Hyperactivity and its primary disorders in the childhood population

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There are nearly 30 population studies of childhood hyperactivity and its disorders in the scientific literature of the last two decades. Close reading reveals some consensus but at first sight there are discrepant findings between studies. There are a number of reasons for this.

DEFINITIONS OF HYPERACTIVITY

Hyperactivity is a word that is easily misunderstood. To some it means merely overactivity, to others excitability, yet again, as in this article, it can refer to *a quality of behaviour which is characterised by inattention, restlessness and impatience or impulsiveness*. Different interpretations of the term cause confusion in epidemiological studies. Although a common practice in the last decade has been to combine rating scales or criteria so that a syndrome of hyperactivity including at least inattention and overactivity is appraised, earlier work did not necessarily do so. Both features are necessary, the restlessness seen in hyperactive children is genuine motor overactivity, not simply excessive off-task behaviour (Porrino *et al.*,1983; Taylor *et al.*, 1991). Some studies also include features such as aggressive behaviour or emotional lability. The adoption of operational criteria for the diagnoses of the principal disorders of hyperactivity: attention deficit disorder (DSM-III), attention deficit/hyperactivity disorder (DSM-IV) and hyperkinetic disorder (ICD-10) has or will lead to greater consensus but the conceptual grounds for creating such disorder categories vary with time and culture.

The earlier American conceptualisation of hyperactive child or hyperactive reaction of childhood became operationalised as attention deficit disorder, with (ADD-H) or without hyperactivity in DSM-III. The criteria for ADD-H yielded high prevalence rates and there has subsequently been a progressive tightening of criteria in DSM-III-R and DSM-IV, particularly with the re-emphasis on overactive and impulsive problems, the presence of impairment and a requirement for symptoms to be evident in at least two sorts of situation. This should lead to an apparent fall in prevalence (see table of findings in Goldman *et al.*, 1988) though it is hypothesised that the emphasis on inattention since DSM-III should lead to more girls being identified (Berry *et al.*, 1985) and in practice the prevalence figures seem to have altered only a little (see below). Conversely the description of hyperkinetic syndrome of childhood in ICD-9, when operationalised in ICD-10 seems to provide a higher prevalence (Steinhausen & Erdin, 1991). Greater rapprochement between European and US usage of the primary hyperactivity disorder concept may result in further apparent shifts in prevalence.

Ascertaining the prevalence of hyperactivity (here taken to mean inattentive, impatient restlessness) will vary according to the methods used for detection. Many studies use rating scales which are cheap and can act as quantitative measures or be geared to diagnostic criteria. Semi-structured interviews with or without direct observational studies of the child are more laborious but have the advantage of being investigator-based rather than respondent-base which means they can be calibrated for reliability and validity with greater accuracy. In general terms they yield lower prevalence figures for diagnoses, particularly because impairment can be assessed.

Within-study variation shows that age is an important factor influencing rates of hyperactivity disorders. There is uncertainty about the distinction of hyperactivity from age-appropriate characteristics in the pre-school population but there is wide-

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spread confirmation that prevalence rates among 5-11 year-olds are higher than among teenagers.

Boys have invariably been shown to exhibit more hyperactivity and more hyperactivity disorders than girls. For broader definitions of hyperactivity the ratio among schoolchildren is 2-3:1. Narrower definitions and older age groups magnify the difference. Pervasive hyperactivity is particularly rare among teenage girls. It is possible, though not absolutely confirmed that the male excess is predominately because behavioural impatient overactivity, a quality skewed towards boys, comes through strongly in rating scale studies. Inattention has less impact on observers and the excess of inattentive boys over girls may be less marked (Berry *et al.*, 1985). Nevertheless it pays to be clear that a prevalence rate obtained for a population of boys is not the rate for the whole child population.

One of the most significant conceptual advances has been the recognition that hyperactivity which is manifest in several types of situation ('pervasive' hyperactivity) is a more robust construct and of more clinical relevance than hyperactivity which is confined to one situation such as the home. Pervasive hyperactivity endures longer and generally has a different set of correlates. What this means for sound epidemiology is that information has to be gathered from at least two situations, preferably from two different sources (e.g. teachers and parents independently). Indeed the research diagnostic criteria for hyperkinetic disorder demand this whilst the DSM-IV criteria for ADHD require symptoms to be manifest in more than one situation but will accept a single informant.

Compared with situational hyperactivity, pervasive hyperactivity is less common, more persistent, and more likely to be associated with educational failure, neurological deficits, developmental delays, lower intelligence, and attentional impairments (Gillberg & Gillberg, 1988; Taylor *et al.*, 1991; Schachar *et al.*, 1981; Goodman & Stevenson, 1989). It is as yet unresolved as to whether this is because pervasive hyperactivity is qualitatively distinct or whether it is a more severe variant of situational hyperactivity. There is certainly a gradient of severity from home-based, through classroom-based to pervasive hyperactivity (Goodman & Stevenson, 1989; McCardle *et al.*, 1995).

Without taking the matter further, it is apparent that the interpretation of epidemiological findings will vary according to diagnostic scheme, age range of population, ascertainment method, type of hyperactivity, and range of sources of information.

RATES

Hyperactivity itself is generally distributed within the population though the distribution curve is skewed to the right and there are, for instance, more children scoring more than two standard deviations above the norm on the Conners abbreviated teacher rating scale. This seems to be true for most developed countries (including Hong Kong) (Luk, 1996).

It can be a symptom of various conditions: anxiety, mania, pervasive developmental disorders and conduct disorder are clear examples. That there exist primary disorders of hyperactivity, separate from underlying conditions and demarcated from the major categories of childhood psychiatric disorder is not disputed. But the boundaries of such conditions are unclear. Currently two major constructs exist. The DSM-IV concept of attention deficit/hyperactivity disorder (ADHD) allows for the possibility of pathological inattentiveness *or* hyperactivity/impulsiveness. It envisages the possibility of a predominantly inattentive type, a predominantly hyperactive-impulsive type and a combined type in which symptoms of inattentiveness and overactivity/impulsiveness are both present to marked degree. There must be pervasiveness and impaired functioning. The combined type concept is therefore effectively a more tightly defined version of ADD-H. Hyperkinetic disorder (HD), on the other hand always requires *both* inattentiveness and overactivity to be present and, in the research criteria, impulsiveness too. ADHD is inevitably more prevalent than HD (Swanson *et al.*, 1998).

As far as older diagnostic criteria are concerned, Buitelaar & van Engeland (1996) in a major review of studies mainly from the 1980's suggested that these showed an overall prevalence of ADD-H in schoolchildren of approximately 4%, varying with age and locality. McCardle *et al.* (1995) also found a 3-4% rate using slightly different, but probably comparable, criteria to ADD-H. Esser *et al.* (1990) in a two-stage study found a prevalence of hyperkinetic syndrome (ICD-9) of 4.2% among 8-year-olds in Germany but 1.6% among 13 year-olds. All studies find hyperactivity diagnoses to be among boys than girls at all ages.

More recent studies on ADHD using DSM-III-R criteria have tended to yield comparable figures of around 4%: Pelham *et al.* (1992), Shaffer *et al.* (1996), Wolraich *et al.* (1996). An exception is the German study by Baumgaertel *et al.* (1995) which found 10.9% for DSM-III-R but 4.8% for DSM-

IV combined type which agrees with the estimated 3-5% prevalence for ADHD described in the text of DSM-IV (combined type is thought to constitute the majority of ADHD cases). The prevalence of HD is lower at about 1%; Taylor *et al.* (1991) in a London study using criteria similar but not identical to ICD-10 found a rate of 1.7% among 7-8 year-old boys, i.e. an overall population rate of just under 1%.

All in all it seems that a prevalence among pre-pubertal schoolchildren of about 4% for ADHD and 1% for HD would be widely accepted figures.

This means that the primary disorders of hyperactivity, however classified, are common conditions within clinic samples. Indeed it is often said that ADHD is the commonest childhood psychiatric disorder (e.g. Cantwell, 1996)

SUBTYPES

Within children who display impairing, long-standing pervasive hyperactivity, there are two versions of a primary disorder: ADHD and HD. ADHD is a massively supported concept internationally. It is widely appreciated that it is a heterogeneous condition, albeit one with a characteristically good response to stimulant medication. Yet improved on-task behaviour and reduced overactivity as responses to medication are also seen in aggressive and indeed normal children (Rapoport *et al.*, 1978; Taylor *et al.*, 1987) so treatment response cannot be the only validating factor in defining a disorder.

ADHD is already formally subdivided into three types: predominantly inattentive, predominantly hyperactive-impulsive, and combined. HD is broadly similar to ADHD combined type though is diagnosed according to more stringent criteria. It has a closer association with neuropsychological impairments. The criteria which support it have been quite widely thought to be those indicative of a single disorder (Barkley, 1982; Sergeant, 1988; Taylor *et al.*, 1991). It may therefore be a subtype of ADHD though how far it differs from ADHD combined type is unresolved.

Most researchers have found a proportion of children with a primary hyperactivity disorder who are also clinically anxious though not necessarily to the point at which the anxiety can be diagnosed as a comorbid disorder. These children do not respond nearly as well to stimulant medication and are more likely to experience adverse drug reactions (Tannock *et al.*, 1995; Taylor *et al.*, 1987).

Rather similarly, the co-existence of aggressive behaviour short of conduct or oppositional-defiant disorder seems to identify a group less responsive to stimulants (Matier *et al.*, 1992). This, and the former finding stimulated Jensen *et al.* (1997) to suggest that the co-existence of anxiety and aggressive disorders (sic) with ADHD represented two subtypes of ADHD since what was happening was an interaction with ADHD which modified it.

Whether attention deficit without impatient overactivity is a subtype of ADHD is unresolved. DSM-IV thinks so, Taylor *et al.* (1991) did not; attention deficit alone was associated simply with low IQ, delayed language development, low socio-economic status and 'poor housecraft' on the part of the parents. It was not associated with poor attention on laboratory tests once IQ had been allowed for. It is not a precursor of ADHD but it just might be a consequence since the natural history of ADHD in clinic samples is for overactivity to subside leaving problems of attention, self-organisation and impulsiveness in its wake (Biederman *et al.*, 1995a; Wender, 1995).

By definition, according to classification scheme rules, inattentive restlessness in pervasive developmental disorders is not ADHD. This is a convention with some evidence to support it (Taylor, 1986) but the position is not yet absolute.

According to diagnostic rules, ADHD and HD have to have been present from an early age. Yet the development of a hyperactivity disorder following encephalitis or brain injury has been clinically recognised and is now beginning to be documented among clinical samples (Max *et al.*, 1998).

COMORBIDITY

If the above suggestions about subtypes were to be adopted the resulting matrix of possibilities would be complex enough. But the position with respect to comorbidities complicates matters further. If impairing disorders (including developmental impairments) are examined, then it appears that between a half and three-quarters of ADHD cases also meet diagnostic criteria for other conditions which are thus comorbid. The comorbidity data is almost exclusively available for ADHD rather than HD because DSM and the diagnostic research instruments derived from it support multiple clinical diagnoses whereas ICD encourages parsimony.

Conduct disorder (here taken as an overall term

including oppositional-defiant disorder) develops in most cases of ADHD. The hyperactivity disorder comes first. Children who are both hyperactive and antisocial are more severely hyperactive and more severely conduct disordered than either disorder alone (Taylor *et al.*, 1991). This is reflected in the category of hyperkinetic conduct disorder in ICD-10 but as far as I can tell this category does not seem to have been very widely accepted in practice.

Approximately 20% of cases of ADHD will also fulfil criteria for mood disorder and 25% for anxiety disorder (Biederman *et al.*, 1991; Jensen *et al.*, 1993).

About 20% of ADHD children with ADHD also have specific learning disabilities including literacy problems, developmental language disorders and motor planning disorder (Jensen *et al.*, 1997).

Only about 7% of children with ADHD have tic disorders including Tourette's syndrome but some 60% of Tourette's syndrome have ADHD (Goldman *et al.*, 1998).

The above figures do not specify the co-occurrence of more than two conditions in any one child yet it is clear, clinically, that this happens. It is of course possible that apparent comorbidity is actually a deficiency of a classification system or a diagnostic instrument. Measures or conceptual boundaries may spuriously distinguish between constructs which are actually interwoven (see Tannock, 1998).

Nevertheless the sheer scale of comorbidity in a condition which has relatively weak aetiological associations with social disadvantage and family dysfunction raises interesting questions as to why it should be such an apparent vulnerability factor for so much psychopathology.

PSYCHOSOCIAL VARIABLES

Although the generally prevalent view is that the primary hyperactivity disorders of childhood are essentially biological and genetic, inheritance is far from 100% and there is evidence for the impact of social factors. Most of the studies examining possible social factors have drawn from clinic samples rather than a general population frame and it is known that clinic-referred hyperactive cases are different; the children are more likely to have comorbid problems and peer group difficulties and the parents are more likely to be depressed, exhibit a mixture of indulgent and aggressive parenting styles, be insensitive and negative to their child and cope less well with parenting (Woodward *et al.*, 1997). It is al-

so the case that in published work the distinction between pervasive hyperactivity, hyperactivity disorders and co-existing hyperactivity and aggressive behaviour has not always been clear. Not surprisingly there are inconsistencies in the data. Social disadvantage is sometimes associated with hyperactivity disorders but not always. Maternal depression and family discord likewise. Nevertheless, for pure, non-comorbid HD no psychosocial factor has been repeatedly identified as being a primary cause.

Although not essentially epidemiological, several studies of selected samples have indicated that psychosocial factors are likely to perpetuate pre-existing hyperactivity (e.g. Barkley *et al.*, 1991; Campbell & Ewing, 1990). Epidemiological work which has not been tightly focussed on hyperactivity has also suggested the same (McGee *et al.*, 1984) and the general thrust of such findings has been to suggest that this comes about through disturbance of parenting. Work with a whole population of London boys, found some evidence from a short-term follow up (9 months) that suggested persistent hyperactivity to be associated with family adversities which in turn were associated with conduct disorder (Taylor *et al.*, 1991). Negative family relationships, maternal depression, high levels of expressed criticism of the child by the mother, and a history of marital breakdown constituted the family adversities. An echo of this is found in Woodward *et al.* (1998) study of hyperactive boys (equivalent to DSM-III-R ADHD) drawn from a community sample. After statistically adjusting for the influence of mental health problems in the primary caregiver and conduct disorder symptoms in the children, poor parental coping and aggressive parenting style continued to correlate with hyperactivity. Parental coping referred to anticipation of problems, using effective strategies to improve child behaviour, monitor these and adapt accordingly. Aggressive parenting was shouting at the child, losing their temper with him and using physical punishment. The strong suggestion is therefore that parenting problems perpetuate hyperactivity.

It is sometimes said that the family discord is the result of having a disruptive child rather than vice versa. There is some truth in this as medication studies have shown. Yet one should not go too far. Clinic based studies (e.g. Marshall *et al.*, 1990) indicate that parental behaviours such as high EE predict parental behaviour on follow-up whereas child aggressive behaviour does not (even though it predicts child aggressive behaviour).

LONG-TERM COURSE

It is increasingly accepted that the principal features of ADHD can persist into adult life. Follow-up studies are complicated because the rules for the initial diagnosis of a primary hyperactivity disorder change with time. Studies for which a cohort was defined 25 years ago would have been initially diagnosed under non-operationalised DSM-II and would have unashamedly included comorbid aggressive behaviour.

It is clear that the rate of hyperactive-impulsive symptoms declines with age but this is not so obvious for inattentive symptoms (Biederman *et al.*, 1995a). Wild claims are made for persistence with the occasional claim that the diagnosis of ADHD is an indication for lifelong medication. It seems clear that a minority of children with ADHD will continue to have inattentive problems in adult life but the size of that minority cannot be estimated with accuracy. In many ways the best follow-up study is that published by Mannuzza *et al.* (1993) but on follow-up information was not gathered from the young men's relatives or partners which would almost certainly lead to under-reporting and the suggested 8% prevalence of ADHD at age 26 might need doubling (personal communication cited in Wender, 1995 p.69). Persistence beyond young adulthood into middle age is poorly documented but several studies have attempted to recruit adult ADHD sufferers through advertisements and special clinics (Wender, 1995) or assessment of relatives of children with ADHD (Biederman *et al.*, 1993) and the subsequent application of standard diagnostic criteria. This cannot yield a prevalence figure but it does enable subjective symptoms of ADHD to be tapped; something it is hard to do with children. What emerges is the possibility that emotional lability is marked and may be the key problem for adult sufferers. This in turn may indicate a different range of drugs to be employed rather than those justified by behaviour change in children.

An important issue revealed by follow-up studies of a clinic sample is the high rate of comorbid or consequential problems that persist as equal or greater problems than the original hyperactivity disorder. Antisocial behaviours, educational and marital failure are well recognised. To these should be added substance misuse (Biederman *et al.*, 1995b). There is no indication that this is related to the prescription of potentially addictive stimulant drugs; indeed methylphenidate dependence is so rare as to be virtually non-existent in treated hyperactive children. Yet to be sure, the subject requires further exploration in whole population studies.

CODA

It is now clear that cases referred to clinics are not typical of community cases, that high levels of comorbidity confound simple statements unless co-existent disorders are specified, and that the conceptual boundaries around primary hyperactivity disorders are elastic, varying with history and culture. In the longer term comorbid and associated problems seem to be the major problem and could prove extremely expensive for society. It may be that they are potentially preventable. The whole subject of the correlates of hyperactivity and its disorders deserves more studies using a whole population approach in order to account for such potential biases.

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