Severe Conduct Disorder—Some Key Issues

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Objective: To examine the state of knowledge about clinically severe conduct disorder and identify key issues.

Method: This paper surveys the literature on conduct disorder and delineates and discusses the critical issues.

Results: Conduct disorder is the subject of a vast and growing amount of research on taxonomy, correlates, etiology, outcome, management, and prevention. There are 2 distinctive types: childhood and adolescent onset. Comorbidity with other disorders is common. It remains a costly disorder, however, with a generally poor prognosis for the childhood-onset type. The validity of the separation of conduct and antisocial personality disorder is questionable.

Conclusions: In view of its huge cost, chronicity, and generally poor outcome, childhood-onset or severe conduct disorder should be considered one of if not the major public health problems of our time, and resources for its study and management should reflect this. The disorder is poorly defined and inadequately studied in females.

Key Words: children, adolescents, conduct disorder, juvenile delinquency, crime, antisocial personality disorder

Antisocial behaviour in children has been a topic of both concern and study for nearly a 100 years. Eclipsed by attention-deficit hyperactivity disorder (ADHD) in the 1970s and 1980s, research on antisocial behaviour in the last decade has intensified both in amount and quality, and it is on this that this review will largely centre. The appellation “conduct disorder” first appeared officially in DSM-III (1) and is described in DSM-IV (2, p 85) as characterized by violation of the rights of others and/or social norms. It outlines 4 domains of symptoms: 1) aggressive, 2) destructive, 3) deceitful, and 4) rule-breaking or defiant. Problems have to be present for more than 12 months, be persistent and repetitive, interfere with social, academic, or occupational functioning, and should occur in a variety of settings. Characteristically, DSM also supplies a polythetic listing of necessary and sufficient major and minor symptoms and has other criteria, like duration and level of impairment, which collectively define a seriously antisocial and disabling disorder. Experimentation with subtyping has also occurred and currently, in a simple but significant data-driven advance, draws a distinction between childhood and adolescent onset. These separations also reflect a crude but generally accurate division between clinically severe and milder degrees of conduct disorder. Much of what follows applies with greatest accuracy and cogency to the severe form of conduct disorder.

Reviewers have drawn attention to a number of problem areas affecting the taxonomic status of diagnosis, which can be summarized very briefly as follows: unreliability of diagnostic methods, especially single-informant bias; threshold for diagnosis; boundaries between conduct disorder and adolescent delinquency found in 25% to 95% of teenagers, depending on the social environment; distinctiveness from other disruptive disorders; and the validity of the dichotomous medical versus the continuous dimensional-personality view (3–12).

Epidemiology

Estimates of the frequency of conduct disorder in any population are bedevilled by the problems of reliability and validity of the diagnosis. Despite these problems, there are some crudely consistent figures. Most estimates lie in the 3% to 5% range for children (3), with a steep rise in prevalence among adolescents of at least twice that. This rise after age 12 is consistent with the 2 types of conduct disorder (childhood and adolescent onset). In her review, Lewis (7) dismisses the possibility of arriving at accurate figures because of the unreliability of measures and the variations in epochs (no doubt due to the varying criteria noted previously) and in places. This is probably more true of adolescence, partly because the single-informant bias is more likely at that time but also because of the very high prevalence of offending and the difficulty of separating conduct disorder from delinquency (5). No great progress in arriving at exact figures is
likely to be made until studies use multiple measures of function and multiple informants (4), including some from the young person’s own peer and cultural group where all agree that the young person is substantially different (or clinically abnormal).

Frequency estimates are intimately tied to the issues that need to be addressed. Scientific research, the planning of health education and welfare services (which society seems more and more unwilling to fund), the imperatives of serious nuisance, and the implementation of feasible, cost-effective interventions all dictate different strategies, objectives, and degrees of exactitude. The public is likely to be most interested in doing something about serious juvenile offenders and young persons with home and school problems, who will be definable largely by parameters of social nuisance, while epidemiologists are more concerned with the true total frequency of the disorder and its correlates.

Correlates

Nearly all studies show a common set of correlates, which were first defined in early studies of delinquency and the Isle of Wight studies (5). These are: disadvantaged socioeconomic or ethnic status, dysfunctional or mentally ill families, and various indicators of poor parenting, all of which are inimical to normal child development (including physical health). It should be noted, however, that these correlations are well below unity, and the pattern is not exactly the same across all studies. The Ontario Health Survey (4,13) failed to replicate the urban–rural differences reported in previous studies and found that low income affected only children, not adolescents. This latter finding may be attributable to the inability of the DSM to discriminate well between conduct disorder and a less serious form of adolescent rebellion. Most studies have shown a greater prevalence among male children of least 4:1, and in some studies, no females were identified. This gender ratio changes to near parity in adolescence, though females are much less severely affected (3,5). The issue of gender has been examined in detail by Zoccolillo (14), who concluded that the diagnostic criteria are male-biased because they rely on lists of behaviours which females rarely enact and that until proper female criteria can be developed, research in conduct disorder is necessarily going to be mostly invalid for females.

Morbidities and Complications

Comorbidities are complex issues; their true relationship to conduct disorder is difficult to disentangle, since any could be precursor (for example, oppositional defiant disorder [ODD]), risk factor (for example, ADHD), consequence (for example, drug abuse) and/or coincidence, in varying admixtures. Only longitudinal studies offer much prospect of disentangling what is primary and what is secondary. This knowledge is important in terms of prevention and effective early intervention.

The mental health comorbidities and complications of conduct disorder include most notably, ADHD, ODD, tobacco, drug, and alcohol abuse, depression, and suicide (3,5,7,10). There is also some relationship with schizophrenia and bipolar disorder, though premorbid and prodromal conduct disorder are too infrequent to be of predictive value (that is, only a tiny fraction of those with conduct disorder will develop these disorders) and will be really useful only when vulnerability to these disorders can be diagnosed accurately by laboratory methods.

Of psychiatric comorbidities, depression is the least studied, but it is one of the most important because suicide is a complication of conduct disorder, especially in older young persons and young adults (10). As Lewis (7) points out, however, suicide in young persons with conduct disorder is not necessarily a sign of depression because impulsivity, one of the core features of conduct disorder, can also lead to suicide.

Educational underachievement and specific language and other developmental disorders are also highly correlated with conduct disorder (5,10). Comorbidity with anxiety disorders is probably low and may even be negatively correlated (10).

Though not well documented quantitatively, health comorbidities and consequences suggest increased morbidity and an alarming mortality (5,7) are associated with conduct disorder, arising principally from generally poor health care and nutrition, sexually transmitted disease, drug and alcohol abuse, hepatitis, motor vehicle accidents, gunshot wounds (especially in the United States), and self-inflicted injuries. Mortality in some groups of presumptively conduct-disordered young persons, such as imprisoned offenders, has been shown to be highly elevated (5).

Cognitive, Motivational, and Personality Correlates

Young persons with conduct disorder have a greatly increased frequency of psychological defects (8–11,15,16). These defects will affect language and its derivatives, such as reading, social perception, and attribution (narcissism and paranoia), social learning (especially moral sense, cooperation, and dispute resolution), self-image (overvaluation), executive function (poor planning, impulsivity, and short attention span), motivation (low persistence, hedonism, excitement bound), and difficult temperament in infancy.

The composite picture, therefore, is one of serious multiple handicaps for a normal life in work, play, and relationships. Not all young persons with conduct disorder have such multiple disabilities, however, though the proportion is unknown and dependent on the definition used.
Biological Correlates

Gender bias in conduct disorder and certainly in its expressions may reflect biological influences. While not discounting any such role, however, Zahn-Waxler (17) properly cautions against assessing it a major or univariate role, going on to describe sex differences in conduct disorder as part of the larger “mystery” of gender differences in behaviour in general. The role of testosterone and other sex hormones in aggressive and violent behaviour has been well demonstrated in animals, including primates, but the evidence in humans is sketchy and unconvincing (18). Lewis (18) has concluded that animal evidence of a biological role in aggression is overwhelming but that in humans there is little evidence so far to support such a role (although there is not too much against it, either). The strongest link so far seems to be the reduced serotonin level seen in some aggressive youngsters.

Lahey and colleagues (6) have summarized the few studies on conduct disorder that have examined biological concomitants such as psychophysiology. Quay (19) believes that there is evidence for an abnormal noradrenergic and serotoninergic function in conduct disorder but not for changes in dopaminergic function. He links these hypotheses to Gray’s motivational personality theory, which has some similarities to that of Cloninger (20) in the adult field. He also documents a strikingly consistent finding of a diminished electrodermal response to novel or powerful stimuli, which he interprets within the theory of reward dominance over behavioural inhibition.

In a reversion to the days of William Healey and eugenics (5), when it was believed that criminality was a sign of hereditary progressive degeneration, genetic studies of conduct disorder are reemerging and again pointing to some hereditary role (18). Most of the data are about antisocial adults and not conduct disorder, but the heritability of criminality provides consistently positive findings. In juvenile delinquency, until recently the only area of any investigation directly relevant to conduct disorder, genetic influences are modest indeed and are greatly overshadowed by shared (primarily peer) environmental influences (21). Most juvenile delinquents, however, do not have true or childhood-onset type conduct disorder, which is the type in which genetic influences would seem most likely, so this finding cannot yet be held as valid. What is needed is research that pays careful attention not only to the new methodology of behavioural genetics, which properly tests for error rather than assigning it to nongenetic influences but also, more particularly, to properly diagnosed conduct disorder. Thus studies should set an appropriate threshold and include subtyping, such as childhood versus adolescent onset.

Etiology

Currently, it is assumed that poor parenting in childhood and a social ecology which facilitates crime in adolescence are the causes of conduct disorder. Most of the evidence presented earlier is, however, correlational. Demonstrating causality, especially the mechanisms by which any cause such as parental criminality or bad companions works, is more difficult. Increasingly, however, focus in research is moving beyond empirical correlates and attempting to define the causal mechanisms which are then used to improve research on causality (9–10, 22).

Studies have demonstrated 2 major kinds of abnormal correlates: those in the young persons’ environment and those within the child. It is the attempts which try to marry these two aspects which lie at the cutting edge of research as investigators try to learn how environmental factors operate to create the core abnormal structures which can be seen in young persons with conduct disorder. Increasingly, such studies look not only at the end product but also at the evolution of the problem by defining what are sometimes called developmental trajectories. It is impossible to do justice to the intellectual vigour and power of this line of research in a short review. This is even more true since it is becoming increasingly difficult for psychiatrists to understand the details of this work because our training is woefully deficient in the requisite knowledge in developmental psychology.

One of the best expositions as to how family and social influences may create conduct disorder is that by Crick and Dodge (22). These authors meld attachment theory (as a window of expectation on the world) with social learning, which shapes the perception we have of others and the emotions, cognitions, and behaviours that we learn to act upon in the world. The most exciting thing about these new theories is that they point to forms of prevention and treatment which move from the practical and common sense to the truly scientific. They also expose how naive and simplistic the views of most monolithic theorists (for example, those who focus on separations or child sexual abuse or male violence) are. Yet they also indicate how drastically the training in child and adolescent psychiatry needs to change and how social policy makers need to upgrade the source and level of their advice.

Among these theories, biological causation is the poor relation. After a strong start at the turn of the century with the eugenic movement, as exemplified in the views of Healey (5), the arrival of sociology and psychoanalysis in the 1930s caused interest in biological causes to lapse and even be dismissed. With the rise of biological psychiatry in the last quarter of this century and the concomitant development of human genetics, interest has become refocused on biological causes.
As in the psychosocial sphere, there are 2 main fields of inquiry: empirical, in which those who have severe conduct disorder are studied to see if biological abnormalities can be found, and etiological, which attempt to define the primary cause of the abnormalities. The biological correlates of conduct disorder have been well reviewed elsewhere (5–7,11,18) and outlined briefly above (for example, male gender, lower skin conductance, serotonin, catecholaminergic abnormalities, and less clearly biological, neuropsychological abnormalities). Unfortunately, most of these correlates could just as easily be end products of psychosocial as of biological variables. In addition, few if any of them are specific to conduct disorder.

More interest now centres on genetic factors, which do strike at primary etiology. As noted, studies of twins and adopted-away siblings suggest that criminality has some hereditary component (5). Unfortunately, these studies are sparse, often old, and do not centre on conduct disorder as it is now defined. Temperament has attracted interest as a presumptively innate factor, but the difficulties of measuring temperament have handicapped what appears to be a promising line of research (5). A recent review by Lynam (23) proposes that ADHD comorbid with conduct disorder defines “juvenile psychopathy” or the most severe group of conduct disorder. This thesis is of interest in that, if correct, it would strengthen the genetic argument, since ADHD is one of the most clearly genetic of all child psychopathological disorders.

In summary, psychosocial theories of etiology based on attachment and social learning theory are currently the most highly developed. Biological theories are relatively underdeveloped, but there is enough smoke to suggest that there is fire below, especially in the genetic–temperamental area. It seems, however, most logical to posit that biological factors are vulnerability or risk factors rather than primary, stand-alone etiology. The same argument can be made for psychosocial factors, though seldom is, because proponents seem to shut down all cognition when confronted by such things as child abuse. For example, Ferguson and Lynskey (24) have shown that among adolescents exposed to significant family adversity, there is a “resilient” group characterized by low novelty seeking and lower affiliations to delinquent peers. Thus, in most cases, biological and psychosocial factors probably interact to create the final clinical picture. In this equation, it is possible to argue that extremes may result in univariate etiology but that most cases will represent varying admixtures of the 2 domains of variables. We can expect to see major advances in the biological side of this equation in the next decade.

Costs

Young persons with conduct disorder incur a huge but unknown cost to both public and private society (25). Most cost estimates have been confined to the rather simple measure of imprisonment (26). This is unfortunate, since if the full extent of cost were to be assessed accurately, it might result in well-funded and properly thought-through prevention and management programs.

Dollar costs arise in several main areas. The first is loss of productivity. Cynically, one might note that society pays for children less from altruism than as an investment for the pensions and health services of their elders! Few persons with severe conduct disorder have or will have regular or productive employment into middle life (27). The second cost area is the direct public contributions to or on behalf of the young person (benefits, health care, housing, and treatment, management, and educational programs). These include costs to the partners and children whom they will often desert in serial irresponsible relationships. The third area is the law enforcement costs, including police and court time, legal aid, social welfare or probation reports, psychologist and other assessments, and imprisonment. The fourth type of cost is victim costs, that is, loss of productivity, health care, property damage, and legal costs and so on. Family costs, consisting of among other things legal, health, treatment, loss of earnings, damage, and reparations, are another cost area. There is a tendency to discount or neglect what most families of children and young persons with conduct disorder have to pay and to blame them instead. Sixth are security costs. These arise as a frightened and beleaguered urban society attempts to build moats and ramparts against property crime, much of which is committed by juveniles. The cost of these preventive security mechanisms greatly outweighs that of the actual damage which would result from the occasional burglary. Finally, there are emotional costs that need to be considered. As any clinician working with young people with conduct disorder knows, the most important, unrecognized, and unevaluated cost is not measurable in dollar terms but in the emotional damage that is caused to parents, siblings, peers, partners, neighbours, teachers, social workers, and victims. It could well be argued that this cost is larger than any other because it impinges on the mental health and efficiency of the nation as a whole. In a sense, it could be said that there is a war on, except that most societies refuse to mobilize the resources that they would for an external threat of this magnitude.

This issue of costs is an important area of research on conduct disorder since professionals, whether they admit it or not, and those who dictate social policy and public expenditure increasingly respond to well-argued dollar costings, especially if these can identify not only costs but savings that could be made.
Treatment, Prevention, and Outcome

Treatement

There is probably no field of child and adolescent psychopathology in which the general public is so involved and so frequently offers such strong views as conduct disorder. With monotonous regularity, the media advances solutions to the problem of youth crimes, most of which are Draconian, impractical, simplistic, costly, and ineffective. One recent example in New Zealand is the boot camp idea, now well discredited overseas (28), and another is diet as cause and treatment.

Among professionals, it is generally assumed that treatment of conduct disorder is difficult and largely unhelpful, especially in the long term (8,13). Less than 50% of children with conduct disorder will become persistently antisocial adults (11), however, and though we do not know how this happens, it does point to the possibility of effective intervention. Some years ago in this journal, Shamsie and Hluchy (26) set out a pragmatic menu of programs needed. Since then, a number of writers (29–33) have offered innovative, more theory-driven, and cautiously optimistic suggestions for treatment.

The principles generally agreed upon are as follows:

1) Intervention should be as early as possible. 2) It should cover as much of the child’s day as possible every day. 3) It should include all caregivers. 4) It should be consistent across all environments and across time. 5) It should be maintained for as long as needed (this may be years). 6) It should be multimodal and focus not just on behavioural control (though this is fundamental) but also on the other key deficits in cognition, moral reasoning, education or vocation, and the recreational and social-interactional skills that propel much of the antisocial behaviour. 7) It should address comorbidities such as depression, drug and alcohol abuse, and ADHD. 8) It must provide protection after discharge against what have been called “snares” such as chronic unemployment, drug and alcohol abuse, and association with delinquent peers and adults, all of which can entrap youth into a life of crime (34).

The difficulties associated with the last principle are probably hardest to overcome because they often lie beyond the power of mental health professionals.

Pharmacotherapy makes at best a small contribution to the management of conduct disorder. Stimulants seem most useful (for comorbid ADHD), intermittent low-dose phenothiazines and possibly serotonergic drugs for reactive aggression are less so, and of dubious if any value despite considerable popularity are lithium, clonidine, carbamazepine, and propranolol (35). It is therefore quite clear that no drug is the answer to conduct disorder.

In summary, treatment of conduct disorder is no field for amateurs—it is complex, expensive, and must be comprehensive and carefully thought through.

Prevention

Because of the cost and severity of the problem of conduct disorder and the poor results of late interventions, interest has shifted to prevention (13). Several notable pilot projects in this area have been discussed in detail by others (5,36).

As noted, research has demonstrated the presumptively pathogenic effect of early environments which are inimical to effective rearing of children and which interfere with attachment, the acquisition of socially adaptive skills, and the young person’s confidence in a good future within normal societal paths. Primary prevention programs focus on vulnerable or high-risk parents, ideally before or shortly after the birth of their children, and in various ways attempt to help them cope with the slings and arrows of outrageous fortune—poverty, solo and young parenthood, ignorance about children and their needs, social isolation, violence in the home, and other deviant social models. Though these programs are not new (for example, the Mental Hygiene movement of the 1930s and Head Start had somewhat similar objectives), the success of these programs has yet to be demonstrated. They do, however, have considerable promise and compelling face validity. Crick and Dodge (22) have offered a way of conceptualizing such preventive programs that takes them beyond the common-sense and merely compassionate techniques, even beyond the domain of simple “parenting,” to include interventions with children to change endogenous structures.

Until it can be shown that such programs are regularly and powerfully effective, I believe that it is a mistake to promote them only in terms of prevention of conduct disorder and crime because, primarily, they embrace what a decent society wants for all its children. Pragmatically, they also have general potential benefits that extend far beyond crime prevention into a healthier, happier, and more productive society.

Primary preventive programs like these are expensive and politically difficult to implement in a world that is reverting to the Victorian idea that misfortune is a sign of personal failure and sin for which the individual and all about him or her should be punished into contrition.

Secondary prevention, which seeks to intervene at the first or prodromal signs of conduct disorder (notably defiance to adults and aggression to peers), is an alternative strategy that has the advantage of limiting the scope and expense of programs to those most clearly at risk. The most ambitious of these programs is the multicentre FAST TRACK program in the United States (35), which begins with first-grade children, has multi-environment interventions, and uses both untreated
remarkable resurgence of interest in the last decade because of ADHD in the 1970s and 1980s, but there has been a good start, the study of conduct disorder was eclipsed by that public health problem in children and adolescents. After a another decade. This means that research on treatment for established conduct disorder must continue.

Outcome

Several longitudinal studies have shown that about 40% to 50% of children with severe conduct disorder become recidivist criminals and/or antisocial personality-disordered adults (5,13). This picture unfortunately may even be optimistic, since there are other poor outcomes, such as psychiatric disorder, alcohol and drug abuse, chronic unemployment, domestic violence, and unstable marital relationships, that closer study of potential outcomes has revealed (36). Additionally, the mortality rate in this group is about 10 times that of the normal population (5). This generally poor outcome gives cogency to research on the disorder. It will come as no surprise to the reader that outcome is correlated with the number of problems or disabilities that the young persons shows (36) and with age of onset (33).

The last point that needs to be made is that, while only some of the children with conduct disorder go on to have antisocial personality disorder, all adults with antisocial personality disorder give a history of conduct disorder (10,11). It is likely, too, that if those who have childhood-onset conduct disorder but who drop out of criminal activity and are thus considered to have a favourable outcome were to be more closely scrutinized, as was done in the Cambridge Youth Study (36), we would see that most of these individuals do in fact have an antisocial personality disorder, though it may be less severe than that of those who end up in jail. This calls for reexamination of the need for 2 diagnostic categories, which create the illusion of discontinuity and barriers to research and treatment.

Outcome in females has been poorly studied, with little differentiation between childhood- and adolescent-onset conduct disorder, which may spuriously improve outlook. Disability seems less centred on crime and more on welfare dependence, multiple partners, and violent relationships (37).

Conclusions

Conduct disorder may be the most important social and public health problem in children and adolescents. After a good start, the study of conduct disorder was eclipsed by that of ADHD in the 1970s and 1980s, but there has been a remarkable resurgence of interest in the last decade because modern affluent societies have become increasingly intolerant of juvenile delinquency. Much of the research in conduct disorder has been clouded by the failure to separate the serious childhood-onset from the much more benign adolescent-onset type. Conduct disorder in females has been poorly defined and less studied than in males.

The cause of conduct disorder probably lies partly in as yet poorly defined genetic and other biological vulnerabilities, partly in pathological child rearing, and partly in deviant adult and peer models. The diagnosis of the disorder is progressing but is still far from robust taxonomically, and DSM-IV is not the end but just a beginning to this process. A persistent problem is comorbidity. Treatment is expensive, prolonged, and of limited value, though there are promising leads, none of which, so far, is biological. Outcome is often poor, and the cost to society of this disorder is incalculable but poorly studied. As in all diseases, the most effective point to intervene would be in primary prevention or early intervention; ways and means of doing this for conduct disorder remain to be developed, implemented, and proven, however, though there are promising leads.

Research has shown that understanding this disorder calls for intellectual power of the highest kind coupled with a good grasp of modern developmental psychology, much of which is absent from the knowledge and training of psychiatrists, including specialists in child and adolescent psychiatry. Psychiatry is among the least equipped to do research and design treatment programs for conduct disorder because our profession is bedevilled by simplistic, univariate medical models and archaic developmental theories, which often preclude effective effort. There is, however, an important role for modern developmental neuropsychiatry in the search for a better understanding of any biological base of conduct disorder and in its correction. This role is likely to grow with the development of molecular genetic medicine and a more diverse and less empirical pharmacotherapy.

On the whole, however, there is reason for optimism in the rising public recognition of the size and cost of the problem of conduct disorder and, one hopes, an associated willingness to underwrite the cost of doing something about it. Another positive note is the high quality of much of the research being undertaken in etiological formulations, classifications, developmental trajectories, treatment, prevention, and the linking of research-bound theory to prevention and treatment, much of which has outstripped thinking about other child and adolescent psychiatric disorders. There is also a need to consider the utility of separating antisocial personality disorder from its progenitor. A combined approach in research could prove highly beneficial, although it would require adult psychiatry, which largely controls the resources, to take off its blinkers and see “adult” disorders in their true perspective as but one stage in a life-long process.
Clinical Implications

- The etiology of conduct disorder is often complex.
- There are 2 types of conduct disorder: childhood-onset type and adolescent-onset type.
- Treatment for childhood-onset conduct disorder is difficult and expensive.

Limitations

- Treatment studies are largely inadequate.
- Biological factors in etiology are poorly studied.
- Genetic studies fail to discriminate delinquency from conduct disorder and childhood-onset type from adolescent-onset type.

References


Résumé

Objectif : Examinier l’état des connaissances au sujet du trouble des conduites cliniquement grave et identifier les questions clés.

Méthode : Dans cet article, on donne un aperçu de la littérature sur le trouble des conduites et on cerne les questions essentielles, qui font l’objet d’une discussion.

Résultats : La recherche, déjà considérable, ne cesse de s’accumuler au sujet de la taxinomie, des corrélats, de l’étiologie, de l’évolution, du traitement et de la prévention du trouble des conduites. Celui-ci se divise en 2 types : l’un débutant à l’enfance et l’autre à l’adolescence. La comorbidité est fréquente entre le trouble des conduites et d’autres affections. Cependant, le trouble des conduites entraîne encore des coûts importants et le pronostic du type débutant à l’enfance est sombre en général. La validité de la distinction entre la conduite et la personalité antisociale est mise en doute.

Conclusions : Étant donné son coût énorme, sa chronicité et son évolution généralement défavorable, le trouble des conduites grave ou débutant à l’enfance devrait être considéré comme un des problèmes de santé publique les plus importants, sinon le plus important, à notre époque, ce qui devrait se refléter par l’importance des ressources affectées à son étude et son traitement. Ce trouble est mal défini, et on ne l’a pas bien étudié chez les femmes.