. There was also a yield of 270 screen-positive junior school children (26.0% of the total population) and 265 of these had also entered the original study; these were considered disturbed or 'at risk' for disturbance.

Because of the organisation of data in the computer and because of the possibility of follow-up, it was decided that we should only include in our analyses those cases for which full data was available on follow-up. At that point in time there was contact with 95% of the cases, but more in the way of data was missing than subjects. The net result was that at follow-up 14% of the original senior cases were not included and 8% of the original juniors. A small number (one senior and three junior) of the original proformas could not be identified and on these it was not possible to gather data on hyperactivity. Full data was therefore potentially available on 263 senior and 241 junior children.

Missing data (and hence excluded subjects) were largely determined by incomplete parent scales. Our subsequent analyses suggest that the small percentage of missing rating scales is unlikely to have distorted the picture. Thus we have compared the original and current groups in terms of gender (55.5% v 55.2% males for seniors and 60.4% vs 60.2% males for juniors) and social class distribution and found the patterns very similar and this applied for both junior and senior groups.

Data on a small sample of screen-negative children who were labelled normal controls was also analysed. The latter were randomly selected from the residual pool of children who had scored less than three on the weighting system with the proviso that they were drawn from all of the six schools and that they reflected the sex ratio found in the group of screen-positives.

'Diagnosis' of hyperactivity

The 'diagnosis' of hyperactivity was based on the diagnostic algorithm employed in reanalyses of Isle of Wight and other data^{3,33} utilising information from the Rutter parent and teacher scales. By this approach, if a

Table 1: Sex distribution

Males n (%)		Screen positive				χ ²
	Screen negative	no HA/CD	HA only	CD only	HACD	
Juniors	30(56.6)	35(54.7)	48(56.5)	5/8	59(70.2)	ns
Seniors	33(52.4)	25(35.2)-	48(55.8)	4/11	69(74.2)+	p < 0.001

Group frequency significantly above + (p < 0.01) or below - (p < 0.01) that for all groups when tested by adjusted residuals

score of three or more on certain symptoms (items) was obtained from either one or both Rutter scales then he or she was designated as hyperactive. The symptoms were (i) 'very restless, often running about or jumping up and down' (ii) 'squirmy, fidgety child' (iii) 'cannot settle to anything for more than a few minutes'. These symptoms have consistently emerged as forming a distinct dimension on factor analysis of Rutter scales.^{3,33,34}

Conduct disorder

All the screen data, more detailed school based measures and data from a semi-structured parent interview,³⁵ were examined by a psychiatrist, who rated the children on clinical grounds as to severity of conduct disorder on a four point scale with moderate and marked equivalent to clinical levels of disturbance. This procedure was found to have satisfactory inter-rater reliability with r = 0.8935. Conduct disorder was indicated by the presence of symptoms such as tantrums, destructiveness, lying, stealing, truanting, fighting.^{26,35} This approach was similar to the ICD-9 diagnosis of conduct disorder³⁶ and this has not changed in a fundamental way.

Classification

The screen-positive children were classified into the categories 'no hyperactivity or conduct disorder' (no HA/CD), 'hyperactivity only' (HA), 'conduct disorder only' (CD) and 'combined hyperactivity and conduct disorder' (HACD).

Blind ratings

Though the clinical rating was not independent of the screen data, the main sources of information for clinical diagnosis were parent interviews concerning child behaviour and temperament. However, the assessing psychiatrists were blind to the screen status of the children. At the time of the original clinical rating, hyperactivity was not a focus of interest and hyperactivity data had not been abstracted from the Rutter questionnaires. Consequently, they were also blind to the presence of hyperactivity.

Definitions of variables

All information apart from intelligence and attainment data was assessed using semi-structured parental interviews, which have been shown to have satisfactory reliability.²⁶ The following have been employed in the current study:

- 1 *Index of organic risk*: this is a heterogenous category with the features including developmental delays (walking, speech, bladder control); head injury involving loss of consciousness, profound deafness or visual problems
- 1 *Low birth weight*: birth weight under the 10th percentile
- 1 *Index of social risk*: this index comprised items, 'voluntary' or 'compulsory' contact with social services

or other social welfare agencies, in relation to the child or siblings

- 1 **Social class:** the occupational strata followed the Registrar General's Classification of Occupations³⁷ and were converted into a five-point ordinal scale for statistical purposes. However, a sixth point was added representing long term unemployment (with few exceptions subjects defined in this way derived from previously unskilled and semi-skilled occupational strata)
- 1 **Parental relationships:** ratings were based on interviews covering separations, rows, fights or violence; and these were summated into an overall 'parental discord' index
- 1 **Parental management techniques:** ratings were made on four-point ordinal scales – nil, slight, moderate, or markedly present or used – of the extent to which parents made use of different forms of management techniques or discipline. These included the use of physical punishment, withdrawal of privileges, use of forms of isolation, and finally the use of reasoning. For statistical analysis purposes, the four point scale was reduced to two by combining the first and last two points of the scale.

Statistics

Differences between the five groups, screen-negative, no HA/CD, HA only, CD only and HACD were investigated using chi-squared tests and analysis of variance (ANOVA) where appropriate. Chi-squared tests were supplemented by calculation of adjusted residuals to identify cells where observed values differed significantly from expected values and ANOVA with the Newman-Keuls test to make allowance for the effect of multiple comparisons.

Findings

In both of the screen positive samples, the HACD group comprised the largest category, while the HA and 'neither HA nor CD' groups were intermediate. Children with CD, without symptoms of associated HA, were uncommon in both age groups (see Table 1). Among both junior and senior groups, there was a predominance of males in the HACD group and this reached significance among the older sample. Females predominated among screen positive older children without hyperactivity or conduct disorder (see Table 1).

There were no differences between any of the diagnostic groups in relation to evidence of organic risk or low birthweight and these data are not shown. There were no significant differences in 'physical punishment', 'withdrawal of privileges' or 'use of isolation' between any of the junior groups. However, parents of children with CD or HACD were significantly less likely than parents of other children to reason with their children. Parents of older children with CD reported significantly higher levels of 'physical punishment', and

'withdrawal of privileges' than those of the other groups. Those whose children had the combined HACD condition reported using high levels of all four disciplinary methods. Hence, for both age groups, the CD and HACD groups were linked with the greatest parental punishment (see Table 2).

Focusing on the quality of parental relationships, younger children with HA experienced low levels of parental 'rows' and of overall parental discord, compared to screen negative children (see Table 3). However, parents of children with HACD reported high levels of 'separations' and of overall discord; CD alone was linked with a violent partnership. Among the seniors, the pattern of results also suggested higher levels of overall parental discord associated with CD and HACD. This difference did not significantly discriminate between the screen positive groups: all four screen-positive groups experienced higher level of parental separations and overall discord than screen-negatives. In relation to inter-parental violence, HACD and also 'no HA/CD' were more exposed than screen-negatives only. However, for each measure, CD obtained a score either equivalent to HACD or higher, but perhaps due to the small numbers, this was not significantly different from other screen-positive groups.

Table 4 presents data concerning social adversity ('social risk'). Among the junior children, the data indicate that those with HACD and particularly CD experienced the greatest social adversity. Among the senior children, while all the screen-positive groups obtained higher mean scores on 'social risk' than screen-negatives, the most deviant scores were again obtained by CD, followed by HACD.

Discussion

The findings show a link between CD and parental violence, high levels of parental punishment, low social class and contact with social agencies. There is no such link with HA. This is consistent with findings of high levels of expressed emotion and of punitiveness in families of children with CD.¹⁰ Especially among younger children the data suggest that CD is likely to require the presence of the most extremely disrupted or stressed parenting. These views are most consistent with the more recent view of CD as a disorder linked with serious breakdowns of parent-child attachment of an order that is relatively uncommon.³⁹⁻⁴¹ It is important in clinical practice, especially with younger children, to be aware that CD is relatively rare.^{13,38} However, when it presents, CD is likely to be a marker for adverse experiences and strongly points to the quality of care, as a necessary focus of assessment.

HACD is linked with the same adversity, but to a lesser extent. There was no significant relationship of HA and any adversity. These

'ZYPREXA' (OLANZAPINE) REPUBLIC OF IRELAND ABBREVIATED PRESCRIBING INFORMATION: Presentation: Coated tablets containing 2.5mg, 5mg, 7.5mg or 10mg of olanzapine. The tablets also contain lactose. Velotab 5mg and 10mg orodispersible tablets. Velotab orodispersible tablet is a freeze dried, rapid-dispersing preparation to be placed in the mouth or alternatively to be dispersed in water or other suitable beverage for administration. Velotabs also contain aspartame, mannitol and parahydroxybenzoates. **Uses:** Schizophrenia, both as initial therapy and for maintenance of response. **Further Information:** In studies of patients with schizophrenia and associated depressive symptoms, mood score improved significantly more with olanzapine than with haloperidol. Velotab orodispersible tablets are bioequivalent to olanzapine coated tablets, with a similar rate and extent of absorption. They have the same dosage and frequency of administration as olanzapine coated tablets. Olanzapine orodispersible tablets may be used as an alternative to olanzapine coated tablets. **Pharmacodynamics:** Olanzapine was associated with significantly greater improvements in both negative and positive schizophrenic symptoms than placebo or comparator in most studies. **Dosage and Administration:** 10mg/day orally, as a single dose without regard to meals. Dosage may subsequently be adjusted within the range of 5-20mg daily. An increase to a dose greater than the routine therapeutic dose of 10mg/day is recommended only after clinical assessment. **Children:** Not recommended under 18 years of age. **The elderly:** A lower starting dose (5mg/day) is not routinely indicated but should be considered when clinical factors warrant. **Renal and/or hepatic impairment:** A lower starting dose (5mg) should be considered. In moderate hepatic insufficiency, the starting dose should be 5mg, and only increased with caution. When more than one factor is present which might result in slower metabolism (female gender, elderly age, non-smoking status), consideration should be given to decreasing the starting dose. Dose escalation should be conservative in such patients. **Contra-indications:** Known hypersensitivity to any ingredient of the product. Known risk of narrow-angle glaucoma. **Warnings and Special Precautions:** Caution in patients with prostatic hypertrophy, or paralytic ileus and related conditions. During antipsychotic treatment, improvement in the patient's clinical condition may take several days to some weeks. Patients should be closely monitored during this period. Caution in patients with elevated ALT and/or AST, signs and symptoms of hepatic impairment, pre-existing conditions associated with limited hepatic functional reserve, and in patients who are being treated with potentially hepatotoxic drugs. As with other neuroleptic drugs, caution in patients with low leucocyte and/or neutrophil counts for any reason, a history of drug-induced bone marrow depression/toxicity, bone marrow depression caused by concomitant illness, radiation therapy or chemotherapy and in patients with hypersensitivity conditions or with myeloproliferative disease. Thirty-two patients with clozapine-related neutropenia or agranulocytosis histories received olanzapine without decreases in baseline neutrophil counts. Rare cases reported as NMS have been received in association with olanzapine. If a patient develops signs and symptoms indicative of NMS, or presents with unexplained high fever without additional clinical manifestations of NMS, all antipsychotic drugs, including olanzapine, must be discontinued. Caution in patients who have a history of seizures or are subject to factors which may lower the seizure threshold. If signs or symptoms of tardive dyskinesia appear, a dose reduction or drug discontinuation should be considered. Caution when taken in combination with other centrally acting drugs and alcohol. Olanzapine may antagonise the effects of direct and indirect dopamine agonists. Postural hypotension was infrequently observed in the elderly. However, blood pressure should be measured periodically in patients over 65 years, as with other antipsychotics. As with other antipsychotics, caution when prescribed with drugs known to increase QTc interval, especially in the elderly. In clinical trials, olanzapine was not associated with a persistent increase in absolute QT intervals. Hyperglycaemia or exacerbation of pre-existing diabetes has been reported in very rare cases during Zyprexa treatment. In some cases, a prior increase in body weight has been reported, which may be a predisposing factor. Appropriate clinical monitoring is advisable in diabetic patients and in patients with risk factors for the development of diabetes mellitus. **Interactions:** Metabolism may be induced by concomitant smoking or carbamazepine therapy. **Pregnancy and Lactation:** Olanzapine had no teratogenic effects in animals. Because human experience is limited, olanzapine should be used in pregnancy only if the potential benefit justifies the potential risk to the fetus. Olanzapine was excreted in the milk of treated rats but it is not known if it is excreted in human milk. Patients should be advised not to breast feed an infant if they are taking olanzapine. **Driving, etc:** Because olanzapine may cause somnolence, patients should be cautioned about operating hazardous machinery, including motor vehicles. **Undesirable Effects:** The only frequent (>10%) undesirable effects associated with the use of olanzapine in clinical trials were somnolence and weight gain. Occasional undesirable effects included dizziness, increased appetite, peripheral oedema, orthostatic hypotension, and mild, transient anticholinergic effects, including constipation and dry mouth. Transient, asymptomatic elevations of hepatic transaminases, ALT, AST have been seen occasionally. Olanzapine-treated patients had a lower incidence of parkinsonism, akathisia and dystonia in trials compared with titrated doses of haloperidol. Photosensitivity reaction, rash or high creatine phosphokinase were reported rarely. Rare reports of hepatitis, priapism, seizures, hyperglycaemia or exacerbation of pre-existing diabetes have been received. Rare cases reported as NMS have been received in association with olanzapine. Plasma prolactin levels were sometimes elevated, but associated clinical manifestations were rare. In most patients, levels returned to normal ranges without cessation of treatment. Haematological variations, such as leucopenia and thrombocytopenia, have been reported occasionally. **For further information see summary of product characteristics.** **Marketing Authorisation Numbers:** EU/1/96/022/002 EU/1/96/022/004 EU/1/96/022/006 EU/1/96/022/009 EU/1/96/022/010 EU/1/99/125/001 EU/1/99/125/002. **Date of Preparation or Last Review:** February 2000. **Full Prescribing Information is Available From:** Eli Lilly and Company Limited, Dextra Court, Chapel Hill, Basingstoke, Hampshire, RG21 5SY. Telephone: Basingstoke (01256) 315000 or Eli Lilly and Company (Ireland) Limited, 44 Fitzwilliam Place, Dublin 2, Republic of Ireland Tel: Dublin 6614377. 'ZYPREXA' and 'VELOTAB' are Eli Lilly and Company Limited trademarks. **References:** 1. Zyprexa Product Monograph. 2. Casey DE J Clin Psychiatry 1997; 58(suppl10): 30-33. 3. Data on file, Eli Lilly & Company. 4. Tollefson GD et al. Arch Gen Psychiatry 1998; 55: 250-258. **website:** www.elililly.ie


 Zyprexa is manufactured in Cork.



Table 2: Parent management of children with no behaviour disorder, hyperactivity, conduct disorder or both: percentage of parents using method

		Screen negative	Screen positive				χ^2
			No HA or CD	HA	CD	HACD	
Juniors	n =	53	64	85	8	84	
Low use reasoning		32.1	34.4	34.1	75+	52.4++	p < 0.05
Seniors	n =	63	73	85#	11	92#	
Physical punishment		12.7	23.3	35.3	72.7+	46.7++	p < 0.001
Withdrawal of privileges		15.9	10.9	28.3	54.6+	33.7++	p < 0.001
Use isolation		15.9	26.0	36.5	45.5	40.3+	p < 0.05
Low use reasoning		23.8	43.8	40.0	36.4	51.1+	p < 0.05

missing case; +adjusted residual > 0.05; ++ adjusted residual > 0.01

Table 3: Mean scores (sd) on measures of parental relationship problems, composite social risk score, and percentage low socio-economic groups for screen negative and positive children with behaviour disorders

		Screen negative	Screen positive				ANOVA Neuman-keuls p < 0.05
		1	No HA/CD	HA	CD	HACD	
Juniors	n =	53	64	85	8	84	
Separations	m (sd)	1.7(0.9)	1.5(1.0)	1.6(0.9)	2.1(1.2)	1.9(1.1)	2,3v5
Rows	m (sd)	1.8(0.6)	1.5(0.7)	1.5(0.6)	1.6(0.9)	1.5(0.7)	1v3
Fights	m (sd)	1.2(0.4)	1.1(0.5)	1.1(0.2)	1.8(1.0)	1.3(0.6)	1,2,3,5v4
Overall discord	m (sd)	4.7(1.4)	4.1(1.5)	4.1(1.5)	5.5(2.2)	4.7(1.6)	2,3v5; 1v3
Seniors	n =	63	73	85#	11	92#	
Separations	m (sd)	1.2(0.5)	1.7(1.1)	1.7(1.1)	2.1(1.2)	1.9(1.1)	1v2,3,4,5
Fights	m (sd)	1.0(0.0)	1.2(0.4)	1.1(0.3)	1.2(0.6)	1.2(0.5)	1v2; 1v5
Overall discord	m (sd)	3.6(0.8)	4.3(1.5)	4.2(1.5)	4.9(1.6)	4.7(1.6)	1v2,3,4,5

findings are consistent with the hypothesis that HACD may be a hybrid of HA and CD.¹³ In addition, since HACD is common and CD rare, it is likely that many of the children receiving diagnoses of CD in current practice in fact have HACD. Hence, many apparently conduct disordered children will have coexisting hyperactivity, even if this does not fully satisfy criteria for, for instance, hyperkinetic or attention deficit hyperactivity disorder. This may go some way to explaining the apparent efficacy of stimulant medication among children diagnosed as conduct disordered. It may also explain the findings from brain imaging studies.²⁴ It is possible that most, if not all, of the antisocial adults in these studies will have had HACD, which compared to CD tends to persist.³³ It is possible that CD will tend to resolve because it is not associated with the underlying frontal cortical dysmaturity of HA.²²

Although the differences did not reach statistical significance, parents of children with HA did report use of physical punishment and less reasoning than among controls. These findings appear consistent with those of a recent study⁴² that showed links between HA and 'poor parent coping and the use of aggressive discipline

methods'. However, children with other disturbances had been excluded from that study. The data presented here indicate that such parenting practices, like parental discord, are also associated with being 'screen-positive' and are not specific to disruptive behaviour disorders. Nevertheless, as the authors concluded,⁴² "the problematic behaviours of hyperactive children... influence (parents') ability to parent effectively...". However, beyond a certain threshold, such stress upon parenting and upon attachment relationships, risks the development of CD among children predisposed by pre-existing HA (ie. to produce HACD).¹²

Other relevant studies have shown consistent results. Our findings are most comparable to those in which identification using symptom scores was linked with another indicator of 'caseness'. For instance, using definitions based on symptom persistence as well as patterns of symptoms, McGee *et al*⁴³ reported CD as most strongly associated with 'solo' mothers and parental separation, HACD as intermediate and the weakest association with HA. They are also similar to those of Schachar *et al*¹² who reported a high level of parental separation in relation to referred children with CD but not

Table 4: Social risk mean scores and percentage low socio-economic status among screen negative and screen positive 7-8 year old and 11-12 year old children

		Screen negative	Screen positive				ANOVA Neuman-keuls p < 0.05
		1	No HA/CD 2	HA 3	CD 4	HACD 5	
Juniors	n =	53	64	85	8	84	
Social risk	Mean (sd)	2.6(2.0)	2.7(2.2)	2.6(2.2)	5.5(3.1)	3.9(2.6)	1v4,5; 2v4,5; 3v4,5
†Low social class	%	30.2	34.4	31.7	75.0	32.1	ns
Seniors	n =	63	73	85#	11	92#	
Social risk	Mean (sd)	1.6(2.0)	3.5(2.6)	3.0(2.3)	5.8(2.3)	4.1(2.5)	1v2,3,4,5; 3v4; 2v4
†Low social class%	%	22.2	28.8	31.4	54.6	38.0	ns

† social class iv + v + unemployed

HACD nor HA.

There was no significant relationship between any behaviour disturbance and perinatal adversity, similar to the findings of Taylor *et al.*¹⁷ The finding is consistent too with the genetic rather than external trauma as the usual origin of the brain dysmaturity of HA (and HACD).

Due to the age of the data, the 'diagnoses' analysed here relied on diagnostic criteria not in current use. However, the concept of CD has not changed substantially over the intervening period. It is true that clinical judgement was not involved in the 'diagnosis' of HA, which relied on questionnaire data. However, there is considerable overlap in the validity and reliability of interview and questionnaire derived information.⁴⁴ The definition of HA included the additional criterion of being screen-positive and so both categories reflect a significant level of dysfunction. However, the cross sectional design does limit the degree to which causal inferences are valid.

The most important conclusion is the relative rarity of CD and when rigorously diagnosed, excluding HA symptoms, it is a marker for considerable psychosocial adversity, probably very impaired attachments. This will have considerable implications for assessment and management. HACD is much more common and should not be confused with the superficially similar CD. It is important to search out evidence of hyperactivity in children presenting apparently with conduct disorder, not least because of the potential benefit of stimulant medication.⁴⁵

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