



## Continuity of aggressive antisocial behavior from childhood to adulthood: The question of phenotype definition<sup>☆</sup>

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### ABSTRACT

Aiming to clarify the adult phenotype of antisocial personality disorder (ASPD), the empirical literature on its childhood background among the disruptive behaviour disorders, such as attention deficit/hyperactivity disorder (AD/HD), oppositional defiant disorder (ODD), conduct disorder (CD), or hyperkinetic conduct disorder (HKCD), was reviewed according to the Robins and Guze criteria for nosological validity. At least half of hyperactive children develop ODD and about a third CD (i.e. AD/HD + CD or HKCD) before puberty. About half of children with this combined problem constellation develop antisocial personality disorder (ASPD) in adulthood. Family and adoption/twin studies indicate that AD/HD and CD share a high heritability and that, in addition, there may be specific environmental effects for criminal behaviours. “Zones of rarity” delineating the disorders from each other, or from the normal variation, have not been identified. Neurophysiology, brain imaging, neurochemistry, neurocognition, or molecular genetics have not provided “external validity” for any of the diagnostic categories used today. Deficient mental functions, such as inattention, poor executive functions, poor verbal learning, and impaired social interaction (empathy), seem to form unspecific susceptibility factors. As none of today’s proposed syndromes (e.g. AD/HD or psychopathy) seems to describe a natural category, a dimensional behavioural phenotype reflecting aggressive antisocial behaviours assessed by numbers of behaviours, the severity of their consequences and how early is their age at onset, which will be closely related to childhood hyperactivity, would bring conceptual clarity, and may form the basis for further probing into mental, cognitive, biological and treatment-related co-varying features.

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### 1. Introduction

That problem behaviours in children may herald psychosocial problems in adult life is basically a universal insight and the mainstay of most educational efforts. The association has also been demonstrated in a number of longitudinal studies and forms the nucleus in phenotype definitions of adult impulsive behaviours, physical aggression, violation of societal norms, and deficient emotional reactions, that is antisocial personality disorder (ASPD, American Psychiatric Association (APA), 1994), dissocial personality disorder (ICD-10, World Health Organization, 1993) or psychopathy (Hare, 1980). Nevertheless, the nosological categories proposed to capture specific problem constellations both overlap and are heterogeneously defined.

Attention-Deficit Hyperactivity Disorder (AD/HD) is an umbrella term by definition consisting of three problem domains, inattention,

hyperactivity and impulsivity, listed in two separate sets of criteria that may be met individually or together. Two persons who both have this diagnosis may theoretically not share a single criterion. The International Classification of Diseases, tenth edition (ICD-10, WHO, 1993) has based its corresponding definition on hyperactivity (Hyperkinetic Disorder), noting attention deficits as a common complication. If hyperkinesia is combined with outright antisocial behaviours, the diagnosis of hyperkinetic conduct disorder (HKCD) may be made. In the DSM-IV, Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) are instead treated as two separate disorders.

Other diagnostic categories that have been implicated in the context of childhood aggressive behaviours are the autism spectrum disorders (ASD), describing deficits in social interaction or “empathy”, verbal and/or non-verbal communication and flexibility, and “paediatric mania” or bipolar disorder with irritable, elated mood swings. A brief overview of the current diagnostic definitions that may be related to early-onset antisocial behaviours provided by the DSM-IV and the ICD-10 is given in Table 1.

Assessing the validity of diagnostic concepts in psychiatric nosology is a continuous process, where, in the absence of knowledge about specific aetiological factors, definitions have to be regarded as preliminary and subject to revision. A seminal paper by Robins and

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**Table 1**  
Currently used diagnostic definitions for childhood-onset behavioural disorders.

Diagnostic categories	Diagnostic code (DSM-IV and ICD-10)	Age at onset	Problem dimensions	Definitional context (cf. temperament as patterns of reactions to stimuli/percepts)
AD/HD	314.01 F90.0	0 years	Hyperkinesia	Behaviours in face of situations demanding motor activity control
ASD	299.00 F84.0, 5, 9	0 years	Social interaction / Communication	Behaviours, cognitions and emotions in relation to others
AD/HD	314.01 F90.0	4 years	Impulsivity	Behaviours in conversations and queues
ODD	313.81 F91.3	4 years	Opposition	Emotional expressions and behaviours in face of other people
AD/HD	314.00 F90.0	~6 years	Inattention	Behaviours in face of situations demanding attention and executive functions (school work)
Mania	296.0x F30.1	?	Agitation, irritability, elation, grandiosity	Dysregulated behaviours related to unstable mood
CD	312.81 F91.0–2	~4.5–5 years	“Criminality”	Behaviours in contradiction with norms and regulations

**Table 2**  
Criteria for diagnostic validity.

According to Robins and Guze (1970) and Andreasen (1995)
1. Clinical description (including unique symptoms that do not occur in other disorders, sex, age, precipitating factors, response to various forms of treatment).
2. Laboratory studies identifying biological or other so called “markers” for the disorder. In addition, Andreasen proposed “external” validators from molecular genetics, molecular biology, neurochemistry, neuroanatomy, neurophysiology and cognitive neuroscience.
3. Delimitation from other disorders.
4. Follow-up studies showing a homotype progression, i.e. that the disorder remains stable over time.
5. Family studies showing higher familial aggregation as compared with control groups.

Guze (1970) argued that a valid classification should be based on systematic empirical studies rather than on “a priori principles”, according to five specific criteria (Table 2). We have reviewed the literature by these criteria in order to

1. assess the validity of current categorical diagnoses and
2. propose more specific clinical descriptions of the development of aggressive antisocial behaviours.

**2. Method**

The studies assembled for this review were identified using systematic PubMed searches in October–November 2007 by the search terms detailed in Table 3. Hand-searches according to the reference lists of the most important textbooks on the field (Lahey, Moffitt, & Caspi, 2003; Patrick, 2006; Quay & Hogan, 1999; Stoff, Breiling, &

**Table 3**  
Search terms for literature searches in PubMed, in October–November 2007.

“ADHD”	“ADD”	“Hyperactivity”	“Hyperkinetic”	“Attention-deficit disorder”
“Oppositional defiant disorder”	“Conduct disorder”	“Disruptive behavior”	“Antisocial behaviour + children”	“Antisocial behaviour + adolescents”
“Delinquency”	“Criminality + children”	“Criminality + adolescents”	“Aggression + children”	“Aggression + adolescents”
<i>Cross referenced</i>				
“Neuropsychology”	“Neurocognitive”	“Cognitive”	“Executive function”	“Inhibition”
“Motivation”	“Reward”	“State regulation”	“Diagnostic imaging”	“Diagnostic techniques”
“MRI”	“fMRI”	“PET”	“EEG”	“HPA”
“Hormones”	“Endocrine”	“Neurotransmitter”	“Gene”	“Genetic”

In addition, the reference list of each paper was reviewed for additional studies. Papers for the analyses were chosen according to relevance for the validity criteria, mainly among publications dating from 2000 or after, following cited publications into earlier decades.

Maser, 1997) were also performed to identify studies published in non-indexed sources. Selected references of importance for the research questions were added for a revision of the manuscript in March 2009.

Clinical and population-based prospective studies of the longitudinal development of childhood hyperactivity were selected if they included: 1. a group size of at least a hundred subjects; 2. a longitudinal design with duration of at least four years starting in childhood and assessments performed in adult age; 3. assessments of behavioural disorders equivalent to contemporary diagnostic criteria; 4. descriptions of prevalence of criminality. An exception from the third criterion was made for the outstandingly important Dunedin study, which was initially based on behavioural descriptions that were not equivalent to psychiatric diagnoses. Papers were considered in detail, summarized in Table 4 and used for a meta-analysis of the adult outcome for cases identified with hyperactivity in relation to controls in terms of: 1. diagnostic stability; 2. ASPD; 3. criminality; and 4. death by violence or accidents by Fischer’s exact tests. These longitudinal studies also address both the overlap between disorders/problem types (Robins & Guze criterion 3) and homo- vs. heterotype development (criterion 4). We subsequently assessed criterion 1, the specificity of clinical description (in relation to non-disordered states and to other types of disorders); criterion 5, patterns of familial aggregation and associations within and across diagnostic categories by family-adoption and twin studies; and finally, criterion 2, “external” validators with the extension proposed by Andreasen (1995), as detailed in Table 2.

**3. Results**

*3.1. Criteria 3 and 4: Delineation and homotype progression*

Six studies on clinic-referred children and six population-based prospective, longitudinal studies following hyperactive children into adulthood were identified (Table 4). All these studies had included children according to behavioural criteria at base-line. Detailed figures for the follow-up of cases and controls in relation to our defined outcome parameters are given in the bottom row of the table with *p*-values for comparisons. Studies included in the meta-analyses are indicated in the table. The studies that were not included in the meta-analyses did not provide precise figures for cases versus controls or did not include certain measures, such as personality disorders, in their follow-ups. Hyperactive children were at significantly increased risk for ASPD, including CD. There were also more violent deaths in this group, but due to small numbers, the difference in risk (1.3% vs 0.3%) did not reach statistical significance. Diagnostic stability was surprisingly low, and only a small minority (6%) of formerly hyperactive children still met full criteria for AD/HD combined or hyperactive subtypes) at follow-up. In contrast, the prevalences of several other mental disorders were higher than that of AD/HD.

Some conclusions may be drawn from the joint studies. ODD is very common in hyperactive children. Almost all children who develop CD have had ODD, while a subgroup of children with ODD

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