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EDITORIAL

Conduct Disorder: A Challenge to Child Psychiatry

The 2 papers in this section review the etiology and treatment of conduct disorder (CD), one of the most common disorders in child psychiatry with an incidence of between 5.5% and 6.9% (1). Even with the best-known treatment, the success rate on follow-up does not exceed 74% (2). The failure in treatment often results in the adolescent becoming an adult with antisocial personality disorder (APD) and a criminal record. Youths with this disorder cause immense suffering to society because of their antisocial behaviour and the enormous costs associated with treatment, court procedures, probation, detention, and incarceration.

As in most disorders, we need to take 2 steps to reduce the suffering caused by the disorder. The first is to prevent the disorder's development. If this is not possible, as in the case of CD, then it is important to identify the disorder at an early stage—when the interventions have a better chance of success—thus preventing it from becoming more pernicious and difficult to treat, with poorer prognosis. The second step is taken when the disorder has developed and is a problem to the individual, the family, and society. At this stage, it is important to use effective treatments that have been proven to cure or reduce the likelihood of recurrence.

Early intervention makes a great deal of sense in the case of CD. The disorder is generally classified in 2 types: early-onset, which if not treated has poor prognosis and late-onset, which has comparatively better prognosis (3). It has been shown that aggressive behaviour in kindergarten, if not treated, can continue into adolescence with greater severity (4). A study by Robins (5) showed that, if aggressive and antisocial behaviour appears before the age of 6 years, the chance of the child developing CD in adolescence and APD in adulthood is 3.2%. If the aggressive and antisocial behaviour starts after the age of 12 years, however, the chances of the child developing APD are reduced to 0.9%. Therefore, it makes sense to identify children in kindergarten and grade 1 who are exhibiting persistent and severe aggressive and noncompliant behaviour and provide help to the child, the parents, and the teacher. The early-intervention programs aim at parent training, home visiting, teaching social skills to children, academic tutoring, and training teachers in classroom interventions.

Many such programs, which provide help to young children at risk, have reported good results (6–8). It does not make sense to wait for these children to grow up and develop full-blown CD in adolescence. Early intervention is effective; it costs less and can prevent the suffering of the child, the family, and society. In fact, few disorders are as preventable as CD. In CD, we have a disorder wherein prevention is possible at a small cost. Conversely, treatment of the fully developed disorder is expensive, and the results are far from satisfactory. There has been remarkable improvement in the results obtained in the treatment of CD in the last 20 years. Evaluating many treatments practised in the past 2 decades indicates that most were no more effective than no treatment; in fact, on follow-up, some had worse results than no treatment (9,10). These treatments included casework, individual psychotherapy, group counselling, milieu therapy, and therapeutic community. The failure of these treatments was due to 2 factors: 1) a lack of understanding of the factors that contribute to the disorder's development and 2) a tendency to focus on a single approach for all youths with CD. At present, there are many treatments that, as described by Dr Frick, do take into consideration the many factors leading to the disorder's development and do individualize treatment. The results of these treatments are better than no treatment but still not as good as one would wish.

It has to be recognized that many youths with CD are not handled in the community: many are charged for breaking the law and placed in some custodial setting where they receive very different treatment from those in mental health settings. Although they may have the same disorder, many are never diagnosed, and seldom in their sentencing is an effort made to choose a punishment proven to reduce the rate of recidivism.

The problem of youth violence can be dealt with more effectively, first, by identifying young children with persistent antisocial behaviour and modifying their behaviour by working with the children, their families, and their teachers; and second, by handling youth with CD who have been charged and found guilty of breaking the law, as well as those not charged, with approaches that have been proven on follow-up to be successful.

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Guest Editor

IN MEMORIAM

Dr JDM (Jack) Griffin died in hospital on July 12, 2001, while recovering from surgery for a fractured femur. His funeral, on July 15, was attended by his sons, Peter and Tony, his companion, Barbara Chipman, extended family members, and many friends. The handful of professional colleagues present remembered with great affection Jack Griffin's profound influence on their lives.

A few weeks earlier, Jack's family, friends, and colleagues young and old gathered to celebrate his 95th birthday. Except for his diminished vision and hearing, Jack looked extremely well and happy. He was elegantly dressed, with the Order of Canada proudly pinned to his lapel. Much later, after most of the guests had departed, Jack entertained us with sentimental songs played on a grand piano—a perfect ending to a memorable evening.

John Douglas Morecroft Griffin
OC, MA, MD, FRCPC
June 3, 1906 to July 12, 2001

Dr Griffin's career as a colonel in the Canadian Army, under the legendary Major General Brock Chisholm, is modestly documented in his book, *In Search of Sanity—A Chronicle of the Canadian Mental Health Association*, published in 1989. It includes an account of the war work of the Canadian National Committee for Mental Hygiene—now the Canadian Mental Health Association (CMHA)—from 1939 to 1945. Working with luminaries such as Clarence Hincks, William Line, John Seeley, and William Blatz, Colonel Griffin devoted himself to developing psychiatric rehabilitation programs for ex-service men and women throughout Canada.

In those days, very few provincial mental hospitals were capable of helping the returning veterans. New forms of interdisciplinary, community-based mental health programs had to be rapidly developed. With Dr Jack as their mentor, a cadre of young psychiatrists played a prominent part in establishing mental health clinics and in transforming the old mental hospitals into active treatment centres. Prominent among them were Don Atcheson, DE Cameron, Rhodes Chalke, Bob Jones, Ab Miller, Sam Lawson, Burdett McNeel, Charlie Roberts, and Jim Tyhurst. Jack Griffin served as General Director of the CMHA from 1951 to 1971. Speaking about him at this time, Dr Hincks grumbled: "Nothing succeeds like one's successor."

As a medical student at the University of Toronto, Jack played piano in a varsity dance band. His experience as a pianist and performer served him well throughout his career. Although basically shy and reserved, Jack was an outstanding communicator. In addition to lecturing to various audiences, Jack used radio, film, and later, television, with great success. His CBC Radio series, *!X!O!A!In Search of Ourselves*, which dramatized common mental health problems of children and adults, was prized throughout the English-speaking world.

A master in the art of spotting talent before anyone else, Jack recruited energetic people with leadership skills and put them to work on various CMHA committees and task forces. An enthusiastic sailor and skipper, he also had an uncanny ability to sniff the winds of change and use them to support social and political action on mental health. Thus, during the 1950s and 1960s, CMHA briefs were presented to various Royal Commissions, including those on broadcasting, industrial and economic development, forensic services and the law relating to mental illness, and health insurance.

While all these CMHA reports were influential, only 2 of them can be mentioned here. *More for the Mind*, also known as the Tyhurst Report, was published in 1963. Its key recommendation, which revolutionized psychiatric services in Canada, was that "mental illness should be dealt with in the same organizational, administrative, and professional framework as physical illness."

Dr Griffin, who did postgraduate work in child psychiatry at the Hospital for Sick Children, continued to be passionately interested in the well-being of children and families. He believed that effective intervention at an early age was the most cost-effective means of reducing the incidence of mental illness. This is why Jack was so very proud of *One Million Children*, the report of the Commission on Emotional and Learning Disorders in Children (CELDIC), published in 1970. The Commission was chaired by Dr Denis Lazure and Dr CA Roberts, with Majorie King as executive secretary.

Their work was premised on the finding that Canada lacked a comprehensive system for treating children with mental illness or severe maladjustments. They were also perplexed to discover that provisions in Canada for training child psychiatrists, psychologists, and social workers were equally primitive.

In this brief tribute, it is impossible to do justice to Dr JD Griffin's international work with the Fifth International Congress on Mental Health, held in Toronto in 1954, and with the World Federation for Mental Health (WFMH). He did, however, maintain contact with the WFMH long after his retirement in 1972. Jack Griffin was the recipient of many awards, but none pleased him more than the Order of Canada, bestowed in 1996.

For about 25 years, I had the great pleasure of working with Jack Griffin on the history of Canadian psychiatry. Our work resulted in the formation of the Archives on the History of Canadian Psychiatry and the Museum of Mental Health Services (Toronto) Inc, both of which are based at the Centre for Addiction and Mental Health in Toronto. The continuation of this work is, perhaps, the best possible memorial to the life and work of Dr JDM Griffin.

Cyril Greenland, MSc, PhD

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Jack Griffin's death will evoke little reaction among today's psychiatrists. Few, if any, will know that with his death the last of a remarkable group of men has gone. Together with Jack Griffin, the outstanding ones were Bob Jones, the founding president of Canadian Psychiatric Association (1951 to 1952), Rhodes Chalke, and Bill Hawke. They had all specialized in the 1930s and played an active part in World War II. In the postwar years, this group that moved Canadian psychiatry forward by serving with dedication, diligence, and modesty. They were intellectuals, scholars, pragmatists, and close friends who loved life and lived it to the full.

Until a few months before his death, Jack regularly played piano for Shellback, a club for landlocked mariners. But his major contribution was bringing together psychiatrists from coast to coast. He was responsible for starting many mental health clinics and for bringing psychiatry into general hospitals.

To the end, Jack was concerned about the future of Canadian psychiatry. Earlier this spring, at our last dinner, we discussed developments in medicine, neurosciences, psychodynamics, psychotherapy, and psychopharmacology and wondered whether these specialties would continue to fragment or coalesce to form a new whole.

I knew Jack for 47 years. He was the most generous, warmhearted, loyal, modest, and supportive friend and colleague. I thank you, Jack, and mourn you.

Ruth Kajander, MD, FRCPC

IN REVIEW

Effective Interventions for Children and Adolescents With Conduct Disorder

Paul J Frick, PhD[1]

Many different types of interventions have been used to treat children and adolescents with conduct disorder (CD). Unfortunately, most have had very limited effectiveness and, in some cases, have even shown iatrogenic effects. A primary reason for this limited effectiveness has been the failure of most treatments to directly address the causal mechanisms implicated in the development of CD. A few exceptions that have based interventions on the available research and that have proven to have some efficacy in reducing the conduct problems in youths with CD are reviewed. More important, a model for intervention is presented. This model emphasizes that interventions for youths with CD need to be comprehensive. That is, they need to take into account the myriad factors both within the child and within his or her social context that can cause and maintain CD symptoms. Further, interventions need to be individualized; they need to take into account the different pathways along which children may develop CD. Two intervention approaches that are consistent with these principles are reviewed, as are important directions for advancing treatment technology for youths with this disorder.

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Key Words: *conduct disorder, antisocial behaviour, treatment, prevention*

Conduct disorder (CD) refers to a chronic pattern of antisocial and aggressive behaviour in which either the rights of others or major societal norms, or both, are violated (1). Although the severity of behavioural disturbance can vary greatly among children with CD (2), many such children show significant psychosocial impairments. These include impaired educational achievement, poor social relationships, significant conflict with parents and teachers, involvement with the legal system, and high rates of emotional distress, to name just a few areas documented by clinical research (3). In addition to these personal costs, CD is also a very costly form of psychopathology for society. The costs are both monetary and social. Monetary costs include those associated with incarceration to prevent further offending by those children with CD who commit serious delinquent acts; they also include the costs of repairing schools damaged by vandalism. Social costs include the inadequate and unsafe learning environments created in schools by the behaviours of many children with CD. As well, they include the reduced quality of life experienced by those victims whose rights have been violated by children with CD and by others living in high-crime neighbourhoods (4). There is no better example of this costliness to society than the effects of violence committed by juveniles. Most juveniles who commit violent acts show a history of antisocial behaviour consistent with a diagnosis of CD (5). As a result, understanding and effectively treating children with CD is a critical component of any plan to reduce juvenile violence.

Given its great societal implications, it is not surprising that the treatment of CD has been the focus of a large number of controlled treatment-outcome studies. For example, a recent review of published treatment-outcome studies focusing only on psychosocial treatments for children and adolescents with conduct problems documented 82 studies involving over 5272 children (6). The extent of this literature far surpasses the published research on the treatment of most other childhood disorders. Unfortunately, it is possible to conclude from this extensive effort that the vast majority of treatment approaches have proven to be largely ineffective (7). Of even greater concern is the evidence that some types of intervention, particularly those that involve antisocial peer group interactions, can have iatrogenic effects on the children being treated: they actually increase the level and severity of antisocial behaviour, as well as the risk for negative life outcomes as adults (8). Therefore, uninformed and ill-conceived treatments can actually do more harm than good.

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One reason for the ineffectiveness of many interventions is that they have often been based on broad theories of intervention developed for treating adults or on political and philosophical pressures to appear "tough on crime" (9). They have not been based on our most current knowledge of the factors that can lead to the development of CD, and they have not considered important developmental issues relevant for working with disturbed youths (10,11). For example, many interventions have ignored the powerful influences of a child's psychosocial context (for example, family, peer, and neighbourhood). Unless these contexts are modified, it is very difficult to bring about or to sustain changes in the child's behaviour over time. This is not to say that individual predispositions in a child do not contribute to the development of CD (3) or that these predispositions may not be actively involved in shaping a child's psychosocial context (12). The critical issue is the need to recognize the transactional nature of the processes that may lead to CD, and most other forms of psychopathology: it is important always to consider the child's context when designing and implementing treatments.

Although this overview of treatment effectiveness is somewhat pessimistic, 4 treatments have proven to be effective in controlled outcome studies. Given the importance of a child's social context, it is not surprising that 3 of the 4 effective treatments come from the cognitive-behavioural tradition that emphasizes the role of social learning. The one nonbehavioural treatment that has proven effective is the use of stimulant medication to reduce the impulsivity that can lead to aggressive and antisocial behaviour in some children with CD. Irrespective of their theoretical underpinnings, one commonality of these 4 treatment approaches is that they target processes implicated by research in the development of CD. Table 1 provides a summary of these treatment approaches and the theoretical basis for their use.

An Overview of Effective Treatment Approaches

Contingency Management Programs

The first intervention listed in Table 1 is the use of contingency management programs. The theoretical rationale for this treatment approach has typically focused on the contention that many children with CD come from families in which they have not been exposed to a consistent and contingent environment—a poor socialization experience that plays a major role in their deficient ability to modulate behaviour (for example, to delay gratification or to conform to parental and societal expectations) (13). A structured behaviour-management system is designed to overcome these deficiencies in their socialization. Another rationale that is also consistent with existing research is that some children with CD have a temperamental vulnerability which makes them more susceptible to a noncontingent environment: they may, for example, be over-focused on the potential positive consequences of their behaviour (such as obtaining a stereo) to the extent that they do not consider potential negative consequences (for example, being arrested for stealing, or affecting the livelihood of the store owner) (14).

The basic structure of contingency management programs is deceptively simple. These programs all involve 1) establishing clear behavioural goals that gradually shape a child's behaviour in areas of specific concern, 2) developing a system to monitor whether the child is reaching these goals, 3) having a system to reinforce appropriate steps toward reaching these goals, and 4) providing consequences for inappropriate behaviour. These programs have proven to bring about behavioural changes for children with CD at home (15), at school (16), and in residential treatment centres (17).

Although they appear quite simple and straightforward, many behavioural management programs are not used effectively. For example, these programs need to be individualized in terms of selecting both appropriate goals for the child and the reinforcers and punishments that will motivate each child. In addition, many programs do not define goals in a way that allows for systematic monitoring of whether the child is meeting them. Further, many of these systems are typically used solely for behavioural control. Negative consequences for inappropriate behaviour are provided (for example, points are lost for misbehaviour, and fighting results in forced isolation), but there is no mechanism to systematically encourage positive behavioural changes (for example, points are gained for appropriate expression of anger, increased prosocial interactions with peers, or respectful comments to adults). Finally, it has been very difficult to find methods to extend the behavioural changes brought about by the contingency management programs to situations in which the consistent and structured contingencies are not operating.

Parent Management Training

The second treatment that has proven to be effective for many children with CD is Parent Management Training (PMT). A critical focus of PMT programs is to teach parents how to develop and implement very structured contingency management programs in the home. PMT programs, however, also focus on 1) improving the quality of parent–child interactions (for example, having parents more involved in their children's activities, improving parent–child communication, and increasing parental warmth and responsiveness); 2) changing antecedents to behaviour to enhance the likelihood that positive prosocial behaviours will be displayed by children (for example, learning how to time and present requests or provide clear and explicit rules and expectations); 3) improving parents' ability to monitor and supervise their children; and 4) teaching parents more effective discipline strategies (for example, more consistent discipline and various approaches to discipline). Deficits in these specific aspects of parenting have been consistently linked to child CD in a large body of research (3,18); of all interventions used to

treat children with CD, the effectiveness of this type of technique has been the most consistently documented (7).

Many very explicit treatment manuals have been developed for implementing PMT for various age groups (for example, preschool [19], school-age [20], and adolescent [21]) and for children with specific needs—for example, children with attention-deficit hyperactivity disorder (ADHD) (22). As a result, readily available sources of guidance exist for the implementation of these programs. Further, these techniques have been provided in many different modalities—with individual parents, with groups of parents, and even through videotaped instruction (23). The level of intensity and method of implementation can be adapted to the needs of the individual family, and these programs can be implemented in a wide variety of settings.

Key limitations of these treatment approaches, however, have been the large number of parents who do not complete the parenting programs and their lack of effectiveness for the most dysfunctional families (7,24). As a result, to increase the effectiveness of these interventions, it is important to focus on ways to engage families in the intervention and to consider the broader family context. This context may include factors that could prevent parents from using the techniques taught in PMT programs, such as parental depression or parental substance use, high rates of marital conflict, or lack of social support for the parents (24). A useful guide for enhancing parental engagement and determining how parenting issues are embedded in the broader family context is an approach called Functional Family Therapy (25). This approach has been shown to be effective in treating older children and adolescents with CD in severely distressed families from diverse ethnic and socioeconomic backgrounds (26,27).

Cognitive-Behavioural Skills Training (CBST)

The third type of intervention that has proven effective is a cognitive-behavioural approach designed to overcome the deficits in social cognition and in social problem-solving experienced by many children and adolescents with CD. Research on children who are aggressive or who have CD has consistently documented deficits in the way they process social information, including the way they encode social cues, interpret these cues, develop social goals, develop appropriate responses, decide on appropriate responses, and enact appropriate responses (28). For example, some severely aggressive children tend to attribute hostile intent to ambiguous provocation situations with peers, making them more likely to act aggressively toward peers (29,30). Other aggressive children tend to associate more positive outcomes for their aggressive behaviour, making them more likely to select aggressive alternatives to solving peer conflict (30).

Most CBST programs include some method of having a child inhibit impulsive or angry responding. This allows the child to go through a series of problem-solving steps (for example, how to recognize problems, how to consider alternative responses, and how to select the most adaptive one to deal more effectively with problems encountered in peer interactions). Despite many commonalities, the various programs do have somewhat different emphases. For example, the Self-Instructional Training Program (31) focuses more on inhibiting impulsive responding, the Anger Coping Program (32,33) focuses more on changing perceptual biases in regard to peer intent by using perspective-taking task exercises, and the Promoting Alternative Thinking Strategies Curriculum (PATHS; 34,35) focuses more on helping the child to develop social skills and gain better emotional awareness.

Each cognitive-behavioural program described above is an explicitly skills-building approach to intervention. The therapist plays a very active role in these programs, modelling the skills being taught, role-playing social situations with the child, prompting the use of the skills being taught, and delivering feedback and praise for appropriate skills use. Most of the programs are designed for a group format. Given the potential dangers in having antisocial individuals interact in groups (8), however, the groups are kept very small, the group interactions are very structured in content, and contingency management programs are typically used to promote the use of the skills and limit inappropriate behaviours. Key limitations to the effectiveness of most cognitive-behavioural programs are the difficulties encountered in getting children to use the skills learned in the program outside the therapeutic setting (36) and to maintain the skills over extended periods of time after the intervention has ended (32). To enhance generalization, several programs have been designed for implementation outside the typical mental health delivery setting (for example in schools [35]), so that the skills are taught in the environment in which they will be used. Also, to promote generalization, most programs include practising skills in various settings. Most important, however, all the programs involve people present in the child's natural environment, such as parents and teachers, to prompt and encourage use of these skills outside the therapeutic context.

Stimulant Medication

The only treatment approach included in Table 1 that is not cognitive or behavioural in nature is the use of stimulant medication. A large proportion (between 60% and 90%) of clinic-referred children with CD also have ADHD (37). The impulsivity associated with ADHD may lead directly to some of the aggressive and other poorly regulated behaviours of children with CD (2,38). In addition, the presence of ADHD may contribute indirectly to the development of conduct problems through its effect on children's interactions with peers and significant others (for example, parents and teachers), or through its effect on the parents' ability to use effective socialization strategies, or through its effect on a child's ability to perform academically (3). Therefore, reducing

ADHD symptoms is an important treatment goal for many children and adolescents with CD.

The use of stimulant medication is one of the more successful treatments for ADHD (39). Their effectiveness for reducing conduct problems in children with both ADHD and CD has been shown in several controlled medication trials (40–42). For example, in a very structured classroom setting, methylphenidate (Ritalin) significantly decreased the rate of disruptive classroom behaviours, including verbal and physical aggression, teasing, destruction of property, and cheating (41). In fact, medication was somewhat more effective in reducing the level of these conduct problems than was a very intensive contingency management system. In addition to reducing the conduct problems themselves, stimulant medication has proven to reduce many of the secondary problems often associated with CD effectively, improving peer relations (43) and reducing conflict with parents (44) and teachers (45).

There are several important considerations in the use of stimulant medication to treat CD. Specifically, there is little evidence to date that stimulants reduce conduct problems in children without a comorbid diagnosis of ADHD. Also, the effects of stimulant medication can be variable across children, requiring a very a carefully monitored medication trial to determine optimal dosage for an individual child (39). Further, optimal behavioural effects are often obtained once the medication is titrated up to a higher dose, but this is also associated with a greater number of side effects (40). And finally, in most studies of children with conduct problems, stimulant medication was rarely given in isolation from other interventions, and the medication's primary effect may be to enhance the child's responsiveness to other interventions (for example, it may make the child more responsive to a contingency management program) (39).

Limitations in Existing Treatment Approaches

Although each of the 4 interventions summarized in Table 1 has proven to effectively reduce the conduct problems associated with a diagnosis of CD, even these efficacious treatments have several substantial limitations (7). First, a significant proportion of children with CD do not show a significant response to these interventions, and, for those that do respond, behaviour problems are often not reduced to a normative level. Second, the greatest degree of improvement seems to occur in younger children (prior to age 8 years) with less severe behavioural disturbances. Although this finding highlights the need to focus on preventing CD in young children who are beginning to show problematic behaviours, it also suggests that there is a need for better interventions for older children and adolescents with more severe conduct problems. Third, with some notable exceptions (46), the generalizability of treatment effects across settings tends to be poor. That is, treatments that effectively change a child's behaviour in one setting (for example, in mental health clinics) often do not bring about changes in the child's behaviour in other settings (for example, in schools). Fourth, and also with some notable exceptions (47), improvements brought about in the behaviour of children with CD are often difficult to maintain over time. This seems to be particularly true of older children with severe conduct problems (32) and for children from very dysfunctional family environments (7,48).

Given these rather substantial limitations in the technology for treating CD, there has been an increasing focus on comparing how well these existing treatments match what we know about how CD develops and using this research base to guide the development of innovative treatment approaches (3,10). Each of the 4 treatments described in Table 1 targets basic processes that research has shown to be important in the development of conduct problems. These treatments have, however, ignored 2 important additional characteristics of children with CD that have been the focus of much recent research.

First, research clearly suggests that CD is multidetermined: for most children and adolescents who develop CD, it is the end result of a complex interaction among many different types of causal mechanisms, including individual vulnerabilities (for example, poor impulse control or low intelligence), problems in their rearing environment (for example, poor parental discipline or psychopathology in parents), and stressors in their larger social ecology (for example, living in poor, high-crime neighborhoods or having poor educational opportunities) (3). Further, as mentioned previously, these causal mechanisms do not operate independently of each other but typically operate in a transactional and mutually dependent manner (49). For example, an impulsive child may be much more difficult to parent effectively than a child who has better emotional and behavioural regulation, yet the need for more effective parenting to prevent the development of severe conduct problems is likely greater for the impulsive child (50). Interventions that target only a single type of process will only target one of a myriad potential causal factors; the process being targeted for change is likely to be influenced by other factors that are not part of the intervention. This contention is supported by research showing that treatment trials which employ more than one intervention approach, such as combined PMT and CBST interventions, typically are more effective than trials using either intervention alone (51,52).

Second, research suggests that children and adolescents with CD represent a very heterogeneous group with respect to the causes of their behaviour problems (2). There seem to be multiple causal pathways along which children develop CD, each with multiple causal factors involved and each with unique mechanisms playing critical roles in the development of the child's antisocial and aggressive behaviour. As a result, any

Table 1. Four individual treatments for conduct disorders that have proven success

Treatment	Theoretical	Overview	Key references
Contingency Management Programs	These programs overcome inadequate socializing environments in which optimal contingencies for behaviour were not provided to the child (13). They are needed due to a temperamental vulnerability that makes a child more susceptible to less-then-optimal contingencies (14).	These programs establish clear behavioural goals and gradually shape behaviour by using a very structured system of monitoring and applying appropriate contingencies to motivate behavioural changes.	(15,16,17)
Parent Management Training (PMT)	Inadequate socialization practices are one of the most consistent correlates to CD (3,18). Deficient practices may be a primary causal factor in the development of CD (13). CD development may also be due to the added importance of appropriate socialization in children with certain temperaments (50).	This training has parents develop contingency management programs at home, improves parent–child interactions, and enhances other parenting skills (for example, parent–child communication, monitoring and supervision, and consistent discipline).	(19,20,21,22)
Cognitive-Behavioural Skills Training (CBST)	Many children with CD show deficits in the way they process social information and in how they use this information to respond to problematic social interactions, which makes them susceptible to acting aggressively in social contexts (28).	This training teaches children in a group format to inhibit angry and impulsive responding, to overcome deficits in social cognition, to use more appropriate social problem-solving skills, and to develop more appropriate social skills.	(33,35,36)
Stimulant medication	The antisocial and aggressive behaviour of many children with CD results from a tendency to act impulsively, without thinking about the potential consequences of their behaviour to themselves or others.	Provide a carefully controlled trial of stimulant medication for children with CD who have a comorbid diagnosis of ADHD.	(39,40)

Source (3,73)

Table 2. Developmental pathways to conduct disorder and potential implications for intervention

Developmental pathways to conduct disorder	Characteristics of children in the various pathways	Potential causal mechanisms	Hypothesized implications for treatment
Adolescent-onset	Rebellious, reject traditional status hierarchies, and associate with deviant peers (54,55).	Exaggeration of the normative developmental process of identity development that leads to increase in authority conflicts in adolescence.	Use interventions to promote more adaptive means of developing identity as an autonomous self and interventions that promote contact with prosocial peers and mentors.
Childhood-onset, primarily impulsive type	High rates of ADHD (49), high levels of emotional reactivity (57), low verbal intelligence (62), and high levels of family dysfunction (61).	Multiple causal pathways leading to deficits in response inhibition and susceptability to angry arousal that make a child more likely to act without thinking of the consequences, often in the context of high emotional arousal.	Use interventions that reduce impulsive behaviour and promote anger control, that teach coping skills to compensate for these propensities (for example, problem-solving techniques), and that focus on improving parental socialization practices to encourage skill development and help to maintain it.
Childhood-onset, callous-unemotional type	Preference for thrill- and adventure-seeking activities (for example, low fearfulness) (57), less sensitive to punishment cues relative to cues for reward (14,58), and less reactive to negative emotional stimuli (59).	Temperament characterized by low behavioural inhibition that can interfere with the development of affective components of conscience and the internalization of parental and societal norms.	Intervene early to promote empathy development and internalization of values, and use motivational strategies that capitalize on reward-oriented response style and appeal to self-interest.

Source (3,38)

single intervention, even if it targets multiple causal processes, is not likely to be effective for all children with CD.

Developmental Pathways to CD

Research has begun to define these various developmental pathways more clearly, and this research could be critical for improving our treatment technology. For example, research has fairly consistently documented 2 distinct developmental trajectories along which children develop CD—trajectories that differ in the timing at which the symptoms begin to emerge, the correlates associated with the disorder, and the long-term outcome of the disorder (13,49,53). Children with a "childhood-onset pattern" begin showing severe antisocial behaviour prior to adolescence, show several enduring psychosocial vulnerabilities (for example, neuropsychological impairments, and family dysfunction), and are at high risk for continuing to show a severe pattern of violent and antisocial behaviour into adulthood. Children in the "adolescent-onset" pattern tend to show a more abrupt onset of severe conduct problems coinciding with the onset of adolescence. They also tend to have less dysfunctional family backgrounds, are less likely to have cognitive impairments, are less likely to have problems of impulsivity and overactivity, show a greater desire and ability to maintain social relationships, and show better adult adjustment than their childhood-onset counterparts (54,55).

Table 3. Focus of needs assessment used to individual fast track intervention

Assessment domain	Examples of specific risk factors	Examples of specific protective factors
Identity development and personal adjustment	Display of antisocial attitudes that glorify violence Presence of deviant role models Highly reactive and impulsive behaviour in social situations Affinity for high-stimulation or high-risk activities	Shows positive sense of self, ethnic identity, and future orientation Exhibits capacity for interpersonal sensitivity, especially empathy and concern for others Demonstrate interests and motivations to support vocational development
Family functioning and adult involvement	Family shows high rate of conflict Problems are evident in parental monitoring of youth activities and setting of appropriate limits	Presence of family members or other adults who are prosocial sources of support Existence of opportunities for supervised leisure time activity at school or in the community
Academic achievement and orientation	Evidence of failing grades at school Reports of frequent school suspensions or expulsions Poor school attendance and negative attitudes toward school	Evidence of academic competence Evidence of parental involvement and support for child's academic success Receipt of special services or support at school
Peer relations	High level of exposure to deviant peers Alienation from normative peer groups	Shows interest or involvement in positive peer activities Shows quality friendships with nondeviant peers

Source (74)

These dramatic differences between children in the 2 developmental trajectories have led to theoretical models that propose very different causal mechanisms operating in the development of CD across these groups. For example, Moffitt (49) proposed that children in the childhood-onset group develop CD through a transactional process wherein a difficult and vulnerable child who often also experiences an inadequate rearing environment (see also 13 and 53). This dysfunctional transactional process leads to enduring vulnerabilities in these children that negatively influence their psychosocial adjustment throughout their lives. In contrast, children in the adolescent-onset pathway are not viewed as having enduring vulnerabilities underlying their CD. Rather, their antisocial behaviour is seen as an exaggeration of the normative developmental process of identity formation that takes place in adolescence. Their engagement in antisocial behaviours is conceptualized as a misguided attempt to obtain a subjective sense of maturity and adult status in a way that is encouraged by an antisocial peer group.

While this distinction has gained fairly widespread acceptance, as evidenced by its inclusion in the diagnostic criteria for CD in the most recent version of the DSM (1), research also suggests that a further distinct pathway can be designated within the childhood-onset group (see 2,3,38 for reviews of this research). Specifically, callous and unemotional traits may designate a subgroup within the childhood-onset group whose behaviour is more strongly related to a temperament defined by low behavioural inhibition. Low behavioural inhibition is characterized physiologically by underreactivity in the autonomic nervous system and behaviourally by low fearfulness in novel or threatening situations as well as poor response to punishment cues (56). This is consistent with research showing that youths with CD who score high on callous and unemotional traits show a preference for thrill- and adventure-seeking activities (that is, low fearfulness) (57), are less sensitive to punishment cues than to cues for reward (14,58), and are generally less reactive to certain negative emotional stimuli (59) than are other children with CD. This temperament can be related to the development of callous and unemotional traits in several ways (60). For example, it could place a child at risk for missing some of the early precursors to empathetic concern, which involves emotional arousal evoked by the misfortune and distress of others; it could lead a child to be relatively insensitive to the prohibitions and sanctions of parents and other socializing agents; and it could create an interpersonal style in which the child becomes so focused on the potential rewards of using aggression to solve conflictual encounters that he or she ignores the potentially harmful effects of this behaviour on others.

In contrast, children with childhood-onset CD who do not show high rates of callous and unemotional traits may instead display poor behavioural and emotional regulation that is characterized by very impulsive behaviour and high levels of emotional reactivity. Such poor emotional regulation can result from several interacting causal factors, such as inadequate socialization in their rearing environments (61), deficits in their verbal intelligence that make it difficult for them to delay gratification and anticipate consequences (62), or temperamental problems in response inhibition and emotional regulation (63). Problems in emotional regulation can lead to very impulsive and unplanned aggressive and antisocial acts for which the child may be remorseful afterwards but which the child has difficulty controlling. Such problems can also make a child particularly susceptible to anger due to perceived provocations from peers, leading to violent and aggressive acts within the context of high emotional arousal.

Implications for Treatment

Basic Principles

The developmental pathway model for conceptualizing CD has several important implications for designing and implementing new and innovative interventions. The overarching implication is that there is not likely to be any single "best" treatment for CD. Instead, interventions must be tailored to the individual needs of children with CD—needs that will likely differ, depending on the specific mechanisms underlying the child's behavioural disturbance. To illustrate this in relation to the developmental pathway model outlined above, Table 2 summarizes the 3 pathways, their differential characteristics, the different mechanisms theorized to underlie the behavioural disturbance of children in each pathway, and some potential treatment implications for each subgroup. As can be seen from Table 2, most of the previously summarized interventions with proven effectiveness mainly target the mechanisms that seem most important for those children within the childhood-onset group who show the primarily impulsive subtype. As a result, the effectiveness of these interventions for this particular subgroup may be even greater than is suggested by the treatment-outcome studies. In contrast, interventions targeting the mechanisms involved in the other 2 developmental pathways, especially those involved in the callous-unemotional subtype, have not been systematically developed and tested.

Apart from these specific implications, several general principles for designing and implementing interventions follow from this way of conceptualizing CD (3). First, to select the most efficacious set of interventions for a child or adolescent with CD, one must understand the multiple causal processes that can be involved in its development. For example, if the developmental progressions that often characterize children and adolescents with CD are recognized, interventions can be implemented as early as possible in the developmental sequence. In addition, this knowledge base can help to determine which processes need to be assessed to develop an understanding of the mechanisms which may be involved in the development of CD for a particular child and, subsequently, can guide decisions as to the most important intervention targets.

Second, this flexible approach to treatment requires that there be a clear, comprehensive, and individualized case conceptualization to guide the design of a focused and integrated treatment approach. A case conceptualization is a "theory" as to what are the most likely factors that are involved in the development, exacerbation, and maintenance of conduct problems for an individual child or adolescent. It uses the research on the developmental pathways to CD and attempts to apply it to an individual child with CD. The case conceptualization also specifies any other problems that may be important targets for intervention, such as secondary problems that are caused by a child's behaviour (for example, peer rejection) or comorbid psychological disorders. Given the myriad of factors that can contribute to the development of CD and the pervasive effect that CD can have on children's psychosocial adjustment, an adequate conceptualization often requires a comprehensive psychological evaluation (3).

Third, successful intervention for children and adolescents with CD typically involves multiple professionals and multiple community agencies all working together to provide a comprehensive and integrated intervention. Professionals must be able to recognize when the needs of children and their families go beyond their area of expertise and be willing and able to make appropriate referrals for intervention. Further, comprehensive and multidisciplinary treatment approaches require strong case coordination over extended periods of time to ensure that the various treatment components are provided in an intensive, integrated, and complementary fashion, rather than in a weak, fragmented, and competing manner.

This comprehensive and individualized approach to intervention outlined here and elsewhere (3) has not been subjected to controlled outcome evaluations. Two approaches to intervention that have been used in the treatment of children with CD are, however, consistent with many of the principles outlined above and have data on their effectiveness. Although not directly based on the developmental model of CD, their flexible and individualized treatment approaches can easily integrate and incorporate these principles.

Families and Schools Together (FAST Track)

The FAST Track Program was developed by the Conduct Problems Prevention Research Group (64) to be a long term, multicomponent, and multisite intervention early in children's development of conduct problems. The program was designed to target children who were showing severe conduct problems at the time of school entry and to intervene intensively and continuously to prevent these problems from worsening over development. The basic structure of the FAST Track intervention involved a very intensive intervention during the kindergarten year that integrated several intervention components designed to promote competence in the family, child, and school in a coordinated and integrated fashion. All these interventions were community-based, primarily being provided at participating schools. After this initial intensive intervention, children and their families were followed continuously, with periodic assessments; additional interventions tailored to the specific needs of the individual child and family were provided as needed.

The initial intensive intervention involved several treatment components. First, a 22-session PMT program was conducted in a group format. In addition to the previously described typical components of most PMT programs, the FAST Track parenting intervention also included components to help parents foster their children's learning, to promote positive

family–school relations, and to help parents develop better anger-control and problem-solving strategies for themselves (65). Second, the FAST Track Program included a CBST intervention that focused on helping the child develop anger-coping and social problem-solving skills and promoted social-skill development (35). These skills were taught in a small group setting and included weekly 30-minute guided play sessions with a classroom peer, during which the child could practise the skills taught in the group, to promote generalization of skill use. Third, the FAST Track Program included an academic tutoring component designed to improve academic skills, especially beginning reading skills, and to encourage parental involvement in their child's academic progress. Fourth, the FAST Track Program included a case-management component in which a case manager visited a family's home biweekly to help the parents implement improvements in family functioning targeted by the PMT intervention. This component also helped families deal with practical problems, encouraged the development of community and neighborhood supports, and generally promoted family organization and stability.

This initial intensive intervention program has several of the components that fit with the previously described intervention model for children with CD. It intervenes early in its development and targets processes that research indicates are important in the development of severe conduct problems. The intervention is community-based (that is, based in the schools), which promotes child and parent engagement in the intervention and allows the intervention to foster community supports that will maintain any changes brought about in the child's behaviour during the intervention. Most important, it recognizes the multidetermined nature of CD by providing a comprehensive intervention that targets many different potential causal processes which may be leading to or maintaining a child's antisocial and aggressive behaviour.

In one area, the initial FAST Track intervention did not reflect the principles outlined above: for the most part, the initial intervention was not individualized for each child and family. The notable exceptions were the case-management and school-tutoring components, which did include some flexibility so that they could be tailored to the needs of the individual case. This individualization, however, was reflected to a much larger extent in the later stages of the FAST Track intervention. After the initial intensive phase, a case manager maintained regular periodic contacts with the child and family and 3 times each year assessed the child and the family's needs in 4 areas: identity development and personal adjustment, family functioning and adult involvement, academic achievement and orientation, and peer relations. Table 3 provides a summary of these areas of need and a description of items used in this assessment. The most appropriate intensity level and intervention type for each child and family was determined based on this needs assessment, and the family was provided with the needed services or referred to professionals who could provide them.

The data on the long-term effectiveness of the full FAST Track Program are not yet available. The program was, however, designed with a strong treatment-evaluation component, which included randomly assigning schools to treatment and control conditions and systematically collecting both outcome and treatment process measures throughout the intervention. Also, information on the effectiveness of the initial intensive intervention is available (66). Specifically, after the first year of the FAST Track intervention, children in the treatment group, compared with control children, showed evidence of better social-coping skills and more advanced word-attack skills. In addition, these improved skills were reflected in more positive peer relations and better grades at school. Parents in the intervention group showed more warmth and positive involvement with their children; used less harsh, and more appropriate and consistent, discipline; and showed more positive school involvement; than did parents in the control condition. On the critical outcomes of child aggressive and disruptive behaviours, assessed by 10 different outcome measures (which included parent and teacher report, peer nominations, and behavioural observations), the intervention groups showed significantly better scores on 4 of the 10 measures, with an average effect size of 0.21 (range 0.02 to 0.53). These changes on conduct problem outcome measures may not be as strong and consistent as would be expected from the intensity of the intervention; however, it is quite possible that the changes in family, child, and peer processes brought about by the intervention had not yet been translated into behavioural changes at this initial 1-year evaluation.

Multi-Systemic Therapy (MST)

MST was originally developed as a general approach to intervention for psychopathological conditions (67), but it has been applied extensively to treat severe antisocial behaviour in children and adolescents (68). MST is an expansion of a systems orientation to family therapy. In systemic family therapy, problems in children's adjustment, such as CD, are viewed as being embedded within the larger family context. MST expands this notion to include other contexts, such as the child's peer, school, and neighborhood contexts. Although MST is not explicitly developmental in orientation, as reflected by its lack of emphasis on the individual child's characteristics that may contribute to the development of CD and that may play a role in shaping these contexts. It does, however, emphasize a comprehensive and individualized approach to intervention that is consistent with the principles outlined above for intervening with children who have CD.

MST involves an initial comprehensive assessment that seeks to understand the level and severity of the child's or adolescent's presenting problems as well as the systemic context of these problems. The information gained from the assessment

is used to outline an individualized treatment plan based on the specific needs of the child and his or her family. To illustrate this individualized approach, and the comprehensive nature of MST, Henggeler and Borduin (67) reported on the treatment of 156 juvenile offenders (mean age 15.1 years), all with multiple arrests (mean, 4.2). Eighty-eight offenders and their families underwent MST ranging in length from 5 to 54 hours (mean, 23 hours). In addition to this variation in intensity, the way in which these hours were used varied, depending on the needs of the clients. Eighty-three percent of the MST group participated in family therapy, and 60% participated in some form of school intervention, which included facilitation of parent–teacher communication, academic remediation, or help in classroom behaviour management. In 57% of the cases, there was some form of peer intervention, which included coaching and emotional support for integration into prosocial peer groups (for example, scouts and athletic teams) and direct intervention with peers. In 28% of the cases, the adolescent had individual therapy that typically involved some form of CBST intervention. Finally, in 26% of the cases, the adolescent's parents became involved in marital therapy.

Unlike the individual interventions described in Table 1, and even to some degree the FAST Track Program, MST does not emphasize the use of specific techniques. Instead, it emphasizes several principles that follow from its orientation to intervention. These principles include the following: 1) the identified problems in the child are understood within their broader systemic context; 2) therapeutic contacts emphasize positive (strength-oriented) levers for change; 3) interventions promote responsible behaviour among family members; 4) interventions are present-focused and action-oriented, targeting specific and well defined problems; 5) interventions target sequences of behaviour within and among multiple systems; 6) interventions must be developmentally appropriate; 7) interventions are designed to require daily or weekly effort by family members; 8) intervention effectiveness must be evaluated continuously from multiple perspectives; and 9) interventions are designed to promote maintenance of therapeutic change by empowering caregivers (68). MST involves a strong system of intensive supervision by the therapists implementing the treatment. They must determine how these principles should be implemented to meet the needs of each individual case, and they must ensure that the principles are followed throughout the intervention. Also, unlike the FAST Track Program, MST is designed to be a time-limited intervention, usually between 3 and 5 months, depending on the family (68). Its goal is to establish sources of support in the child's and family's natural context that will help to maintain any changes brought about by the intervention over longer periods of time. Consistent with the FAST Track Program, however, MST is designed to be community based, with services being provided, as much as possible, in the family's natural environment (for example, at home, in school, or in the neighborhood).

One important contribution of MST to the treatment-outcome literature is its ability to demonstrate that these individualized and community-based interventions can be rigorously evaluated through controlled treatment-outcome studies. The initial findings from the studies on MST's effectiveness for reducing antisocial and aggressive behaviour in even very severely disturbed children have been quite promising. For example, in a controlled treatment-outcome study undertaken by doctoral students at a university-based outpatient clinic, 88 adolescent repeat offenders underwent MST. Their outcomes were compared with a control group of 68 offenders who received traditional outpatient services, typically focusing on individual psychotherapy (69). At a 4-year follow-up, only 26% of the youths who underwent MST were rearrested, compared with 71% of the control-group adolescents. In a second outcome study of MST, intervention was provided by master's level therapists at a community mental health centre (70). The sample included adolescents who had been adjudicated as delinquent and had multiple arrests. These adolescents were randomly assigned to receive either MST or the standard services provided by the juvenile justice system. The group receiving MST showed one-half as many arrests and spent an average of 73 fewer days incarcerated than did adolescents who received standard services. These 2 studies illustrate the very promising nature of MST for treating heretofore very difficult-to-treat persons with CD, namely, adolescent juvenile offenders with multiple arrests. Henggeler and others provide examples of several additional ongoing outcome studies of MST (68).

The Future of Interventions for Children and Adolescents with CD

This review illustrates that the traditional view of mental health treatment, in which the optimal treatment for persons with a disorder is uncovered through treatment-outcome research and then applied to all persons with the disorder, is not consistent with our most current understanding of the development of CD. Not surprisingly, treatments based on this view have proven to be woefully inadequate. Instead, there is emerging evidence that, to be effective, treatment must be comprehensive, taking into account the myriad factors within the child and within his or her social context that can cause and maintain CD symptoms. Moreover, treatment must be individualized, taking into account the different pathways along which children may develop CD. The FAST Track Program and MST are examples of 2 different models for implementing this type of intervention. The development and use of this approach to intervention is still, however, in a very early stage; there are several important elements that could increase their effectiveness and lead to their more widespread use.

First, a key aspect of the interventions that have proven even minimally effective in treating CD is that they were based on our understanding of the causal and maintaining factors for its symptoms; there is a clear dependence between advances in research on the causes of CD and the development of more effective interventions. As the various pathways along which children and adolescents develop CD become more fully understood, our ability to design interventions specifically tailored to alter these processes, or to modify their consequences, is also likely to be enhanced. For example, in the previously outlined developmental model of CD, distinctions have only recently been made between those children with callous-unemotional traits and those without such traits. Therefore, studies of the unique processes involved in the development of CD for children in the 2 groups are equally recent. As a consequence, most of the developed and tested interventions do not address the processes that may be most important for children with callous and unemotional traits. Therefore, support for basic research into the causal pathways that lead to CD needs to be considered a priority, and future interventions need to be responsive to advances in this research.

Second, service delivery models to implement comprehensive and individualized treatment approaches are only beginning to be tested, and much more needs to be done to determine how they can be designed in the most effective and cost-efficient manner. Critical components of program development are knowing what processes to consider in designing an intervention plan, knowing how to design a system to assess these processes, and knowing how to use the assessment results to meaningfully inform treatment decisions. Unfortunately, the assessment and diagnosis technology has not always been responsive either to advances in basic research or to the need to make assessment results relevant to practice (71). Similarly, the training and the variations in organizational structure required to establish a system for implementing effective, cost-efficient, comprehensive, and individualized interventions need to be systematically studied (68). Finally, there is emerging evidence that these interventions are most effective when they are community-based and provided outside traditional mental health service-delivery settings. This implies that mental health practitioners need to become better trained and more knowledgeable about establishing community linkages for service provision.

Third, dissemination of knowledge about effective treatments is critical, as is provision of supportive services in the design of care systems that reflect this knowledge. This treatment approach does not fit with many political philosophies regarding the treatment of antisocial youths, and it differs in many respects from the way many mental health professionals were trained to deliver services. As a result, a concerted effort is needed to inform both the professional and lay community about the current status of intervention for CD.

Clinical Implications

- Interventions for children and adolescents with conduct disorder (CD) must be implemented cautiously because many treatment approaches have proven ineffective, and some have even proven to have harmful effects.
- Designing interventions for youths with CD must be guided by basic research; most successful treatments have focused on processes that research has shown to be important in the development or maintenance of the disorder's symptoms.
- Successful interventions need to be comprehensive, addressing the many different factors that can lead a child to develop CD, they also need to be individualized, addressing the different causal trajectories that can lead to this disorder.
- Multi-Systemic Therapy and the FAST Track Program are 2 models of an individualized and comprehensive approach to intervention that can be used to guide current practice.

Limitations

- More research is needed to understand the various causal pathways involved in the development of CD to guide individualized interventions.
- More research is needed to design service delivery models that are both effective and cost-efficient.
- Greater effort to disseminate model treatment programs is needed, given that effective interventions use a treatment approach in which many mental health professionals were not trained and given that political values may influence the selection of alternative treatment methods in some settings.

Supporting efforts to use this information in designing interventions are also needed. An example of such an effort is the Blueprints for Violence Prevention program, which identified 10 violence-prevention programs that met very rigorous scientific standards of program effectiveness and 20 promising programs with evidence of effectiveness, but which require further support (72). The Blueprints provide practical descriptions that allow states, communities, and agencies to evaluate a program's effectiveness, estimate the implementation cost, assess their organizational capacity to implement it, and assess potential barriers to implementation. The Blueprints also list references for the necessary contacts for each program (72).

In recent years, there have been many advances in our understanding of what causes of severe antisocial and aggressive behaviour and in our development of effective treatments for youths with such behaviours, many of whom are diagnosed with CD. These interventions, however, require models than the ones in which many mental health professionals were trained. In addition, the goals of these interventions may be different from the goals of interventions based on political ideologies of how aggressive and antisocial behaviour should be treated. Given the severe and impairing nature of CD, and the social costs that result from the behaviours of youths with this disorder, it is imperative that mental health professions promote interventions that reflect these advances and contribute to the development of further advances in both research

and service delivery. The conclusion that many children with CD are untreatable is not supported by the available evidence; instead, it seems that those in the field are only now beginning to understand how best to treat them. Granted, the documented evidence for treatment success is still minimal, and this optimism may prove to be unfounded. Nevertheless, there clearly is a definite framework within which mental health professionals can design intervention programs, and this fact alone provides great cause for optimism.

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Résumé— Interventions efficaces auprès d'enfants et d'adolescents souffrant des troubles de conduite

De nombreux types différents d'interventions ont été utilisés pour traiter les enfants et les adolescents souffrant des troubles de conduite (TC). Malheureusement, la plupart se sont révélés d'une efficacité limitée et, dans certains cas, ont même provoqué des effets iatrogènes. Cette efficacité limitée est principalement attribuable à l'incapacité de la plupart des traitements d'aborder directement les mécanismes causals du développement des TC. Nous avons examiné quelques exceptions qui ont fondé les interventions sur la recherche disponible et qui se sont révélées d'une certaine efficacité pour réduire les problèmes de conduite chez les jeunes souffrant de TC. Et surtout, un modèle d'intervention est présenté. Ce modèle souligne que les interventions auprès des jeunes souffrant de TC doivent être complètes, c'est-à-dire qu'elles doivent prendre en compte la foule de facteurs, à la fois chez l'enfant et dans son milieu social, qui peuvent causer et maintenir les symptômes de TC. En outre, les interventions doivent être individualisées; elles doivent tenir compte des différents mécanismes par lesquels les enfants peuvent développer les TC. Deux approches d'intervention conformes à ces principes sont étudiées, comme le sont les grandes orientations de l'évolution de la technologie des traitements pour les jeunes souffrant de ces troubles.

IN REVIEW

Conduct Disorder: A Biopsychosocial Review

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Objective: *To review published works on the epidemiology, risk factors, protective factors, typologies, and genetic aspects of conduct disorder (CD).*

Method: *Findings from refereed journal articles and current texts in the field are briefly summarized.*

Results: *CD is commonly encountered in clinical practice. Factors strongly predictive of future delinquency include past offenses, antisocial peers, impoverished social ties, early substance use, male sex, and antisocial parents. Factors that moderately predict recidivism include early aggression, low socioeconomic status (SES), psychological variables such as risk taking and impulsivity, poor parent–child relationships, poor academic performance, early medical insult, and neuropsychological variables such as poor verbal IQ. Mildly predictive variables include other family characteristics such as large family size, family stress, discord, broken home, and abusive parenting, particularly neglect. Protective factors include individual factors such as skill competence (in social and other arenas), adult relationships, prosocial and proeducational values, and strong social programs and supports.*

Conclusions: *We know a great deal about psychosocial risk factors for CD. Some research into protective factors and genetic contributions exists but is in its early stages. Future work will increase our knowledge about subtypes, developmental pathways, and CD treatment.*

(Can J Psychiatry 2001;46:609–616)

Key words: *conduct disorder, delinquency, risk factors, protective factors, genetics, subtypes*

> For our discussion is on no trifling matter, but on the right way to conduct our lives.
>
> *Plato, The Republic*

This article summarizes relevant findings on epidemiology, environmental contributions, subtypes, and genetic aspects of CD. Other important aspects, such as assessment, comorbidities, neuropsychiatric vulnerabilities, and treatment have been recently well-reviewed (1–3). This paper updates the reader on current thinking about psychosocial risk and protective factors and hints at emerging biological risk factors, such as genetics. We consulted original articles and reviews in both Medline and Psycinfo, particularly those published over the last 15 years. For further information, please consult the original referenced works. Clinical and public health implications are indicated, where appropriate.

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Definitions

CD is 1 of 3 disruptive behaviour disorders (4), the others being oppositional defiant disorder (ODD) and attention-deficit hyperactivity disorder (ADHD). As indicated in the DSM-IV, CD connotes a severe externalizing disorder comprising serious aggressive and antisocial behaviours such as fighting, bullying, cruelty, robbery, forcing sexual activity, firesetting, theft, conning, truancy, and other rule violations. Antisocial behaviour describes actions contrary to the rights of others and rules of society. Adolescent antisocial behaviour that breaks the law (and gets caught) may result in contact with police and the courts; the terms "delinquent" and "young offender48 would then apply. Thus, CD represents a constellation of antisocial behaviours; a subgroup of youths with severe CD will be delinquents (5). We will also use the terms "aggression" and "violence" in this overview. Aggression is defined as outward destructive behaviour that results from the confluence of longer-term factors (for example, biological, psychological and personality, family, peer, school, and community), short-term influences (for example, internal states of anger, boredom, or intoxication) and situational opportunity. Violence—a particular form of overt and intentional aggression—uses or credibly threatens to use physical force such as beating, kicking, choking, using a weapon, forcing sex, and throwing objects.

Epidemiology

CD is the most common reason for psychiatric evaluation of children or adolescents (6). Between 30% and 50% of all child psychiatry referrals tend to involve CD (7). The Ontario Child Health Study indicated that for ages 4 to 16 years, 5.5% suffered from this condition (8).

Not all youths with CD have a criminal record, and not all youths with a criminal record have CD. Crime statistics therefore can be informative but extremely controversial. Inflated statistics could result from population growth, police reporting practices, or growth of police forces. Equally possible, underreporting may occur. This could be due to the existence of special youth crime units and more community-based policing, along with conflict resolution strategies rather than formal prosecution. In any event, Statistics Canada data indicate that youth offences increased steadily until 1991 but have declined since. In 1986, there were 136 787 charges laid against youth in Canada; in 1991, there were 171 673; by 1994, these had declined to 143 337. By 1998–1999, courts heard 106 665 adolescent cases in Canada. While this decline in overall numbers is comforting, another disturbing trend is occurring. The proportion of youth charged with violent offences seems to be increasing. In 1986, 408 youths per 100 000 were charged with a violent crime (that is, homicide, attempted murder, assault, sexual offences, abduction, and robbery). In 1995, the rate was up to 938, representing a growth rate of 13% per annum. We expect a 31% increase in the juvenile population over the next 2 decades (9). Accordingly, CD is and will continue to be a fairly significant public health problem, warranting much attention from both the mental health community and the juvenile justice system.

Several researchers have explored CD subtypes and developmental pathways to antisocial or violent behaviour. One such example is life-course persistent (LCP) offending versus adolescence-limited antisocial behaviour (10). The LCP pathway involves early onset, active and diverse delinquency, increasingly serious crime, and continuation into adulthood. It accounts for about 5% to 8% of the offender population, but most offenses. The behaviours to watch for in children and youth are as follows: biting and hitting at age 4 years; shoplifting and truancy at age 10 years; selling drugs and stealing cars at age 16 years; robbery and rape by age 22 years; fraud and child abuse by age 30 years; addictions, underemployment, drunk driving, violent assault, domestic violence, child neglect and abandonment; and psychiatric illness past age 40 years. Adolescence-limited offenders tend to have less preadolescence history of conduct problems. They can offend with peers but may behave well in school and at home. Only 25% of these late starters continue their delinquent careers in adulthood. Similarly, a recent Canadian study found that 4% of kindergarten boys continued through adolescence to have a high rate of aggression and that the 2 most predictive variables were teenage pregnancy and low educational achievement in the mother (11). Thus, more and earlier psychosocial intervention—especially with the early-onset aggressive and antisocial children and their high-risk parents—would hopefully save the child, family, and society much emotional, physical, and financial damage.

Psychosocial Risk Factors

Risk factors are those individual, familial, or environmental factors that have been shown in research to increase a child's risk of future conduct problems. While 20% to 30% of adolescents commit a violent act, only 5% to 8% of males and 3% to 6% of females are responsible for most such acts (12). Knowledge of these youths' characteristics—especially if used to inform effective interventions—could be quite therapeutic and preventive in reducing their suffering and associated social damage.

A huge body of longitudinal research that discusses risk factors exists (for example, 13–16). There are, however, a great variety of methodologies, samples, measurements, and outcome variables. Accordingly, metaanalyses of these prospective studies also exist (17,18). Clinical implications will be discussed where relevant.

Strongly Predictive Risk Factors

Prior Antisocial Behaviour. Prior antisocial behaviour (for example, stealing, property destruction, early sexual intercourse, and drug selling) is probably the strongest predictor of future offending for males, but not necessarily for females (19). Early onset of delinquent behaviour has consistently predicted more chronic and serious violence and offending in many studies (20).

At any age of childhood, past charges, aggression, and violence raise one's clinical suspicion of future delinquency risk. In one study, 50% of boys convicted for a violent crime between age 10 and 16 years were convicted again of violence by age 24 years, compared with 8% of those not convicted as youths (21). Also, discipline problems at ages 8 to 10 years, self-reported conduct problems by 14 years, regular cigarette smoking by 14 years, and sexual intercourse by 14 years predicted later violence in males (22). An early history of problematic behaviours and delinquency should therefore be taken fairly seriously, because these high-risk children and youths tend not to grow out of these behaviours and may require more intensive and sustained intervention.

Antisocial Peers. By adolescence, delinquent peers contribute greatly to the overall spread of antisocial behaviour and delinquency. Youths with CD tend to have problematic families in the first place, and they are also rejected frequently by prosocial peers. As they separate, they can become attached to delinquents' mutual friends or to other youths with longer criminal histories (23). Further, gangs are also responsible for a significant proportion of serious and violent offenses (24); gang involvement appears to add an additional risk level

above and beyond delinquent peers and groups. Clinically, it may be easier to steer children toward prosocial peers and activities before adolescence.

Social Ties. This category includes low popularity and few social activities, especially in adolescence. Children who suffer from early peer rejection are more likely to be bullied, disruptive in class, and socially aggressive in interactions (25). These children often already have higher levels of aggressive and externalizing behaviours; thus, peer rejection likely increases their risk for future conduct difficulties (26). The clinical implications are several. Getting a sense of the youth's extracurricular hobbies, interests, and activities is important and will help build a therapeutic alliance. In addition, exploring personality traits, such as neuroticism or extraversion, could be relevant, as in the Five Factor Model of personality (27). The presence of cluster A personality traits—particularly paranoid or schizoid—should be ascertained. Caution, however, should be used in diagnosing personality disorder in children and youth.

Substance Use. Early cigarette smoking and alcohol or other substance use, particularly before age 12 years, confers independently a greater risk of future antisociality. This may be due to any of 3 interrelated types of associations between drugs and crime: psychopharmacologic, economic compulsive, and systemic (28). Psychopharmacologic mechanisms increase violent outcomes more directly, either through intoxication or withdrawal. Economic-compulsive associations refer to situations in which children (or adults) commit a crime to obtain money to purchase drugs. Systemic issues relate to involvement in the drug trade (for example, dealers' disputes, problems with informers, and punishment for adulterated drugs or unpaid debts). The 3 mechanisms outlined above support the notion that substance use leads to antisocial behaviour. There are, however, many other postulated explanations: youths with CD may self-medicate with marijuana if they have ADHD; situational factors such as bars or raves may increase the propensity for aggressive behaviours in vulnerable youths. No single model has yet been found to neatly explain the relation between substances and conduct problems, and we likely need to develop more complex interactive models, because the rate of comorbidity between substance abuse and CD is as high as 91% (29).

Male Sex. In studies and statistics, being male has consistently been shown to confer much higher risk of CD (the ratio of boys to girls is 5:1). Why the sex difference? Psychological explanations include differences in socialization and advanced adaptive development in girls. There are, however, likely hormonal, anthropological, and evolutionary aspects, as well. Even so, so-called indirect aggression or relational aggression likely applies more to girls (30). This involves such acts as spreading rumours, shunning or ostracizing peers, and conscripting others to "get even" physically. Criminology data indicate that males are more likely to be arrested for violent crimes, and females for "status offenses" (for example, truancy, prostitution, running away, underage drinking, or purchasing tobacco). When aggressive, girls are more likely to fight with a parent or sibling than with strangers—34% vs 9% for boys (31). Consequently, a clinical search for interpersonal or covert antisocial behaviours in females may yield additional information.

Antisocial Parents. Having a convicted mother, father, or sibling significantly predicts boys' convictions. Further, this finding holds both for self-report and official criminal records (32). One study that used the Danish National Register found that men aged 18 to 23 years whose fathers had at least 2 criminal convictions were more likely to commit violent criminal acts (33). Whether this reflects genetic or social learning mechanisms, or both, is being debated (34).

Moderately Predictive Risk Factors

Early Aggression. Many have noted stability and continuity in early-onset aggressive behaviours in males (35). Having an aggressive style of behaviour and interaction may, however, have earlier precursors. Persistent attention-seeking at age 12 months has been found to relate to noncompliance at age 18 months, which then correlates with aggression at age 24 months. This has been related to maternal reports of externalizing problems at age 36 months (36).

Interestingly, in one study, two-thirds of 10- and 13-year-old boys with high teacher-rated aggression scores had, by age 26 years, a criminal record of violence. This correlation did not hold up for girls (37). Some researchers have opined that by age 8 years, children have an idiosyncratic aggression level that is fairly stable over time (38). The opposite end of the debate is, however, that aggression is not a stable trait and is entirely influenced by situational and contextual factors (39). Clinically, a careful history of the developmental course of various oppositional, aggressive, and conduct behaviours may suggest an LCP trajectory and its implied need for intensive treatment and resources.

Low Family Socioeconomic Status (SES). So many studies have found correlations between poverty and crime that it is considered by some to be a given that poor families and neighbourhoods are associated with delinquency. This is highly contextual and not necessarily causal, however. In the US, we found a difference between poor families in inner-city neighbourhoods and "other urban poor" neighbourhoods, with inner-city communities having delinquency rates over 2.5 times the national average (40). Further, families characterized by social isolation, broken homes, sparse networks, weak social supports, and poverty are much more likely to physically abuse children, increasing their risk of aggression (41).

From a public health perspective, it has been found that good parenting, stable family environment, and good early health and development can mitigate against the effects of impoverishment and unemployment; thus, the perceived association of poverty with crime may be justified only if there are biological and psychosocial vulnerabilities operating together (42).

Psychological Characteristics. These include a high activity level, risk taking, impulsiveness, and a short attention span. Together, these features were associated in a Swedish study of boys at age 13 years with a much higher rate of arrest for violence by age 26 years than was found for boys without these characteristics (43). A relevant finding in the adult literature is that antisocial personality disorder (APD) is associated with traits such as high novelty seeking (with impulsive and tempestuous behaviour), low harm avoidance (for example, fearless and daring), and low reward dependence (that is, aloof and independent) (44).

Prospective and retrospective studies have linked hyperactivity and impulsivity to later violent behaviour (45,46). In fact, several studies suggest that youths with comorbid ADHD and CD in childhood are at high risk for chronic and persistent conduct and attention problems (47,48). This comorbidity therefore should be detected and aggressively treated in an attempt to reduce future risk.

Parent–Child Relationships. These include discipline practices (for example, mixed, inconsistent, or punitive), minimal involvement, poor supervision, low emotional warmth, and a negative attitude toward the child. Numerous studies have shown that these parenting practices are consistently linked with later delinquency and substance abuse (49). Similarly, low involvement and interaction—particularly between sons and fathers at age 8 years—was also found to predict later violence (22). In another study, a high degree of "negative family labelling" of the youth (a measurement of family bonding) was found to be associated with sexual aggression in later teenage years from ages 13 to 19 (50). A positive parenting style has been found to improve the outcome when environmental risk factors such as poverty exist (51). Clinically, family-focused treatments, such as Functional Family Therapy, Parent Management Training, or Multisystemic Therapy are among the most promising to reduce recidivism (1).

School Attitude and Performance. This category includes a low interest in education, dropping out, low school achievement, and truancy. Poor academic achievement in elementary school, and particularly in high school, has been related consistently to later conduct difficulties (52).

There has been a specific linkage demonstrated between attention deficits and underachievement (53). Thus, ruling out comorbid subtypes of ADHD at any age is imperative. Reviewing school records is time-consuming but invaluable. We recommend intelligence and achievement testing (54). If possible, neuropsychological testing for verbal and executive function deficits, among other developmental delays, is also indicated.

Medical and Physical Conditions. These include pertinent findings in developmental history, medical conditions, and physical development. Prenatal and delivery complications somewhat predict, in particular, later violent offending (55). In a study to test biosocial interactions, birth complications (for example, forceps extraction, breech delivery, umbilical cord prolapse, preeclampsia, and long birth duration) greatly increased the risk of violent offending in adulthood, but only when associated with early child rejection (for example, unwanted pregnancy, attempted abortion, and institutional care for at least the first 4 months of life) (56). This has profound medical and public health implications. In similar interactional style, large body height and weight at age 3 years, coupled with fearlessness and novelty-seeking traits, were found to predict aggression at age 11 years (57). Other medical and psychiatric conditions worth ruling out include minor physical anomalies, head injuries, neurological conditions, bipolar disorder, dissociative disorders, and paranoid disorders (1,2,86).

IQ. Most studies have found that IQ scores of children with CD are on average 8 points lower than those of nondelinquent children, even when other variables such as SES, ethnicity, academic achievement, and motivation are controlled. Another consistent finding is that performance IQ is greater than verbal IQ (58). This strongly suggests specific language difficulties and possible neuropsychiatric dysfunction in children and youths with CD. Actually, when the subset of early-onset delinquents was examined, they were found to have IQ scores 17 points lower on average than those of nondelinquent subjects, a finding also observed in children with early brain damage (59,60). Thus testing, where available, could aid assessment and inform us about needed educational resources.

Mildly Predictive Risk Factors

Other Family Characteristics. These refer to factors such as high family stress, large family size, and marital discord. An early finding was that boys raised in high-conflict families were more likely to be convicted later of a violent crime (61). More specifically, being exposed to violence between parents increases the risk of later violence in the children when adults (62). This, however, has not always been found: in the Seattle Social Development Study, family conflict at age 10 years was not correlated with violence, according to self-reports, at age 18 years (63).

Broken Home. This category includes both family breakdown (for example, divorce) and separation from parents for other reasons. The relation between broken homes and child or youth aggression is complex, because families separate for many reasons. Notwithstanding this, parent–child separation

before age 10 years has been found in more than 1 study to predict convictions for violence in later adolescence and up to age 21 years (64).

Abusive Parents. This includes all categories such as emotional, physical, and sexual abuse, as well as maltreatment and neglect. It has been consistently found that, compared with those without an abuse history, adults who were sexually abused as children were less likely to commit violent crimes than were control subjects. Adults who were physically abused were slightly more likely, while those neglected as children were the most likely, to be arrested for violence (65). Thus, neglect—the most common and highest-risk form of abuse—should be seen as a serious public health issue with implications for violence prevention.

Risk Factors: Synthesis

No single factor accurately predicts later conduct problems. Factors aggregate to produce increased vulnerability in these children. An excellent example is the Cambridge Study in Delinquent Development (66), in which 411 boys in working-class London, England, were followed from age 8 to 40 years. Vulnerability scores were calculated based on 5 risk factors: low family income at age 8 years, large family size by age 10 years (4 or more biological siblings), low nonverbal IQ at age 8 to 10 years, and poor parenting (harsh or inconsistent discipline and parent conflict) at age 8years. For those with no risk factors, convictions for violence were increased by only 3%, compared with control subjects. Having 4 to 5 factors present, however, increased one's risk by 31%. Thus, the interaction between individual and environmental risk factors really determines the overall variance in the outcome of these youths.

Protective Factors

Protective factors are not simply the absence or opposite of risk factors, although this is sometimes the case. In fact, despite exposure to multiple known risk factors, many children avoid serious antisocial behaviour (67). Protective factors are best defined as those variables that offset the effects of risk factors. Research, however, has largely ignored these factors in favour of elucidating risk. It is quite likely that more emphasis on these variables could significantly influence practice and policy.

Individual Protective Factors (13,68)

Female Sex. Being female may be protective via different parenting or socialization patterns; also, girls generally mature and acquire skills more quickly.

High Intelligence. This is as measured by standard IQ testing. Such testing, however, has been criticized as too limited with respect to types of intelligence and cultural diversity.

Positive Social Orientation. This would likely include the absence of antisocial attitudes and cognitive biases, such as interpreting social cues as necessarily hostile or threatening.

Resilient Temperament. This usually means possessing good coping skills and the ability to endure stress, hardship, or trauma without mental decompensation.

Competence at a Skill. Attaining at least 1 area of good ability at a skill, hobby, or interest has been noted to be protective, perhaps leading to other prosocial activities and interactions.

Anxiety. According to some research, having anxiety, worry, and guilt tends to protect against the development of antisocial behaviour.

Social Factors (67,69,70)

Warm, Supportive Relationships with Adults. Positive interactions, warmth, or mentorship from adults has been found to be greatly beneficial in influencing away from CD.

Individual and Family Commitment to Social Values Such As Prosocial Norms or Academic Achievement. Aspiring to universal values such as positive social interactions with others and school success helps children decrease their risk of conduct problems and increases their achievements and opportunities.

Recognition for Involvement in Positive Extracurricular Activities. Not just being involved in prosocial activities but actually being acknowledged, and even rewarded, helps perpetuate such positive behaviour.

Societal Factors (71,72)

Increased Economic Equality and National Social Program Support. Developed countries with larger social-program spending tend to have lower homicide rates.

Social Organization. Strong and stable community institutions (for example, church, neighborhood organizations, and extended families), as opposed to disorganized, chaotic, and crumbling communities, tend to be protective.

Genetic Contributions

It is well known that psychiatric disorders often run in families. Simple mendelian inheritance, however, is not found with such complex conditions. Given the diagnostic heterogeneity of CD, a more appropriate phenotype, such as aggressivity or violence, is used in research. Genetic studies can be broadly categorized into 2 types: those using twin, adoption, or familial methods, and those using molecular biology methods. Discussing the science behind these is beyond the scope of this article. One excellent representative twin study looked at the relation between child behaviour checklist (CBCL) syndromes such as mood and anxiety disorders, attention deficits, antisocial behaviour, and aggressive behaviour (73) and temperament ratings using the Emotionality, Activity,

Sociability Temperament Survey (74,75). The study looked at heritability (the relative impact of genes as opposed to environment) and operated under the assumption that temperament consists of stable and largely genetically determined traits. Emotionality in boys and high activity scores—particularly in younger children—were found to predict aggressive behaviour. Further, aggressive behaviour, but not general antisociality, was found to have significant association with temperamental traits and higher heritability. Among the drawbacks of such studies, however, are reporting biases and environmental influences on gene expression.

Some of these problems are addressed by molecular approaches such as association studies, candidate gene studies, and linkage analysis (76). This discussion will focus on the search for candidate genes, which entails educated guesses about known genes that may be related to the study of aggression.

There have, for example, been 2 reports finding an association between novelty seeking (characterized by qualities such as being impulsive, quick tempered, and seeking stimulation) and the dopamine receptor gene *DRD4* (77,78). Unfortunately, this association was not replicated in another study (79).

A later study looked at catechol-O-methyltransferase (COMT) (80). This enzyme is involved in the breakdown of the neurotransmitters norepinephrine, epinephrine, and dopamine. Strous found that having 2 copies (homozygosity) of the low-activity allele of the COMT gene was associated with medium-to-high risk of dangerous behaviour in subjects with schizophrenia. Monoamine oxidase (MAO) activity is also genetically controlled by a gene on the X chromosome. Low MAO activity has been associated with violent offenders (81) and, in a large family, with several male subjects displaying mental retardation and aggression (82).

Finally, the gene for tryptophan hydroxlyase (the rate-limiting enzyme in serotonin production) has also been studied. Manuck and others found that people who had at least 1 copy of the *TPH*U* allele had higher anger and aggression scores (83). This genotype has also been associated with impulsive aggression in male patients with personality disorders (84) and in violent suicide attempters (85).

There are some tantalizing early findings in this field; however, more work is needed. We are progressing on genetic markers, as well as on specific phenotypes: in the future, it is likely that specific genes will be linked with, and provide information about, risk factors for aggressive and violent behaviours. Early identification can then lead to more successful prevention.

Clinical Implications

- Conduct disorder will continue to occupy much energy and interest from the public and the profession.
- Knowledge of known risk and protective factors can guide assessment and treatment recommendations.
- Emerging research, particularly neurobiological research, will greatly influence our future thinking and practice regarding antisocial behaviour in children and youths.

Limitations

- Studies cited have differed in methodologies. Thus, caution is indicated when extrapolating directly to clinical populations.
- In practice, youths and families usually exhibit various combinations of risk and protective factors and must be conceptualized in more complex terms than actuarial analysis of unidimensional factors.
- Biological risk factors were not addressed. These include structural or functional neuroimaging findings (for example, prefrontal cortical deficits), neurophysiological variables (such as neurotransmitter roles), and psychophysiological variables (such as low heart rate and underarousal).

Summary

Evidence from clinical and criminological sources indicates an increasing trend of aggressive and violent behaviours among youths within an overall pattern of declining offending. Most of the children and youths who commit antisocial acts come into our offices daily. Knowledge of risk and protective factors helps assessment, recommendations, and treatment. Incarcerated youths and those with CD who are treated in mental health settings are quite similar (86). They suffer from neurological, psychiatric, and cognitive difficulties; they also have similar ages of behavioral problem onset and similar family backgrounds. Analysis of risk and protective factors can guide early prevention initiatives and interventions. Caution regarding overinterpretation of the literature is warranted, however: statistical correlation in studies does not equal causation. Many life experiences also intervene to mediate between immutable risk factors (for example, male sex) and risk. Lastly, our understanding is still evolving in this area, and clinicians may expect further developments from the biological, psychological, sociological, criminological, anthropological, and philosophical disciplines. In particular, studies from genetics, neuroimaging, and other biological sciences, when coupled with psychosocial research, will add immensely to the field. All of these will be necessary as our society debates prevention, treatment, and punishment for children, youths, and adults with antisocial behaviours.

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Résumé— Trouble des conduites : une étude biopsychosociale

Objectif : *Examiner les travaux publiés sur l'épidémiologie, les facteurs de risque, les facteurs de défense, les typologies et les aspects génétiques des troubles de conduite (TC).*

Méthode : *Les résultats des articles de revues examinés et des textes courants du domaine sont brièvement résumés.*

Résultats : *Les TC se rencontrent souvent dans la pratique clinique. Les facteurs fortement prédicteurs de délinquance future incluent les infractions passées, les camarades antisociaux, des liens sociaux médiocres, la consommation précoce de substances, le sexe masculin et des parents antisociaux. Parmi les facteurs qui prédisent modérément la récidive, on trouve les agressions précoces, le faible statut socio-économique (SSE), les variables psychologiques comme la prise de risques et l'impulsivité, les mauvaises relations parent-enfant, un faible rendement scolaire, une blessure médicale précoce, et les variables neuropsychologiques comme un piètre quotient intellectuel verbal. Les variables peu prédictives comprennent d'autres caractéristiques familiales comme une grande famille, le stress familial, la discorde, la famille désunie et la violence des parents, en particulier, la négligence. Les facteurs de défense incluent des facteurs individuels comme les compétences (dans le domaine social et autres), les relations adultes, les valeurs prosociales et proéducatives ainsi que de bons programmes et soutiens sociaux.*

Conclusions : *Nous en savons beaucoup sur les facteurs de risque psychosociaux des TC. Il existe quelques études sur les facteurs de défense et la contribution génétique, mais elles en sont aux premiers stades. Les travaux futurs accroîtront nos connaissances sur les sous-types, les mécanismes de développement et le traitement des TC.*

ORIGINAL RESEARCH

Physical and Sexual Abuse Issues Among Youths With Substance Use Problems

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Objectives: *To evaluate the prevalence of reported physical and sexual abuse among youths with substance use problems, to explore whether youths report relying on substances to cope with the abuse, and to examine whether individual factors related to substance use were associated with the outcome measures of reported physical abuse, sexual abuse, and using substances to cope.*

Method: *We assessed 287 male and female youths (age 14 to 24 years) who presented for help for substance use problems, using a semistructured interview that focused on substance use, history of previous sexual and physical abuse, and coping strategies.*

Results: *One-half of the female youth substance abusers reported having been sexually abused (50.0%), while male youth substance users reported a significantly lower rate (10.4%). Similarly, one-half of the female youths had a history of physical abuse (50.5%), and males again had a lower rate (26.0%). Of those who endorsed a history of abuse, more females (64.7%) than males (37.9%) reported using substances to cope with the trauma. Specific associations between the outcome measures and substance use variables were found for youths in both sexes.*

Conclusion: *These findings underscore the importance of why clinicians should explore abuse issues with substance-using youth of both sexes. Identifying concurrent factors will help provide better intervention strategies. Suggestions for assessing sexual and physical abuse in youths with substance use disorders are provided.*

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Key Words: *youth, addictions, sexual abuse, physical abuse, substance abuse*

Substance use problems (SUP), when combined with other psychiatric disorders, are often complex and difficult to treat. These problem combinations are the rule rather than the exception with substance-using youths. One area that has not been adequately explored is that of youths with SUP who have had previous physical and sexual abuse.

Physical and sexual abuse are major public health issues. Incidence rates reported in the literature vary (1,2). In Canadian high school youths, the prevalence of reported physical abuse is 15% among males and 25% among females. The prevalence of sexual abuse is 5% for males and 25% for females (2).

The sequelae of physical and sexual abuse can have long-lasting negative consequences for the victim. Both abuse types have been associated with a myriad of problems, including anxiety, aggressive behaviour, anger-management problems, interpersonal difficulties, posttraumatic stress disorders, depression, suicidal behaviour, attention-deficit hyperactivity disorder, conduct disorder, and the vulnerability for developing other psychiatric disorders (3–8). Short- and long-term effects of abuse are related to many variables, among which sex is important (9,10).

Although it has been found that individuals who report physical and sexual abuse have an increased incidence of SUP in later life (3,7), there are few reliable data for specific prevalence rates. Important clinical implications of comorbid sequelae of abuse and SUP—such as drug-pattern use, clinical course, prognosis, and treatment response—have not been studied in adolescents and young adults. This project focuses on these issues. Reviewing initial assessment intake data of youths with SUP can provide a greater understanding of the

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issues required to address future research and implement effective treatments.

We hypothesized that

1. The incidence of both sexual and physical abuse would be higher in youths with SUP than in the general population.
2. Youths with SUP and reported histories of physical and sexual abuse would report using substances to cope with its sequelae.
3. Variables typically related to substance use would be differentially associated in individuals who reported physical abuse, sexual abuse, and using substances to cope with the abuse.

Method

Participants

The sample consisted of 287 individuals (204 males and 88 females) aged 14 to 24 years, who sought treatment for SUP in a large metropolitan addiction treatment centre over a 1-year period. The mean age was 19.82 (SD 2.67) years for male patients and 20.15 years for female patients (SD 2.52). In this sample, the most frequently used substances were cannabis, alcohol, and cocaine. There were no demographic differences between those who reported physical or sexual abuse and those who did not. There were no reliable data on ethnicity differences in the initial intake data.

Procedure

After giving informed consent, the participants underwent a 1.5-hour, face-to-face, semistructured interview with a clinician who inquired about their demographics, history of drug use, past and present drug-use consequences, and treatments, including addiction and psychiatric therapies.

Data Analyses

A series of frequency distributions was conducted to identify the prevalence of reported sexual and physical abuse and the use of substances to cope with the sequelae of the trauma.

To compare the youths who endorsed physical and sexual abuse with those who did not, chi-square analyses were performed on demographics, family history, negative drug-use consequences, anger management, previous treatment, and previous psychiatric diagnosis. Chi-square analysis was also conducted to compare those that endorsed using substances to cope with sequelae of trauma with those who did not.

Results

Frequency distributions suggested high rates of physical and sexual abuse in both sexes, almost double those in the general population (Table 1). Although females reported using substances for coping more than did males, both sexes had high frequencies (Table 2).

Chi-square analyses showed a significant association between age and reported sexual abuse in males. Those who endorsed this abuse were 1.4 years older than those who did not report it ($P = 0.025$).

Further chi-square analyses were conducted to determine whether reported physical abuse, sexual abuse, and substance use for coping were associated with the following variables: negative consequences of substance use, family history of substance use, previous psychological treatment, and anger management. The sample data were first analyzed as a whole and then separated by sex (Table 3):

Variables Associated With Reported Sexual Abuse

Male and Female Combined. Maternal and paternal substance use; health, cognitive, and vocational impairment; legal and financial problems; previous psychiatric diagnosis; and previous psychological treatment.

Male. Maternal substance use; cognitive, interpersonal relationship, and vocational impairment (trends: paternal substance use, physical and verbal abuse behaviour, legal problems, and anger-management problems).

Female. Family history of substance use, previous psychiatric diagnosis, and previous psychological treatment (trends: paternal substance use, cognitive impairment, and financial problems).

Variables Associated With Reported Physical Abuse

Male and Female Combined. Maternal and paternal substance use; health, cognitive, and affect impairment; financial problems; anger-management problems.

Male. Family history of substance use; maternal and paternal substance use; health, cognitive, and interpersonal relationship impairments; anger-management problems (trends: physical and verbal abuse behaviour).

Female. Family history of substance use and maternal substance use, financial problems.

Variables Associated With Reported Substance Use for Coping

Male and Female Combined. Anger-management problems.

Male. Anger-management problems.

Female. Family history of substance use, anger-management problems (trends: paternal substance use).

Discussion

The prevalence of reported physical and sexual abuse in this sample is almost doubled for males and over doubled for females when compared with the incidences reported in the general population (1,2). This underscores the importance of assessing for previous trauma in substance-abusing youth,

Table 1. Frequency of reported physical and sexual abuse

	Reported physical abuse		Reported sexual abuse	
Sex	Yes	No	Yes	No
Male				
n = 204	53	151	21	181
%	26	74	10.4	89.6
Female				
n = 82	48	34	41	41
%	58.5	41.5	50	50

Table 2. Frequency of reported use of substances to cope

Sex	Yes	No
Male		
n = 58	22	36
%	37.9	62.1
female		
n = 52	33	18
%	64.7	35.3

because its occurrence can have important clinical implications. As previously noted, numerous concurrent issues can arise from traumatic abuse.

Both sexes reported using substances at high rates to cope with previous physical or sexual abuse. Although using substances for coping and self-medicating psychiatric symptoms is a contentious issue in the addiction field, clients' self-reports on motivations to use substances have implications for treatment planning (that is, addressing their attitudinal factors for better alliance-building for the therapeutic work) (11,12).

The present results suggest that there are different associated variables for substance-abusing youths who report physical and sexual abuse, compared with those who do not. There are also significant sex differences. This indicates that further research may differentiate specific profiles to distinguish youths with substance abuse problems who report physical and sexual abuse from those who do not. Noteworthy findings of our study include the following:

- Among youths who reported sexual abuse, females had significant histories of acquiring previous treatment for psychological or emotional issues, while males did not. Perhaps this may relate to expectancy help-seeking barriers for males (13–15).
- Interestingly, reported sexual abuse in males was significantly associated with maternal, but not paternal, substance use. This finding needs further research to understand the association.
- Reported physical abuse was associated with paternal substance use in males and maternal substance use in females. This may be due to sex-role permissibility to inflict physical punishment on the same sex, but further exploration of this finding is necessary. The results, however, are consistent with the existing literature (16–18).
- Among youths who reported using substances to cope, there were significant associations for anger-management problems across the entire sample and by sex. This suggests that substances may be used to cope with labile moods, such as anger, or conversely, that the substances themselves allow suppressed impulses to manifest. Understanding the motivation would help tailor treatment to these individuals.
- Among youths who reported using substances to cope, there were significant associations for a past suicide attempt across the entire sample and by sex. This underscores the importance of screening for self-harm behaviours as part of the physical- and sexual-abuse assessment. The significant associations of previous abuse and suicidal behaviour complement existing literature (3,7,10).

Several limitations of the present study need acknowledgment. First, this is a correlative study that does not necessarily imply causation. Although youths reported using substances for coping, other factors may also be operating (for example, withdrawal symptoms). Second, the nature of the assessment, which covers many issues and serves as a first contact and initial alliance-building session for youths seeking help with their substance use, limited the depth of inquiry about trauma and other issues (for example, age, frequency, and context of trauma; using to cope with emotions or flashbacks). We did

Table 3. Associated variables

Variables	Reported Sexual Abuse			Reported Physical Abuse			Reported Use of Substances to Cope		
	χ^2	Male	Female	χ^2	Male	Female	χ^2	Male	Female
Age	NS	0.025	NS	NS	NS	NS	NS	NS	NS
Family history substance use	NS	NS	0.001	NS	0.001	0.022	NS	NS	NS
Maternal substance use	0.001	0.050	NS	0.009	0.011	NS	NS	NS	NS
Paternal substance use	0.001	0.070	0.069	0.001	0.001	0.014	NS	NS	0.61
Negative consequence of drug use									
Health impairment	0.022	NS	NS	0.027	0.016	NS	NS	NS	NS
Cognitive impairment	0.001	0.024	0.060	0.014	0.001	NS	NS	NS	0.73
Affect impairment	NS	NS	NS	0.018	NS	NS	NS	NS	NS
Relationship impairment	NS	0.010	NS	NS	NS	NS	NS	NS	NS
Physical and verbal abuse	NS	0.086	NS	NS	0.092	NS	NS	NS	NS
Vocational impairment	0.046	0.005	NS	NS	NS	NS	NS	NS	NS
Legal problems	0.005	0.080	NS	NS	NS	NS	NS	NS	NS
Financial problems	0.001	NS	0.080	0.007	NS	0.015	NS	NS	NS
Previous psychiatric diagnosis	0.010	NS	0.051	NS	NS	NS	NS	NS	NS
Previous therapy for emotional and psychological issues	0.001	NS	0.006	NS	NS	NS	NS	NS	NS
Past suicide attempts	0.001	0.001	0.001	0.001	0.001	0.006	0.001	NS	0.009
Anger-management problem	NS	0.089	NS	0.002	0.002	NS	0.002	0.016	0.014

NS = Not significant .

not report ethnicity because many participants identified themselves differently according to their cultural heritage, race, or both. Despite these limitations, the results suggest high levels of abuse among youths, as well as the tendency for them to use substances to cope with the sequelae of such trauma. Future research should address these factors.

Conclusion

The findings indicate the need for routine and systematic evaluation for past trauma and its sequelae. If youths are influenced by their belief that they use substances to cope with trauma, it can be used as a tool to engage them in therapy.

There are significant differential associations for those who report past physical and sexual abuse and those who do not. This is further differentiated by sex. Further understanding of the associated variables will allow clinicians to tailor treatment to such individuals.

Although more research is needed to further the characterization of physical and sexual abuse in this population, clinicians should always ask about trauma histories, ensuring the proper level of detail for each client.

Due to the number of sequelae—such as suicidal behaviours—that can manifest from past trauma, the treatment plan may be complex.

Future research should include prospective studies that further explore the character of the reported trauma and its

Clinical Implications

- Youths with substance use problems (SUP) should be screened routinely for past trauma and its sequelae.
- Most youths who endorse physical or sexual abuse assert the use of substances as a coping technique for the trauma.
- Due to the number of sequelae, such as suicidal behaviours that can manifest from past trauma, the treatment plan requires a concurrent approach to the substance abuse and previous abuse treatment.

Limitations

- This sample consists of youths seeking help at a tertiary addiction-treatment centre and may not be applicable to other youth populations.
- The results are based on self-report and may be biased in certain areas (for example, not reporting the full extent of negative consequences of substance use).
- The initial assessment data analyzed were problematic in that they did not adequately cover certain areas that would better characterize the population (for example, ethnicity) nor did they cover the factors of physical abuse, sexual abuse, and coping strategies, which might have allowed better correlation to substance-use patterns.

impact on the individual, along with its relation to substance use. This would include specific psychiatric diagnoses that may significantly associate with this population.

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Résumé— La violence physique et sexuelle chez les jeunes ayant des problèmes de consommation

Objectifs : *Évaluer la prévalence de la violence physique et sexuelle déclarée chez les jeunes ayant des problèmes de consommation d'alcool et de drogues, chercher si les jeunes déclarent consommer pour composer avec la violence, et examiner si les facteurs individuels liés à la consommation sont associés aux mesures des résultats de la violence physique ou sexuelle déclarée, et de la consommation d'alcool et de drogues pour y faire face.*

Méthode : *Nous avons évalué 287 jeunes hommes et femmes (de 14 à 24 ans) qui ont demandé de l'aide pour des problèmes de consommation d'alcool et de drogues, à l'aide d'une entrevue semi-structurée qui portait sur la consommation, les antécédents de violence sexuelle et physique, et les stratégies de réaction au stress.*

Résultats : *La moitié des jeunes femmes toxicomanes déclaraient avoir été victimes d'agression sexuelle (50,0 %), tandis que les jeunes hommes toxicomanes déclaraient un taux significativement plus faible (10,4 %). De même, la moitié des jeunes femmes avaient des antécédents de violence physique (50,5 %), et une fois de plus, les jeunes hommes affichaient un taux plus faible (26,0 %). Parmi ceux qui déclaraient des antécédents de violence, plus de femmes (64,7 %) que d'hommes (37,9 %) déclaraient avoir recours aux substances pour faire face au traumatisme. Des associations spécifiques entre les mesures des résultats et les variables de consommation ont été constatées chez les jeunes des deux sexes.*

Conclusion : *Ces résultats soulignent l'importance pour les cliniciens de sonder les cas de violence chez les jeunes toxicomanes des deux sexes. L'identification de facteurs concurrents contribuera à fournir de meilleures stratégies d'intervention. Des suggestions pour évaluer la violence physique et sexuelle chez les jeunes toxicomanes sont offertes.*

ORIGINAL RESEARCH

Supportive-Expressive Group Psychotherapy for Persons With Inflammatory Bowel Disease

Robert G Maunder MD, FRCPC[1], Mary Jane Esplen RN, PhD[2]

Objective: *Supportive-expressive (SE) group psychotherapy is designed to be applicable to medically ill populations. In this open trial, SE therapy was adapted for use in treating inflammatory bowel disease (IBD).*

Method: *Thirty subjects with Crohn's disease (CD) or ulcerative colitis (UC) were enrolled in 4 psychotherapy groups. Each group met weekly for 20 weeks to discuss emotional and interpersonal issues associated with illness. Physical and psychological variables were measured at the onset and at the finish of the group sessions.*

Results: *There was no mean group change in quality of life (QL), anxiety, or depression over the course of treatment, although there was a mean group reduction in maladaptive coping.*

Conclusion: *Although the trial was uncontrolled and the sample size small, which limits interpretation, the results are consistent with an ineffective intervention. Because a null result would be consistent with previously reported psychotherapeutic trials in cases of IBD, and because SE therapy has been effective in treating other medically ill populations, we discuss characteristics of IBD that may account for a relative resistance to psychotherapeutic support.*

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Key Words: *supportive-expressive therapy, group psychotherapy, inflammatory bowel disease, Crohn's disease, ulcerative colitis, quality of life*

Inflammatory bowel disease (IBD) is the common name given to ulcerative colitis (UC) and Crohn's disease (CD), 2 recurrent idiopathic inflammatory conditions of the gut that in their most severe forms can be chronically debilitating. Despite effective treatment, IBD has significant personal costs due to the effects of systemic symptoms, surgery, and medication. The most frequent concerns of people with IBD are lack of energy, medication effects, and the uncertainty of the disease course (1). People with IBD who seek psychological counselling have relatively greater concerns in regard to physical suffering and financial difficulties. They also have a range of interpersonal concerns that include being a burden and feeling out of control, alone, and dirty. Further, they are concerned that they will be treated as different, and they are concerned about sexual performance (2).

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Psychosocial interventions have been used previously in the treatment of IBD (3), often focused on stress reduction (4) and relaxation (5). The concerns reported by people with IBD who seek counselling suggest that more intensive psychotherapy may be beneficial. Two controlled trials using cognitive-behavioural or interpersonally focused psychotherapy for IBD have, however, found psychotherapy to be ineffective (6,7).

Supportive-expressive (SE) group psychotherapy, developed for women with metastatic breast cancer (8) and subsequently adapted to other groups (9,10), focuses explicitly on concerns and feelings related to illness (11). The existential origins of SE therapy appear to provide a good match for the IBD population whose core concerns include ongoing suffering, uncertainty, and lost potential (3), but SE therapy has not previously been adapted for use in treating IBD.

Supportive-Expressive Group Psychotherapy for IBD

In this adaptation of SE therapy, a group of 6 to 10 people with either UC or CD, met weekly for 20 weeks. SE therapy involves group-therapy techniques that encourage participants to express their thoughts and feelings about illness and its impact on their life and relationships in a supportive environment. SE therapy is expected to provide the benefits

associated with many forms of group therapy, such as imparting information, instilling hope, and experiencing the universality of individual concerns (12). This latter benefit is expected to be of particular value to people with IBD who experience their symptoms as embarrassing or stigmatizing, and themselves as "different." The group is intended to provide a supportive context for discussion of interpersonal difficulties that may interfere with members' obtaining support in important relationships outside the group, including relationships with family, friends, and health care providers. SE therapy also includes introductory training in relaxation techniques (13).

The purposes of this open trial were 1) to adapt SE therapy to patients with IBD, 2) to determine the illness-related concerns that need to be addressed in patients with IBD who seek SE therapy, 3) to investigate the potential of SE therapy to improve IBD-related quality of life and enhance coping with IBD, and 4) to assess qualitatively the feasibility and utility of a larger-scale randomized controlled trial.

Methods

The Human Subjects Review Committee of the University of Toronto approved this prospective, uncontrolled open trial.

Subjects

Subjects were self-referred, having obtained information about the study from gastroenterologists or psychiatrists at Mount Sinai Hospital or through public awareness information published by the Crohn's and Colitis Foundation of Canada or other community agencies. Inclusion criteria were as follows: a confirmed diagnosis of UC or CD; age over 18 years; informed consent; availability and commitment for 20 weeks; proficiency in English; and self-identified concerns about IBD, its treatment, or its complications. Exclusion criteria were untreated severe psychiatric illness or personality disorder, colectomy (in UC), or concurrent treatment in another psychotherapy group.

Therapy sessions

Four groups were conducted, each facilitated by 2 experienced group leaders. Leaders were familiar with the treatment manual for SE therapy in cases of recurrent breast cancer (13) and with the psychosocial treatment of gastrointestinal disorders. Group sessions were 90 minutes long and occurred weekly for 20 weeks.

In determining focus topics for discussion, leaders balanced 2 imperatives. On one hand, to elicit issues spontaneously, they used unstructured enquiry and encouraged participants to discuss emotion-evoking issues. On the other hand, they used directed efforts to facilitate discussion, especially for topics that were expected to be important but that might be withheld without direct enquiry—such as issues related to self-image, stigma, and conflict with health care professionals. Clinical judgement was required to balance these sometimes conflicting methods of enquiry. Co-leaders met before and after each session to review individual and group themes and therapy decisions. Supervision with an experienced colleague was used intermittently.

Measurement Instruments

We characterized participants based on self-reported demographic information, disease history, and current prescription medication. IBD-related concerns were quantified by the Rating Form of IBD Patient Concerns (RFIPC) (14).

Self-report measures were obtained at the start and end of therapy. The main outcome variable was IBD-related quality of life (QL), measured by the Inflammatory Bowel Disease Questionnaire (IBDQ) (15). A gastroenterologist gave the participants a physical examination and assessed disease activity at both the start and finish of SE therapy. An insufficient number, however, attended the physical exam at both the start and end of treatment to analyze disease activity data. As an alternative, disease activity was estimated by the summed score of 3 symptoms (abdominal pain, frequency of diarrhea, and general well-being), each self-reported on a 4-point Likert scale. This method has previously shown a high correlation with the Crohn's Disease Activity Index (CDAI) in a mixed IBD sample (16).

Current symptoms of depression and anxiety were measured by the Hospital Anxiety and Depression Scale (HAD) (17). Coping behaviour was measured with the Ways of Coping inventory (WOC) (18). Maladaptive coping was calculated as the summed total of 3 traits (self-control, accepting responsibility, and escape-avoidance) expressed as a fraction of total coping behaviour (the sum of the 8 subscales of the WOC). Maladaptive coping was measured because previous work indicated a correlation between it and poor QL outcome after treatment for IBD (19) and because improving coping style was a therapy target.

Data Analysis

Description of most prominent IBD concerns was done by rank ordering the concern items for which mean values were greater than the overall mean RFIPC item score.

In subjects who completed therapy, changes in QL and other outcome variables from start to end of therapy were tested using *t*-tests for paired samples. All group means are reported as mean, standard deviation (mean, SD).

Results

Participant Characteristics

Thirty subjects were enrolled in the pilot study. Twenty-four completed the trial, giving a dropout rate of 20%. Of the 30 participants, 24 (80%) were women, 25 (83.3%) were white, and 21 (70%) had CD. The age range of participants was 20 to 52 years (mean 34.9, SD 7.8). Disease duration ranged from

Table 1. Most intense inflammatory bowel disease (IBD) concerns[a] in 30 group-therapy participants

	Mean	Standard Deviation
Illness uncertainty	77.0	23.3
Loss of potential	77.0	25.5
Medication effects	75.8	27.7
Lack of energy	75.4	23.6
Financial concerns	68.1	31.8
Availability of quality medical care	67.7	31.7
Pain and suffering	66.2	28.4
Being a burden	66.0	33.8
Loss of bowel control	63.3	34.9
Having surgery	62.9	32.7
Feelings about body	62.5	31.0
Feeling out of control	61.3	28.0

Table 2. Therapy completers' change in psychological and physical dimensions over the course of 20-week therapy (*n* = 19)

Dimension	Start of therapy	End of therapy	Change	*P*[a]	95%CI
	Mean (SD)	Mean (SD)	Mean (SD)		
IBD Symptoms	11.0 (6.5)	9.2 (4.7)	–1.8 (4.9)	0.14	–4.2 to 0.6
Quality of life (IBDQ)	145.5 (39.0)	151.3 (37.3)	+5.6 (26.0)	0.35	–6.8 to 18.3
Depression (HAD)	5.3 (3.6)	6.6 (5.0)	+1.4 (4.3)	0.18	–0.7 to 3.4
Anxiety (HAD)	9.0 (4.6)	9.0 (4.6)	0.0 (5.0)	0.97	–2.3 to 2.4
Maladaptive Coping (% of total WOC)	48.2 (24.3)	35.5 (24.9)	–12.7 (26.0)	0.05	–25.2 to –0.2

HAD = Hospital Anxiety and Depression Scale; IBDQ = Inflammatory Bowel Disease Questionnaire; WOC = Ways of Coping Inventory.
[a]Significance of *t*-test for paired samples.

28 years to less than 1 year (mean 8.2 years, SD 6.9). Thirteen subjects with CD had a history of surgery. Thirteen participants (43.3%) were taking prednisone (dosage range 5 to 75 mg daily, mean dosage 21.2 mg.).

The mean HAD depression score was 6.3 (SD 3.5). Ten subjects (33%) were above the clinical cut-off (HAD depression score ≥ 8). The mean HAD anxiety score was 10.6 (SD 4.9). Twenty-one subjects (70%) were above the clinical cut-off (HAD anxiety score ≥ 8). Seven participants (23.3%) were regularly taking an antidepressant, 4 (13.3%) a benzodiazepine.

With respect to IBD-related concerns, the mean RFIPC score for the 30 participants was 60.4. Table 1 lists the 11 IBD-related concerns reported at greater intensity than this mean (out of 25 surveyed by the RFIPC).

Of the 24 subjects who completed the trial, 5 did not submit the IBDQ at the end of the sessions. For the remaining 19 subjects, change in QL over therapy ranged from a decrease of 60 IBDQ points to an improvement of 43 IBDQ points. Ten subjects reported improved QL from the start to the end of the therapy, 5 were unchanged, and 4 were worse. With respect to the group as a whole, there was no overall change in IBD-related QL over the course of the trial (Table 2). Nor were there total group differences in depression, anxiety, or IBD symptoms over the course of therapy. Maladaptive coping as a proportion of total coping behaviour was reduced between the start and end of therapy (Table 2).

It is noteworthy that among subjects whose corticosteroid use changed over the course of treatment, all 3 subjects who were on corticosteroids at the start of treatment, but not at the end, reported improved QL over the course of SE for IBD. The only subject who started corticosteroids during treatment had unchanged QL.

Dropouts' Characteristics

All 6 dropouts were women. Four had CD, and 2 had UC. Contact could be made with 4 dropouts: 2 had expected a more structured "classroom" approach, 1 was too ill to attend, and 1 felt that she did not fit in. The difference in anxiety at treatment onset between dropouts (HAD-anxiety 14.3, SD 2.0) and all other subjects (HAD-anxiety 8.8, SD 4.7) was large and statistically significant ($P = 0.001$).

Discussion

In this pilot study, we found that demand for a supportive intervention was strong enough that recruitment was not difficult and did not require active marketing. The group therapy was provided in a busy tertiary care IBD service of a large urban centre (population 2.5 million). The intervention was generally well received by participants, several of whom maintained supportive relationships with one another after the project's termination. The dropout rate of 20% compares favourably with dropout rates of 17% to 57% reported in other psychiatric therapy groups (12).

The top IBD concerns reported were a mix of those common to all IBD sufferers (for example, illness uncertainty, medication effects, and lack of energy [20]) and those which are more common among IBD patients who seek psychological support (that is, feared loss of potential and control, financial concerns, fear of being a burden, and pain and suffering [2]), suggesting that participants were representative of the group of IBD patients who seek some form of psychological intervention.

The predicted changes in coping behaviour occurred over the course of the therapy. There was a reduction in the frequency with which escape-avoidance (denial), self-control (suppression), and assuming responsibility (self-blame) were used. This is in keeping with the goals of an expressive therapy and, as previous work suggests, would be associated with improved QL (19).

With regard to QL outcome, the small sample size and open design limits our conclusions. The main outcome measure, QL, improved in 10 of 19 subjects, and declined in only 4, but there was no change in mean QL in the group as a whole. The lack of a control group prevents us from drawing conclusions about the reasons for changed QL in 14 of 19 subjects. Given the costs involved in the randomized controlled trial that would be required to answer this question, however, some cautious interpretation is warranted. The null hypothesis is that observed changes are unrelated to treatment—in particular, that subjects who enter with lower QL improve, and those who enter with higher QL decline, in an overall regression to the mean.

The study was not designed to test the null hypothesis, but a study design that could test it may be deemed unpromising, based on these results. If this is the case, it is important to attempt to explain these negative results—both because people with IBD want effective supportive interventions and because the SE model has been effective in other populations. Four general explanations are possible. The first is that the lack of apparent efficacy was due to deficits in group-leader competence and adherence to the SE model, although we do not believe this to be the case. The second is that the lack of efficacy is due to changes in the SE therapy format in this adaptation. These changes include the introduction of mixed sex groups and the change to a time-limited format. Mixing sexes clearly changes dynamics within the groups and may inhibit discussion of some sexual and interpersonal themes. It is not the group leaders' impression, however, that the groups were greatly inhibited in this regard. Time-limited groups are not part of the original SE model but have been effectively used in subsequent adaptations (9,10,21).

Third, the intervention may have been less effective because the groups were heterogeneous for such disease factors as time since diagnosis, UC or CD diagnosis, and disease severity. This may have resulted in group members being at very different stages of personal challenge and coping, which would have reduced group cohesion.

The fourth potential explanation for the lack of efficacy, and one which may be important to others adapting psychotherapeutic interventions to IBD, is that persons with IBD and the original target population (persons with breast cancer) differ in key aspects. One difference is that IBD, although associated with QL challenges, functional limitation, and loss of potential, is not generally associated with impending mortality. Another key difference relates to the social and interpersonal stigma that accompanies bowel diseases. It may be that the atmosphere of mutual understanding found within a treatment group is valued but that this intragroup understanding does not adequately translate into improvements in the conflicts and alienation experienced at home or at work.

Clinical Implications

- Interpersonally focused psychotherapy can be adapted for people with inflammatory bowel disease (IBD).
- The clinical demand for this intervention is sufficient to allow relatively easy recruitment of subjects in a large urban centre.
- Supportive-expressive (SE) group psychotherapy for IBD is not associated with a significant improvement in disease-related quality of life.

Limitations

- The study design was open and uncontrolled.
- The sample size was small.
- The participants were heterogeneous for disease type and length of illness.

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Résumé— La thérapie de soutien par l'expression en groupe pour la maladie intestinale inflammatoire

Objectif : *La thérapie de soutien par l'expression (SE) en groupe peut s'appliquer à des populations souffrant de maladies somatiques. Dans cet essai ouvert, la thérapie de SE a été adaptée au traitement de la maladie intestinale inflammatoire (MII).*

Méthode : *Trente sujets souffrant de la maladie de Crohn (MC) ou de colite ulcéreuse (CU) se sont inscrits dans 4 groupes de psychothérapie. Chaque groupe s'est réuni chaque semaine pendant 20 semaines pour discuter des questions émotionnelles et interpersonnelles liées à la maladie. Les variables physiques et psychologiques ont été mesurées au début et à la fin des séances de groupe.*

Résultats : *Il n'y avait pas de moyenne dans le groupe concernant le changement de qualité de vie (QV), d'anxiété ou de dépression en cours de traitement, mais il y avait une réduction moyenne des mécanismes d'adaptation mésadaptés.*

Conclusion : *Même s'il s'agit d'un essai non contrôlé et d'un petit échantillon, ce qui limite l'interprétation, les résultats sont conformes à une intervention inefficace. Étant donné qu'un résultat nul serait conforme aux essais psychothérapeutiques déjà rapportés dans des cas de MII, et que la thérapie de SE a traité efficacement d'autres populations souffrant de maladies somatiques, nous présentons les caractéristiques de la MII auxquelles on peut attribuer une résistance relative au soutien thérapeutique.*

ORIGINAL RESEARCH

Attachment Disorganization and Dissociative Symptoms in Clinically Treated Adolescents

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Objective: *To examine the association of unresolved and unclassifiable attachment with dissociative symptomotology in a sample of 133 adolescents in psychiatric treatment.*

Method: *The study compared 69 adolescents who were unresolved and unclassifiable with 64 adolescents who were not unresolved and unclassifiable. Attachment organization was assessed using the Adult Attachment Interview (AAI). Dissociative symptomotology was assessed using a scale derived from the Youth Self Report (YSR) behaviour checklist.*

Results: *A continuing unresolved and unclassifiable response to attachment-related trauma was correlated with dissociative symptomotology for both male and female adolescents.*

Conclusions: *Cognitive disorganization may be an important variable mediating between the effects of earlier traumatic caregiving experiences and later dissociative symptoms.*

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Key Words: *trauma, unresolved attachment, dissociative symptoms*

In theorizing the relation of disorganized attachment to the risk of psychopathology, Liotti (1,2) and Main and Morgan (3) have advanced the hypothesis that early attachment disorganization may render the individual vulnerable to the later development of dissociative psychopathology. The line of reasoning is as follows: Based on a phenotypic similarity between some behaviour patterns in disorganized infants (for example, stilling or dazed expressions) and trancelike states, Liotti proposed that frightening or frightened behaviour on the part of the primary caregiver is likely to lead to a failure of the integrative functions of memory and the emergence of multiple and incompatible working models of self and the attachment figure. According to Liotti, when caregiver–child interactions are markedly contradictory (that is, encompass experiences of fear or aggression, as well as comfort), the development of multiple and unintegrated working models is necessarily inevitable. Such incompatible models, in which relatively complete forms of defensive exclusion operate, are liable to interfere with the disorganized child's ability to establish a coherent sense of self, thereby leaving him or her vulnerable to the development of dissociative symptomotology in the face of traumatic circumstances in later life.

Evidence for the linkage hypothesis between early attachment disorganization and later dissociative symptomotology comes from 2 recent studies. In 1 study, infants at ages 12 and 18 months were assessed for attachment using the Strange Situation Procedure (4) and then evaluated for later adjustment through to age 17.5 years (5). An infant history of attachment disorganization was significantly correlated with adolescents' self-report of dissociative episodes, as assessed by a scale representing dissociative symptoms derived from the teacher version of the Child Behavior Checklist (CBCL) (6).

Using additional data from the same longitudinal sample, Ogawa and others assessed dissociative symptomotology in young adults (7). In this study, dissociative symptoms were determined by the Dissociation Experience Scale (DES;8). Compared with young adults classified as disorganized during infancy who had not been exposed to trauma and young adults not previously classified as disorganized, young adults

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classified as disorganized during infancy who had been exposed to trauma scored significantly higher on the DES.

This study determines whether a continuing unresolved disorganized response to attachment-related trauma concurrently relates to dissociative symptoms in adolescents in psychiatric treatment.

Method

Participants

We consecutively recruited 145 adolescents to the study upon their admission to 1 of 5 participating treatment centres in 3 Canadian cities, following such severe family disruptions as unwanted or forced separations, loss of parent by death, and abuse by parent. The sites included inpatient and outpatient programs, longer-term residential settings, and day programs. All participants were adolescents between ages 12 and 19 years. We obtained informed written consent to participate from each adolescent, and for participants under age 18 years, we obtained written consent from their parent or guardian. Exclusion criteria included presence of active psychosis or organic brain or central nervous system disorder. We eliminated 12 of the 145 participants from the study (3 who failed to appear for the attachment interview, 5 who gave incomplete interviews, and 4 whose interviews were not recorded for technical reasons), yielding a 92% participation rate. The complete sample comprised 59 females and 74 males who were predominantly white (86.5%); of the sample, 58.3% were age 15 years or under and 41.7% were older than 15 years. At the time of the study, 53% of the participants were in residential treatment. There were no significant differences between males and females in age ($\chi^2 = 0.17$, df 1, $P = 0.67$) or residential treatment status ($\chi^2 = 1.05$, df 1, $P = 0.31$).

Instruments

All participants completed a brief demographic questionnaire, the Youth Self-Report (YSR) and the Adult Attachment Interview (AAI; 9) with minor modifications for adolescent participants.

The AAI is a semistructured interview that focuses on attachment relationships and events in early childhood. The coding of the AAI is a complex task, requiring several steps based on verbatim transcripts of the audiotaped interviews. Each transcript is rated on fourteen 9-point scales. Each subject is then coded for presence or absence of unresolved and disorganized loss or trauma (U_d rating), and for 1 of 3 primary organized attachment patterns: autonomous-secure (F rating), dismissing (D_s rating), or preoccupied-enmeshed (E rating) (10). These classifications parallel the fourfold infant–caregiver strange situation classifications of insecure-disorganized, secure, insecure-avoidant, and insecure-ambivalent, respectively. Transcripts that do not appear to fit one of the primary F, D_s, or E categories, or show a striking mixture of organizations are coded "cannot classify" (CC). Three trained coders, who did not conduct the interviews, coded the transcripts independently. The coders were blind to all other information about the participants until all transcripts had been coded. One coder scored all transcripts; the other 2 coders scored overlapping subsets of the transcripts. All transcripts were coded by at least 2 coders.

Several studies have established the validity and reliability of the AAI. High concordances between parental AAI and infant strange situation classifications have been reported retrospectively (11,12), as well as prospectively (13,14). Test–retest reliabilities of 78% (kappa 0.63), and 90% (kappa 0.79) have been reported by Bukermans-Kranenburg and van IJzendoorn (15) and Benoit and Parker (13), respectively.

In an analysis of interrater reliability for the AAI in this study, the mean concordance rate across all AAI classifications (U_d, F, D_s, E, and CC) for all pairs of raters was 78.6%. The mean kappa for primary classifications (F, D_s, E, CC) is 0.71.

The Youth Self-Report (YSR) is part of a family of instruments, including the CBCL and the Teacher Rating Form (TRF), developed and refined by Thomas Achenbach to assess competencies and problems of individuals 11 to 18 years (6). Seventeen items comprise 3 competence scales: activities, social, and total competence. Principal components analysis of 103 items was used to delineate 8 problem scales: somatic complaints, anxious or depressed, social problems, thought problems, attention problems, self-destructive and identity problems (males only), delinquent behaviour, and aggressive behaviour. Somatic complaints and anxious or depressed scales are combined to yield the superordinate internalizing scale; delinquent behaviour plus aggressive behaviour scales yield the superordinate externalizing scale. The YSR's validity and reliability have been tested in large samples ($n > 1000$) of both community-treated youths and youths referred for psychological and psychiatric treatments. All scales of the YSR have demonstrated strong content validity, criterion-related validity, and discriminant validity, as well as high internal reliability and test–retest reliability. Only the activities scale ($\alpha = 0.38$) and the total competence scale ($\alpha = 0.46$) had Cronbach coefficients less than 0.55; Pearson *r* coefficients of 1-week test–retest reliability ranged from 0.47 (thought problems) to 0.81 (anxious or depressed and externalizing).

We constructed a scale to assess the presence of dissociative symptomotology from the YSR similar to that derived by Carlson (5) from the TRF. Based on DSM-IV criteria and moderate-to-high item total score correlations, Carlson constructed a 5-item dissociative scale for the high school assessment period in her study. Of the 5 TRF items, 3 had corresponding items on the YSR (confused or seems to be in a fog, strange behaviour, and deliberately harms self or attempts suicide). For the 2 remaining items (daydreams and screams a lot), selection was based on thematic similarity

Table 1. Linear regression model predicting dissociative scores

Variable	Estimate	Standard Error	Significance
Constant	2.29	0.32	< 0.001
Unresolved classification	0.97	0.40	0.015
Sex (female)	1.34	0.40	0.001

Table 2. Means and 95%CIs for the dissociative scale by unresolved and unclassifiable classification stratified by sex

	Unresolved Classification	Nonunresolved classification
Female subjects		
n	32	25
Predicted value	4.60	3.63
95%CI	4.21 to 4.99	3.24 to 4.02
Male subjects		
n	34	39
Predicted value	3.26	2.29
95%CI	2.87 to 3.65	1.90 to 2.68

with the TRF items (stares blankly, explosive and unpredictable behaviour) and high item total score correlations. Each item is rated on a 3-point scale from 0 (not true) to 2 (often true). Summation scores for the YSR-derived dissociative scale are produced (scores ranging from 0 to 10), with higher scores indicating more problems. Cronbach's alpha was 0.64, indicating acceptable structural coherence of the scale, comparable with Carlson's TRF-derived dissociative scale (0.63).

Results

We assigned participants to 1 of 2 groups: unresolved and unclassifiable ($n = 69$) and not unresolved and unclassifiable ($n = 64$), based on their responses to the AAI. The creation of the U_d/CC category warrants comment. As discussed above, individuals are placed in the CC category when D_s, E, or F categories cannot be assigned. Because disorganized and disoriented attachment status has been clearly linked to CC attachment status, and because CC status is rare, most investigations include CC individuals within the U_d attachment category.

We used multiple linear regression analysis to examine the effect of unresolved and unclassifiable classification and sex on dissociative symptomotology. The dependent variable in the model was the dissociative score, and the independent variables were unresolved and unclassifiable classification and sex, each coded as an indicator variable. The significant difference in residential status between the 2 groups warranted its inclusion in the model to assess its possible confounding effect. All possible 2-way interactions among these 3 variables were examined.

There was a significant effect of both unresolved and unclassifiable classification ($P < 0.015$) and sex ($P < 0.001$) on dissociative scores in the multiple regression analysis. There was no evidence that residential status had a confounding effect or of any significant 2-way interactions. Table 1 presents the regression model. The results indicate that for adolescents in psychiatric treatment, an unresolved and disorganized response to attachment-related trauma is associated with higher dissociative scores in both sexes.

Independent of this, females generally report more dissociative symptoms than do males, as evidenced by their higher mean scores on the dissociative scale, regardless of unresolved status. Table 2 illustrates the significant sex effect on dissociative scores, the predicted values (with 95%CIs) from the regression equation, including both unresolved and unclassifiable classification and sex.

Discussion

This study examined the association between unresolved and disorganized attachment status (as assessed by the AAI) and dissociative symptoms (based on a 5-item dissociative scale) within a clinical sample of adolescents. We found that an unresolved and unclassifiable response to attachment-related trauma was related to dissociative symptoms. Specifically, unresolved adolescents had significantly elevated scores on the dissociation scale, even after controlling for residential treatment status. This finding, together with the study results of Carlson (5) and Ogawa and others (7), provides empirical support for Liotti's (1,2) argument that the disorganization attachment construct offers a specific, theoretically grounded way to understand dissociative psychopathology.

Whereas the unresolved and unclassifiable combination results in higher dissociative-symptom scores in both sexes, females have higher scores independent of the presence of unresolved and unclassifiable attachment. Among the 133 adolescents, females were significantly more likely than males to be preoccupied (10% vs 3%; likelihood ratio = 3.763). Individuals classified as preoccupied on the AAI appear confused or entangled and enmeshed in past relationships. We may also note that 1 subcategory (E_3) of the preoccupied group is preoccupied with frightening, often traumatic, attachment-related experiences. The finding that both unresolved and preoccupied attachment in females was related to dissociative symptoms suggests an underlying link at the level of mental processes between preoccupied and unresolved mental states, particularly in the case of the E_3 or traumatic preoccupation subgroup. It is important to note that the preoccupied classification is frequently assigned as the alternative classification in individuals who have been judged unresolved on the AAI (16).

According to Bowlby, complete defensive exclusion is the action taken in response to conditions of severe threat to attachment (17). Through this process, trauma-related attachment, memories, and feelings are encoded in a separate representational model that is segregated from consciousness. Debarred from consciousness, this model is closed to new information about attachment and is therefore likely to interfere with the individual's ability to comprehend and assess subsequent experience. Liotti proposed that later experiences that are appraised as threatening acquire dissociative potential because these experiences reactivate the previously segregated (dissociated) model of the past in the present, thereby creating a representational identity between these experiences (1,2).

Unlike Carlson's (5) and Ogawa and others' (7) studies, our data do not permit the identification of attachment disorganization as a precursor to dissociative symptoms. Taken together, however, the results from these studies provide first empirical evidence of a link between attachment disorganization and dissociative symptomotology. How can we make sense of this link? In line with Liotti's model outlined above, the finding that lapses in the metacognitive monitoring of reasoning and discourse are related to dissociative symptoms suggests that attachment disorganization may be an important variable mediating between the effects of earlier traumatic caregiving experiences and later dissociative experiences. Main and Goldwyn (10) propose that such lapses in the AAI when discussing traumatic attachment experiences indicate that attention, working memory, and consciousness are being overwhelmed. Following Liotti, unresolved adolescents may be especially prone to disorganized responses in circumstances that they perceive as repetitions of previous traumatic experiences. It is in these circumstances, when the adolescent's attachment system is intensely activated, that acute cognitive disorganization may be unleashed, thereby contributing to the emergence of dissociative symptoms.

Clinical Implications

- This study examined dissociative symptoms from an attachment perspective.
- Dissociative symptoms in clinically treated adolescents are strongly associated with disorganized attachment.
- Current interpersonal crises (for example, loss and rejection) that draw attention to earlier traumatic experiences may trigger disorganized responses.

Limitations

- The cross-sectional design precludes identification of disorganized attachment as a precursor to dissociative symptoms.
- A self-report instrument measured dissociative symptoms.
- The findings cannot be generalized to nonclinically treated adolescents.

We need to make an important caveat. We used a self-report assessment as the index of dissociative symptoms. As a result, we do not know whether the presence of dissociative symptoms would meet the criterion of psychiatric caseness for dissociative disorder. For this reason, future studies that examine the link between attachment disorganization and dissociation should use a DSM-IV–based structured interview for the categorical determination of the presence or absence of dissociative disorder.

Despite this limitation in assessment, the findings underscore the need to consider not only the dimensions of security and patterns of insecurity, but also the dimension of disorganization of the attachment system. Research in the last decade now indicates that it is disorganized attachment, rather than the organized insecure groups, that is most closely linked to psychopathological outcomes (5,18).

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Résumé— Désorganisation de l'attachement et symptômes dissociatifs chez les adolescents en clinique

Objectif : *Examiner l'association entre l'attachement non résolu et inclassifiable, et la symptomatologie dissociative auprès d'un échantillon de 133 adolescents en traitement psychiatrique.*

Méthode : *L'étude a comparé 69 adolescents qui étaient non résolus et inclassifiables avec 64 adolescents qui ne l'étaient pas. L'organisation de l'attachement a été évaluée à l'aide de l'entrevue sur l'attachement adulte (AAI). La symptomatologie dissociative a été évaluée par une échelle tirée de la liste de vérification du comportement de l'auto-évaluation des jeunes (YSR).*

Résultats : *Une réponse non résolue et inclassifiable constante au traumatisme lié à l'attachement a été corrélée avec la symptomatologie dissociative chez les adolescents, filles et garçons.*

Conclusions : *La désorganisation cognitive peut être une variable importante intermédiaire entre les effets d'expériences antérieures de soins traumatiques et de symptômes dissociatifs ultérieurs.*

REVIEW PAPER

Alcohol Consumption and Major Depression: Findings From a Follow-Up Study

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Objective: *To investigate whether alcohol consumption predicts major depressive disorder episodes (MDEs) in the general population.*

Method: *The respondents without depression (*n = *12 290) in the longitudinal cohort of the Canadian National Population Health Survey (NPHS) were classified into cohorts based on any drinking, frequency of drinking, maximum number of drinks on a maximal drinking occasion, and average daily alcohol consumption, based on data collected in the 1994–1995 survey. Major depression frequency 2 years later, in 1996–1997, was evaluated and compared across drinking categories.*

Results: *The respondents who reported any drinking, drinking daily, having more than 5 drinks on a maximal drinking occasion, and having more than 1 drink daily on average, did not have an elevated risk of major depression. A trend in the data suggested that women who reported having more than 5 drinks on a maximal drinking occasion might be at a higher risk of major depression. No evidence of confounding or effect modification by demographic, psychological, and clinical variables was found.*

Conclusion: *In a general population sample, alcohol consumption levels were not associated with major depression. Having more than 5 drinks on a maximal drinking occasion, however, may be associated with an increased risk of major depression among women. Extreme patterns of alcohol consumption, which tend to characterize clinical samples, are associated with depression. These patterns of drinking, however, are relatively uncommon in the general population, and the current analysis may have lacked power to detect these associations.*

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Key Words: *alcohol consumption, major depression*

Cross-sectional studies using community samples have provided evidence that heavy alcohol use is associated with an elevated risk of depressive symptoms (1,2) and major depression (3–5). It remains unclear whether alcohol consumption is a causal factor for major depression. Relevant information has significant public health and clinical implications in terms of formulating prevention strategies and managing depressive disorders. Yet, few prospective studies using community-based samples have evaluated the relation between alcohol consumption and depression.

The existing follow-up studies that use community samples failed to find an association between alcohol consumption and depressive symptoms (6,7). Moscato and others followed 1358 household subjects from 1986 to 1993 (6). They examined whether the initial alcohol problems predict depressive symptoms. The subjects were considered to have alcohol problems if they met DSM-III criteria for alcohol abuse or dependence in the previous year or reported having 5 drinks or more daily, at least 1 to 2 times weekly in the previous year. Depressive symptoms were evaluated using the Center for Epidemiological Studies–Depression Scale (CES-D). Alcohol problems were not found to predict depressive symptoms. In another study, Lipton classified subjects into groups of abstainers, light drinkers, light-moderate drinkers, moderate drinkers, and heavy drinkers (7). One year later, the subjects were evaluated using the CES-D, and it was found that the mean CES-D scores did not differ significantly between light or no drinking and heavy drinking groups. Similarly, a recent metaanalysis study (8) failed to find evidence that initial alcohol consumption levels per occasion predicted later depressive symptoms (ranged from 2 to 10 years).

As noted above, most existing studies have measured depressive symptoms rather than depressive disorders. Depressive symptoms do not necessarily indicate clinically significant depressive disorders, such as major depression. Schuckit and

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others conducted a 12-month follow-up study to measure the incidence of major depression by DSM-III-R criteria among 239 alcoholic men in an inpatient setting (9). In their study, they failed to find a significant difference between the incidence of major depression during heavy drinking and the incidence of major depression independent of heavy drinking. Similarly, Hodgins and others found no evidence that heavy drinking could reliably predict later occurrence of major depression, using a clinical sample (10). Data from clinical populations, however, may not apply in the general population and may not be a suitable basis for forming public health policy.

The Canadian National Population Health Survey (NPHS) provides a unique opportunity to examine the relation between alcohol consumption and major depression in a prospective perspective. The NPHS target population included household residents in all provinces, with the exclusion of residents of Indian reserves, Canadian Forces bases, and some remote areas (11). The NPHS contains a longitudinal cohort which was reinterviewed in the 1996–1997 survey, 2 years after the initial interview. Major depression was evaluated using the Composite International Diagnostic Interview–Short Form (CIDI-SF) in the NPHS, which was derived from the World Health Organization's CIDI (12). The CIDI-SF was developed and validated at the University of Michigan (13,14) as a major depression predictor for use in survey research. Major depression, as defined in the NPHS, represents a purported 90% predictive cut-point for the CIDI-SF. This cut-point corresponds to reporting 5 of 9 DSM-IV diagnostic criteria for major depression—the number of symptoms required to make the diagnosis—at least 1 of which must be depressed mood or loss of interest. Therefore, this choice of a cut-point can be justified by its face validity for the DSM-IV criteria (15). The same cut-point has been chosen in previous studies (5,16,17). The NPHS also collected the information regarding alcohol consumption, such as any alcohol use, maximum amount of drinks on a single occasion, frequency of drinking in the past 12 months prior to the interview, and the average daily alcohol consumption in the week before the interview (11).

This study investigates the relation between alcohol consumption and major depression by comparing the incidence of major depression among the cohorts classified by various alcohol consumption variables.

Method

The members of the NPHS longitudinal cohort who did not have major depression at the baseline (1994–1995 survey) were included in this analysis. The longitudinal cohort included 15 670 respondents from the baseline survey. Of those who were eligible (over the age of 12 years, nonproxy interview) and provided valid information (n = 13 071) at the baseline, 12 290 respondents (94.0%) did not have major depression. These respondents were classified into cohorts based on alcohol consumption status in 1994–1995 and were linked to the follow-up data collected in the 1996–1997 survey. The proportion of these subjects with a major depressive episode (MDE) identified in the follow-up interview was regarded as an incidence proportion in this analysis. Thus, the incidence, as opposed to prevalence, of major depression in various categories of drinkers, was evaluated.

Four separate analyses were conducted. In the first analysis, subjects were classified into 2 cohorts by their drinking status: drinking and no drinking. In the second analysis, subjects were reclassified in terms of frequency of drinking at 2 levels: drinking every day and drinking less frequently than every day. In the third part of the analysis, subjects were classified into 2 groups according to the maximum number of drinks on a single occasion: having more than 5 drinks on at least 1 occasion and having 5 drinks or less on 1 occasion. In the fourth part of the analysis, average daily alcohol consumption was used to categorize the subjects at 2 levels: having more than 1 drink daily on average and having 1 drink or less daily on average.

For each analysis, the incidence of major depression was calculated in each group. Stratified analyses by sex, age, marital status, employment status, income adequacy, long-term medical conditions, antidepressant use, and perceived social support were performed to detect possible effect modification and confounding effects. In this analysis, 95% CIs were used to judge whether the estimates of incidence were different from each other. Although exploring more extreme patterns of alcohol consumption would have been interesting, the sample size did not permit such analyses due to the small numbers in the relevant groups in the general population.

In 3 of the 4 analyses, the nondrinkers were included in the groups of drinking less frequently than daily, having 5 drinks or less on a single occasion, and having 1 drink or less daily on average. Considering that the nondrinkers may have specific characteristics, which may differ from the drinkers, analyses 2, 3, and 4 were repeated with the exclusion of the nondrinkers.

Research personnel from Statistics Canada collected and have maintained the NPHS data. Unlike the publicly available microdata files, the master file data were used for this analysis. The actual data (master files) are not routinely available to the public because they are confidential. Statistics Canada permits the use of "dummy files," which have the same data structure as the master files; however, the data are not meaningful. A remote access procedure was used to perform data analysis, after Statistics Canada approval. The data analysis programs were written on a local computer using the dummy data and were submitted to Statistics Canada by E-mail, as either SPSS syntax files or SAS command files. Statistics Canada ran the programs using the master files. For this

Table 1. Description of the subjects in 1994–1995

	Number (%)
Sex	
Men	5547 (45.1)
Women	6743 (54.9)
Total	12 290 (100)
Age in years	
12–24	2125 (17.3)
25–44	4670 (38.0)
45–64	3165 (25.8)
65+	2330 (19.0)
Total	12 290 (100)
Marital status	
Married or common law or partner	6710 (54.6)
Single	3244 (26.4)
Divorced or separated or widowed	2334 (19.0)
Total	12 288 (100)
Employment	
Currently working	6585 (56.1)
Currently not working	5160 (43.9)
Total	11 745 (100)

study, we used SPSS 9.0 (18) for the preliminary descriptive analysis; we used the SAS (19) micros released by Statistics Canada to calculate bootstrap variance estimates.

The NPHS is based upon a complex design, with stratification and multiple stages of selection and unequal probabilities of selection of respondents. Using data from such complex surveys presents problems, because the survey design and the selection probabilities affect the estimation and variance calculation procedures that should be used. To account for the effect of multistage sample selection, Statistics Canada developed bootstrap weights for the variables in the NPHS database and coefficient of variance tables (CV tables). Statistics Canada suggests that investigators using the NPHS data should apply either the bootstrap weights or CV tables, so that parameter estimates and their standard errors are valid. All incidence rates and corresponding 95%CIs reported in this study were calculated using the bootstrap weights.

To release the results based on the NPHS data, Statistics Canada set up release guidelines. Specifically, Statistics Canada does not recommend the release of any estimates calculated based on fewer than 30 sampled respondents. All the incidence rates reported in this study meet this requirement. For the weighted estimates based on sample sizes of 30 or more, the sampling variability of the estimates is determined by the bootstrap coefficient of variance in this study. The estimates marked in the tables with a superscript "b" indicate that the bootstrap coefficient of variance of the estimates is between 16.6% and 33.3%. This means that high sampling variability is associated with the estimates. Estimates were not reported where the bootstrap coefficient of variance is greater than 33.3%.

Results

There were 12 290 respondents who did not have major depression in 1994–1995. Table 1 presents the demographic characteristics of these subjects. Of these subjects, 475 individuals (3.9%) did not provide valid responses for the major depression predictor in 1996–1997. Respondents with missing data were more likely to be age 65 years and over (62.5%), to be unemployed (79.7%), and to have chronic illnesses (77.1%). Compared with those without missing data, these respondents were also more likely to be divorced, separated, widowed (38.7%), and to have less social support (22.5%). These respondents were not included in the analyses. Additionally, some subjects had valid information about major depression in 1996–1997 but did not provide information regarding alcohol consumption in 1994–1995. Where information was missing for drinking status ($n = 4$), frequency of drinking ($n = 8$), maximum amount of drinking on a single occasion ($n = 103$), and average daily alcohol consumption ($n = 37$), these subjects were not included in the analyses. Ultimately, there were 11 811 subjects for analysis 1, 11 807 subjects for analysis 2, 11 712 subjects for analysis 3, and 11 778 subjects for analysis 4.

Table 2 presents the weighted estimates of incidence in each group of the 4 analyses. As indicated by the 95% CIs, the subjects who reported drinking in the past 12 months did not have an elevated risk of major depression relative to those who reported no drinking. The incidence of major depression in the subjects who reported having more than 5 drinks on a maximal drinking occasion was not significantly different from that in the subjects who reported having 5 drinks or less on a maximal drinking occasion. There was no evidence that the subjects who reported having more than 1 drink daily on average in the past week prior to the 1994–1995 interview were at a higher risk of major depression than those who reported having 1 drink or less in the past week. The incidence in the daily drinking group was too imprecise to report because the bootstrap coefficient of variance associated with this estimate is greater than 33.3%. The incidence of major depression was 3.5% (95% CI, 3.1% to 4.0%) in the group reporting nondaily drinking.

Table 3 presents stratification by sex. Because of the high sampling variability (bootstrap coefficient of variance 33.3%) associated with the incidence among the daily drinkers and those who reported having more than 1 drink daily on average, these specific estimates were not reported. The stratified analyses showed no evidence that the lack of association was due to confounding effect by sex. The

Table 2. Incidence of major depression in each group

	Subjects with depression	Total	Incidence (%)	95% CI
Analysis 1				
Drinking	287	8976	3.3	2.9 to 3.8
Nondrinking	106	2835	4.3	3.3 to 5.4
Analysis 2				
Daily drinking	11	484	Not reported[a]	Not reported[a]
Nondaily drinking	382	11 321	3.5	31 to 4.0
Analysis 3				
> 5 drinks per occasion	112	3235	3.9	2.8 to 4.9
5 drinks or less	276	8477	3.5	3.0 to 3.9
Analysis 4				
> 1 drink daily	30	946	4.3[b]	1.6 to 6.9
1 drink or less daily	362	10 832	3.5	3.1 to 3.9

[a]Not reported because the bootstrap coefficient of variance is greater then 33.3%; [b]The bootstrap coefficient of this estimate is between 16.1 and 33.3%.

Table 3. Results of stratified analyses by sex

	Incidence (95% CI)	
	Men	Women
Analysis 1		
Drinking	2.5 (1.8 to 3.1)	4.2 (3.5 to 4.9)
Nondrinking	3.3 (1.7 to 5.0)	5.0 (3.4 to 6.4)
Analysis 2		
Daily drinking	Not reported[a]	Not reported[a]
Nondaily drinking	2.5 (1.9 to 3.1)	4.5 (3.8 to 5.1)
Analysis 3		
> 5 drinks per occasion	2.9 (1.7 to 4.2)[b]	6.4 (4.4 to 8.3)
5 drinks or less	2.4 (1.6 to 3.2)	4.1 (3.5 to 4.8)
Analysis 4		
> 1 drink daily	Not reported[a]	Not reported[a]
1 drink or less daily	2.4 (1.8 to 3.0)	4.4 (3.8 to 5.1)

[a]Not reported because the bootstrap coefficient of variance is greater then 33.3%; [b]The bootstrap coefficient of this estimate is between 16.1% and 33.3%.

associations were stratified by the remaining demographic, psychological, and clinical variables. There was no evidence of confounding or effect modification by these variables.

Since alcohol abstainers may differ from drinkers in risk of major depression, analysis 2, analysis 3, and analysis 4 were repeated with the exclusion of nondrinkers. Table 4 presents the incidence of major depression in the 3 analyses groups. The findings were consistent with the previous analyses. The incidence of major depression in the group having 5 drinks or less on a maximal drinking occasion was 3.0% (95% CI, 2.6% to 3.5%) after the nondrinkers were excluded. This was not significantly different from the incidence in the group having more than 5 drinks on a maximal drinking occasion.

Table 5 contains the results of stratified analyses by sex with the exclusion of nondrinkers. Because of the high sampling variability (bootstrap coefficient of variance 33.3%), the incidence among the daily drinkers and those who reported having more than 1 drink daily on average was not reported. There was a trend, however, suggesting that sex might be an effect modifier in the relation between the maximum amount of drinking on a single occasion and major depression. The incidence among women who reported having more than 5 drinks on a maximal drinking occasion was 6.4% (95% CI, 4.4% to 8.3%), and the incidence among women who reported having 5 drinks or less on a maximal drinking occasion was 3.7% (95% CI, 3.0% to 4.4%). Among men, the incidence in respondents who reported having more than 5 drinks on at least a single occasion was not significantly different from that in those who reported having 5 drinks or less on a

Table 4. Incidence of major depression in each group with the exclusion of nondrinkers

	Subjects with depression	Total	Incidence (%)	95% CI
Analysis 2				
Daily drinking	11	484	Not reported[a]	Not reported[a]
Nondaily drinking	276	8488	3.3	2.8 to 3.7
Analysis 3				
> 5 drinks per occasion	112	3235	3.9	2.8 to 4.9
5 drinks or less	170	5642	3.0	2.6 to 3.5
Analysis 4				
> 1 drink daily	30	946	4.3[b]	1.6 to 6.9
1 drink or less daily	250	7997	3.2	2.8 to 3.6

[a]Not reported because the bootstrap coefficient of variance is greater then 33.3%; [b]The bootstrap coefficient of this estimate is between 16.1% and 33.3%.

Table 5. Results of stratified analyses by sex with the exclusion of nondrinkers

	Incidence (95% CI)	
	Men	Women
Analysis 2		
Daily drinking	Not reported[a]	Not reported[a]
Nondaily drinking	2.3 (1.7 to 2.8)	4.3 (3.6 t o5.0)
Analysis 3		
> 5 drinks per occasion	2.9 (1.7 to 4.2)[b]	6.4 (4.4 to 8.3)
5 drinks or less	2.0 (1.3 to 2.7)	3.7 (3.0 to 4.4)
Analysis 4		
> 1 drink daily	Not reported[a]	Not reported[a]
1 drink or less daily	2.1 (1.6 to 2.7)	4.2 (3.5 to 4.9)

[a]Not reported because the bootstrap coefficient of variance is greater then 33.3%; [b]The bootstrap coefficient of this estimate is between 16.1% and 33.3%.

single occasion. Additional stratified analysis found no evidence that other variables confounded these associations.

Discussion

This study demonstrates that alcohol consumption at the levels evaluated was generally not associated with major depression in the general population. There was no indication that the null results were due to confounding by demographic, psychological, and clinical variables. These findings are consistent with previous community-based follow-up studies in the literature (6,7). Nevertheless, this analysis extended our knowledge about the etiology of depressive disorders by evaluating the impact of alcohol consumption at various levels on the incidence of major depression rather than prevalence or depressive symptoms.

This study's findings apparently contradict those from cross-sectional studies using community-based (1,2,5) and clinical samples (20–23). These studies indicate a strong association between alcohol consumption and major depression or depressive symptoms. It is unlikely that the nonsignificant associations observed in this analysis were due to the thresholds chosen for the alcohol-consumption indices. These cut-off points were chosen in this analysis because a previous study using these data showed that subjects who reported alcohol consumption within these levels had a homogeneous risk of major depression (24). Subjects who reported extreme drinking patterns might have an elevated risk of major depression. Our analysis, however, could not definitely confirm this because the estimates that were calculated based on subjects with extreme drinking patterns were not precise. But a possible explanation about the nonsignificant association is that alcohol consumption at these exposure levels may not directly cause major depression, although it may affect the prognosis of major depression. More specifically, alcohol consumption may prolong the duration of the MDE. Since prevalence is approximately proportional to the product of incidence and disease duration (25), a poor prognosis for major depression in drinkers could explain the phenomenon of strong association in cross-sectional studies but no association in incidence-based follow-up studies. Another possible explanation could be reverse causation. A past history of major depression could lead to some proportion of the excessive drinking observed in 1994–1995.

Since the exposure variables used in this study were unidimensional measurements of alcohol consumption, it is certain that the cohorts classified by these variables overlapped. For instance, some frequent drinkers might consume a small amount of alcohol each time; whereas, some infrequent drinkers might drink a large amount of alcohol on a single occasion—especially among young people (26). We performed a series stratified analyses to evaluate the combined impacts of frequent drinking, high daily average consumption, and high maximum number of drinks on a single occasion. The lack of association between alcohol consumption and major depression persisted, despite alternative classification of the cohorts.

This analysis suggested, but did not confirm, that sex might be an effect modifier of the relation between maximum amount of drinks on a single occasion and major depression. An effect of this nature is plausible from a biological perspective. Men and women, on average, have different body mass and body water volume (27). Hence, the same volume of alcohol consumption may have a greater biological impact on women. The findings of this analysis suggest that women may be more vulnerable to the depressive impact of alcohol consumption than men. It is also possible that women who reported having more than 5 drinks on a single occasion may have a history of major depression, wherein the episodes were not detected in the 1994–1995 survey because none had occurred in the preceding year.

From a biological point of view, at low levels of alcohol–blood concentration, alcohol may act as a central nervous system stimulant. At higher levels of blood–alcohol concentration, alcohol may act as a central nervous system depressant (28). Increasing consumption levels may increase the chance of depression (29), but we are more likely to encounter such high consumption levels in clinical settings than in the general population. Therefore, the possibility that alcohol consumption is part of a causal mechanism involving major depression in some contexts should not be discounted based on these findings. The results of studies conducted in clinical settings cannot necessarily be generalized to the population as a whole, and the reverse is also true.

There are some weaknesses in this analysis. First, since successful follow up was less than 100%, bias may have resulted from incomplete follow-up data. We compared the 475 subjects who did not have valid information regarding major depression status in 1996–1997 with those without missing data. The 2 groups were not substantially different in terms of alcohol consumption status. This does not exclude the possibility of bias, but it provides some reassurance that the subjects with incomplete data resembled the remaining subjects in terms of alcohol consumption. Second, CIDI-SF is a brief predictor, which may introduce misclassification of major depression relative to the full CIDI. Third, the term "incidence" used in this analysis is not identical to the conventionally used term. The CIDI-SF measures major depression occurring in the past 12 months, and major depression was evaluated every 2 years in the NPHS. Therefore, the major depression status during the year following the 1994–1995 survey was unknown. Further, although this analysis found that alcohol consumption did not predict major depression during this follow-up period, this could not rule out the possibility that alcohol consumption was associated with major depression beyond this 2-year period. Fourth, history of major depression was not controlled in this analysis, due to lack of available information in the NPHS data set.

Clinical Implications

- Various indices of alcohol consumption are not associated with major depression in a 1-year period, 2 years later.
- Women who reported having more than 5 drinks on a maximal drinking occasion displayed a trend toward an elevated risk of major depression.
- The analysis did not suggest that the findings were due to confounding by demographic, psychological, and clinical variables.

Limitations

- The NPHS survey relied on self-report measures of alcohol consumption; data concerning diagnostic comorbidity were not evaluated.
- The NPHS data set did not provide past histories of major depression and alcohol consumption.
- Due to infrequency within the general population, extreme drinking patterns could not be explored.

This analysis was one of the few longitudinal studies investigating the relation between alcohol consumption and major depression, using a large and representative community sample. We did not find a strong association between alcohol consumption levels and major depression in this prospective analysis. Women who reported having more than 5 drinks on a maximal drinking occasion may have an elevated risk of major depression. Reverse temporal effects in the case of the possible association in women could not be entirely excluded, however.

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Résumé— Consommation d'alcool et dépression majeure : résultats d'une étude de suivi

Objectif : *Déterminer si la consommation d'alcool prédit des épisodes de trouble dépressif majeur (TDM) dans la population générale.*

Méthode : *Les répondants sans dépression (*n = *12 290) de la cohorte longitudinale de l'Enquête nationale sur la santé de la population (ENSP) ont été classés en cohortes selon la consommation d'alcool, la fréquence de la consommation, le nombre maximum de verres en une occasion de consommation maximum et la consommation quotidienne moyenne, selon les données recueillies dans l'enquête de 1994-1995. La fréquence de la dépression majeure, ultérieurement en 1996-1997, a été évaluée et comparée entre les catégories de buveurs.*

Résultats : *Les répondants qui ont déclaré consommer de l'alcool, consommer chaque jour, boire plus de 5 verres lors d'une occasion de consommation maximum, et boire plus d'un verre en moyenne chaque jour n'étaient pas à risque élevé de dépression majeure. Une tendance des données indiquait que les femmes qui déclaraient boire plus de 5 verres lors d'une occasion de consommation maximum pouvaient être à risque plus élevé de dépression majeure. Aucune preuve de facteur confusionnel ou de modification de l'effet par des variables démographiques, psychologiques et cliniques n'a été établie.*

Conclusion : *Dans un échantillon de la population générale, les niveaux de consommation d'alcool n'ont pas été associés avec la dépression majeure. Toutefois, boire plus de 5 verres lors d'une occasion de consommation maximum peut être associé à un risque accru de dépression majeure chez les femmes. Les modèles extrêmes de consommation d'alcool, qui tendent à caractériser les échantillons cliniques, sont associés à la dépression. Ces modèles de consommation sont toutefois relativement rares dans la population générale, et la présente analyse manquait peut-être de l'efficacité nécessaire pour détecter ces associations.*

REVUE

Le concept français d'athymhormie de 1922 à nos jours

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Le concept psychiatrique d'athymhormie (perte de l'élan vital et de l'affectivité) a été créé au début du 20e siècle en France, où son usage était resté confiné jusqu'à ce qu'il soit heureusement repris par des neurologues, essentiellement francophones. Pour ses auteurs, M Dide et P Guiraud, il désignait une entité autonome, héritière de la « démence précoce » dont ils n'acceptaient pas le remplacement par une « schizophrénie » aux limites floues. Par la suite, ce terme désignera pour eux et, jusqu'à nos jours pour la majorité des psychiatres français, le noyau fondamental des formes déficitaires de psychoses. Le terme d'athymhormie (ou perte d'auto-activation psychique) est actuellement aussi utilisé en neurologie pour désigner les conséquences comportementales et affectives de lésions acquises des noyaux gris et plus généralement d'un circuit assimilé à la « boucle limbique ». Toutefois, la question se pose de la réalité phénoménologique des troubles correspondants et de l'intérêt d'une même appellation. Au terme de cette revue, il est conclu que même si les tableaux dans lesquels l'athymhormie s'insère en clinique psychiatrique et neurologique sont bien différents, le choix d'un même terme se défend dans le cadre d'une approche neuropsychiatrique renouvelée et symptomatique. L'objectif d'une telle approche est de rechercher en quoi certains symptômes, communs ou similaires, pourraient représenter le déficit d'une fonction physiologique que l'on suppose ici être celle de la motivation.

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Mots clés: *athymhormie, schizophrénie déficitaire, perte d'auto-activation psychique, neuropsychiatrie, motivation*

Le terme français « athymhormie » a été introduit dans la terminologie médicale pour décrire un aspect fondamental des psychoses déficitaires, et il reste toujours utilisé dans ce sens par les psychiatres français. Toutefois, l'usage de ce mot a été repris ces dernières années en clinique neurologique, surtout francophone, pour désigner un trouble majeur du comportement et des affects observé le plus souvent à la suite de lésions sous-corticales minimes.

Cette présentation de l'athymhormie sera surtout historique mais nous amènera à aborder, *in fine*, les deux questions suivantes : 1) les troubles ainsi désignés, en neurologie et psychiatrie, sont-ils de même nature et ont-ils alors valeur de syndrome? 2) si ce syndrome représente un déficit de la motivation, des hypothèses testables peuvent-elles être proposées?

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Historique

Le néologisme « athymhormie » (1) a été créé en 1922 par les psychiatres Maurice Dide et Paul Guiraud à partir du privatif latin a et de deux mots grecs, l'un thumos qui signifie l'humeur, l'autre hormé dont le sens est moins connu. Le mot hormé, formé à partir du verbe « ὸρμαω » qui veut dire aller vers l'avant ou se précipiter était déjà utilisé par les Stoïciens. Sa signification est très riche car il désigne l'impulsion, mais également l'appétit, la tendance (2). Il est remarquable que ce terme ait été réutilisé presque au même moment au début de ce siècle par des auteurs qui ne se connaissaient pas : non seulement par Dide et Guiraud mais aussi par MacDougall (3) qui dans sa théorie hormique le définissait comme un processus intentionnel propre à l'homme, et par Von Monakow et Mourgue (4) pour lesquels il représentait la tendance primitive propulsive de l'être vivant, la « matrice des instincts ». En utilisant ce terme, tous ces auteurs se rattachaient à la tradition vitaliste et le mot hormé est ainsi proche du concept d'« élan vital » développé par Bergson (5).

La création par Dide et Guiraud du terme « athymhormie » répondait à plusieurs objectifs. En introduisant l'expression « athymhormie juvénile » dès les premières lignes de leur chapitre sur la démence précoce (DP) de leur manuel de

psychiatrie (1), Dide et Guiraud proposaient d'abord cette expression pour remplacer le terme de DP introduit par Kraepelin (6), lequel leur paraissait « inadéquat ». La condamnation du terme de DP n'était pas originale et Dide et Guiraud suivaient en cela la majorité des psychiatres de leur époque qui estimaient que la maladie ainsi désignée n'évoluait pas vers une démence, au sens spécifique où ce terme était employé et qui est celui que nous utilisons toujours (pour Kraepelin, la DP n'était en réalité qu'une pseudo ou para-démence).

Dide et Guiraud voulaient surtout empêcher le remplacement du terme de DP par celui de schizophrénie, qui avait tendance à s'imposer. Ainsi c'est contre la schizophrénie de Bleuler (7) que l'« athymhormie juvénile » a été créée, car la schizophrénie de Bleuler d'une part rassemblait, selon Dide et Guiraud, une « foule d'affections disparates », et d'autre part proposait pour cette affection une conception psychogène inspirée de la psychanalyse, ce qui paraissait « inadmissible » à Dide et Guiraud.

Avec le terme d'« athymhormie juvénile », Dide et Guiraud proposaient une définition plus précise du trouble fondamental de la DP Leur description va en effet mettre l'accent sur son noyau même : « l'affection se caractérise par un fléchissement d'emblée et précoce des sources instinctives de la vie mentale...; l'affaiblissement de l'élan vital et de l'affectivité étant l'élément nécessaire et suffisant pour caractériser la maladie ». L'athymhormie constituait ainsi un « syndrome minimum essentiel » comprenant, selon les auteurs, une série de symptômes fondamentaux (désintérêt, inertie, fléchissement des sentiments affectifs, ambivalence, sentiment pénible d'étrangeté) auxquels s'ajoutaient des symptômes secondaires tels que troubles intellectuels, délires, troubles de l'activité motrice et troubles physiques. Les symptômes de la DP, renommée « athymhormie juvénile », correspondaient à une organisation hiérarchisée à partir d'un déficit fondamental (l'athymhormie). On retrouve ici la façon dont Bleuler avait lui-même ordonné les symptômes de la DP, renommée par lui « groupe des schizophrénies », mais à partir d'un tout autre trouble fondamental : la Spaltung. Ce n'est donc pas dans cette construction que réside l'originalité de l'athymhormie mais dans son étio-pathogénie, car les auteurs attribuaient ce trouble à « l'involution élective de groupes de cellules du système sympathique situés au niveau de la région sous-optique et comprenant notamment le locus niger mais aussi le putamen et le noyau caudé ». L'atteinte intellectuelle leur paraissait secondaire et liée à l'extension des lésions au cortex cérébral notamment frontal. Ils s'opposaient de la sorte à la conception, défendue par Klippel et Lhermitte (8), d'une atteinte corticale primitive au cours de la DP.

Le concept d'athymhormie correspondait ainsi à un modèle anatomo-clinique classique. Il s'agissait pour ses auteurs d'une maladie de l'encéphale, et plus précisément du diencéphale, et l'on comprend leur refus d'abandonner la conception d'une psychose déficitaire juvénile, d'origine pour eux organique (la DP), au profit d'une schizophrénie psychogène et aux limites floues (les auteurs simplifiaient la pensée de Bleuler qui n'éliminait nullement une origine organique à la schizophrénie).

La volonté de sauvegarder la notion d'une maladie déficitaire juvénile ne sera jamais abandonnée par les auteurs même si par la suite ce sera le terme d'hébéphrénie (9) qu'ils utiliseront pour la désigner. Quant au terme d'« athymhormie », il va toujours désigner pour eux le déficit psychopathologique fondamental caractérisant cette affection (l'hébéphrénie).

Dide et Guiraud, comme d'autres grands psychiatres de cette époque, avaient une formation et une pratique de neurologiste, d'anatomiste et de neuropathologiste.

Dide avait décrit en 1902, avec Botcazo, un syndrome d'amnésie occipitale qui porte leur nom (10) et reste connu des neurologues. Sa rencontre avec Guiraud fut décisive en ce qui concerne l'athymhormie et surtout l'affirmation que ce syndrome clinique relevait de lésions neuropathologiques précises à localisation sous-corticale. Malheureusement les auteurs, en se montrant si affirmatifs, faisaient plus état de leur conviction et de « travaux en cours » que de travaux publiés et reproduits. Dide poursuivait depuis des années des recherches de corrélations clinico-biologiques entre les signes physiques végétatifs de la DP et des lésions du système sympathique (11). En 1920 il avait présenté avec Guiraud un procédé de coloration électif des granulations lipoïdes cellulaires (12) grâce auquel ils avaient pu affirmer qu'il existait au niveau des noyaux gris une dégénérescence granulograisseuse, et que ce type de lésion se retrouvait, mais de façon secondaire, au niveau du cortex chez ces malades.

Dide continua ses recherches dans cette voie et en 1926 il publia, dans la Revue Neurologique, une note préliminaire sur ces lésions cellulaires du sympathique qui lui paraissaient confirmer la théorie d'une « essence végétative de la DP, maladie de l'élan psycho-génétique » (13). Dans sa dernière publication en 1939, qui fait la synthèse de tous ses travaux, il continuait à affirmer, à partir d'une centaine de cas d'athymhormie vérifiés, la réalité de lésions cyto-dystrophiques neurovégétatives diencéphaliques (14). Sur la vie et l' euvre de Dide on pourra consulter l'ouvrage de Mangin-Lazarus (15).

Guiraud de son côté s'intéressait aux analogies entre les manifestations neurologiques de certaines affections cérébrales démontrées et certains symptômes de la DP, et il avait

été attiré par le modèle représenté par l'encéphalite léthargique (16,17). La fréquence des états catatoniques que l'on pouvait y observer lui paraissait confirmer l'origine sous-corticale des grands syndromes mentaux. Dans son opus magnum, Psychiatrie générale (17), Guiraud développa longuement sa conception d'un psychisme sous-cortical et d'un « système hormothymique » ainsi que la notion dérivée d'un « éprouvé vital continu » correspondant à un sentiment d'être non conscient, par rapport à un constaté réflexif et conscient. Dans cet ouvrage et dans la dernière édition de son traité en 1956 (18), il distinguait dans l'athymhormie trois composantes : 1) l'anhormie correspondant au fléchissement du dynamisme vital instinctif, auquel il attribuait un rôle direct dans l'hébéphrénie ; 2) l'athymie qui correspondait à l'aspect subjectif du même phénomène ; 3) une composante effectrice dont le défaut expliquait l'inertie de ces malades.

Malgré les efforts de Dide et Guiraud, le terme de schizophrénie va s'imposer en France car il était défendu par de jeunes psychiatres liés au mouvement de la psychiatrie dynamique alors en plein essor (19). On soulignera ici l'influence d'Eugène Minkowski, élève de Bleuler et de Bergson, qui avait développé dans sa thèse (20) l'idée qu'il existait, au cours de la schizophrénie, un trouble fondamental qu'il avait pour sa part appelé « perte de contact vital avec la réalité » mais qu'il reconnaissait proche de l'athymhormie de Dide et Guiraud. Privé de l'adjectif juvénile et ne désignant plus une maladie mais un symptôme fondamental, le terme d'athymhormie va néanmoins rester vivace en France pour définir, surtout au cours des formes hébéphréniques de la schizophrénie, un noyau déficitaire fait de froideur émotionnelle, d'indifférence et de dévitalisation, et il est ainsi décrit dans tous les manuels français actuels de psychiatrie (21–23).

Reprise du terme en neurologie

L'intérêt porté au concept d'athymhormie a été renouvelé en 1988 quand deux neurologues, Habib et Poncet (24), publièrent dans la Revue Neurologique un article où ils utilisaient ce terme pour désigner le tableau comportemental très particulier réalisé par des lésions lacunaires des corps striés, mais également attribuable à des lésions bi-pariétales et frontales. Le tableau comportait, selon les auteurs, les trois aspects correspondant à la définition de l'athymhormie selon Guiraud (1956), à savoir une composante anhormique faite d'une perte spécifique de désir et d'intérêt pour les préoccupations et motivations antérieures, une composante athymique avec perte du sentiment subjectif de l'« éprouvé » agréable ou désagréable d'une situation, et une composante effectrice responsable d'une perte de la spontanéité avec dépendance et passivité. Un point essentiel était que ce trouble était isolé, sans détérioration intellectuelle notable et sans dépression.

Les auteurs estimaient que le fait que ce tableau clinique puisse survenir à la suite de lésions cérébrales de sièges divers soutenait l'hypothèse d'un circuit fonctionnel unique, qui d'après eux correspondait au système hormothymique de Guiraud. Ce circuit fonctionnel était assimilé à la boucle limbique de Nauta (25), laquelle oppose le striatum dorsal (ou moteur) à un striatum ventral (ou limbique) ici impliqué. La boucle limbique correspond à un circuit reliant l'aire cingulaire antérieure du cortex frontal à la partie ventrale du noyau caudé, mais ce striatum ventral reçoit aussi de nombreuses afférences de divers éléments constitutifs du système limbique et projette sur le pallidum ventral puis le noyau médiodorsal du thalamus, mais également sur d'autres structures : noyau amygdalien, aire tegmentale ventrale. Les auteurs rapprochaient le comportement de leurs malades de celui de patients atteints d'hébéphrénie tout en soulignant, d'une part, que l'apathie, le désintérêt et l'apragmatisme appartiennent classiquement au syndrome frontal, qu'il soit acquis ou provoqué, et d'autre part, que des troubles de l'affectivité et de la motivation sont des caractéristiques des démences sous-corticales. Habib et Poncet, en présentant leurs cas, avaient signalé l'importance des travaux antérieurs de Laplane et autres. (26,27) qui avaient décrit, dès 1981–1982, sous le terme de perte d'auto-activation psychique (PAAP puis PAP) un tableau similaire survenant lors de lésions bilatérales du pallidum, mais aussi lors de lésions frontales. Avec ce terme de PAP, Laplane tenait à souligner deux aspects comportementaux essentiels de son syndrome, à savoir d'une part l'impossibilité d'accomplir spontanément des actes qui étaient par ailleurs parfaitement exécutés sur ordre, et d'autre part le fait que ces malades qui, laissés à eux-mêmes, sont inertes décrivent un état de vide mental. Ce syndrome pouvait aussi comporter, selon Laplane et autres, des phénomènes obsessionnels parfois au premier plan.

De nombreux auteurs, surtout français (pour une revue voir 28), ont décrit les années suivantes des tableaux semblables utilisant soit le terme d'athymhormie, soit celui de PAP tandis que Habib et Laplane et Dubois (29) poursuivaient une controverse fructueuse sur l'utilisation de ces deux termes. Laplane (30) soulignait notamment la valeur heuristique du terme de PAP, insistant sur la remarquable réversibilité des troubles cognitifs mais également affectifs sous l'effet d'une hétéroactivation. Dans sa conception des rapports cerveau-pensée (31), il sous entend qu'un système de « l'action et de la pensée », par ailleurs intact et probablement localisable aux parties les plus nobles du cerveau (dont le lobe frontal) n'est plus actif.

Habib, dans ses derniers travaux (28) et à partir, soulignons-le, des mêmes situations cliniques, a entrepris une étude neuro-psychologique et neuro-anatomique de la motivation. Il en conclut que l'origine du syndrome athymhormique

serait une perte de la signification affective des stimuli, c'est à dire de la motivation. Celle-ci dépendrait à la fois de besoins internes primaires, dont les renforcements positifs sont innés, autant que des stimuli de l'environnement créant l'envie d'agir (incentive stimuli des Anglo-Saxons). L'auteur, en accord avec des travaux contemporains, fait une part essentielle à l'amygdale et à ses connexions avec le striatum limbique tout en reconnaissant d'une part le rôle du mésencéphale en tant qu'il est réservoir de dopamine (DA) et d'autre part, celui des neurones dopaminergiques qui ne modulent leur activité que lorsque le stimulus a une signification (un intérêt). Pour lui, le syndrome athymhormique est ainsi un trouble spécifique de la capacité des sujets à convertir leur affect en action, et il souligne que « l'attribution à des lésions cérébrales de troubles spécifiques de la motivation constitue une contribution récente de la neurologie clinique, principalement francophone, au domaine des neurosciences et à la connaissance du substrat cérébral des fonctions mentales » (28). Par rapport à la conception de Laplane, M. Habib défend l'idée que les structures critiques intervenant dans la pensée ne sont pas corticales (récentes) mais sous-corticales (archaïques). On voit la continuité avec l'euvre de Guiraud (17).

Situation actuelle

Si nous voulons maintenant essayer de répondre aux questions que nous nous étions posés, il semble bien que le déficit spécifique du comportement et des affects que l'on observe après des lésions circonscrites des noyaux gris constitue une réalité phénoménologique indiscutable, et cela, quelle que soit la façon dont on le nomme : PAP, athymhormie, voire même aboulie ou apathie selon Habib (28). Tous les cas publiés correspondent à des atteintes bilatérales, mais qui ne sont pas forcément symétriques ni de même nature. Ces constatations anatomiques s'intégreraient bien à l'hypothèse selon laquelle le syndrome athymhormique serait lié à l'interruption bilatérale, à un niveau quelconque, d'un circuit qui serait celui de la motivation (Habib) ou de la volonté (Laplane). Toutefois, la question se pose du rôle respectif de la partie gauche ou droite de ce circuit avec l'hypothèse selon laquelle c'est l'atteinte de la partie gauche qui serait responsable, chez les droitiers, de l'aspontanéité verbale (32,33).

Si nous revenons à la psychiatrie, l'« athymhormie juvénile » de Dide et Guiraud paraissait avoir complètement disparu, englobée dans la schizophrénie et « vaincue » par elle. Il est remarquable que des travaux contemporains, nous pensons à ceux de Carpenter et de l'école de Baltimore (34) réhabilitent et défendent la réalité d'une entité négative primaire (c'est à dire excluant le rôle de la dépression et de l'effet des neuroleptiques), qu'ils incluent cependant à titre de sous groupe dans la schizophrénie (35). Cette forme négative primaire aurait, selon Ribeyre et autres (36), une base biochimique spécifique (hypoactivité dopaminergique) qui, si elle était confirmée, redonnerait du poids à une individualisation de cette forme, par rapport à la schizophrénie.

Mais la dopamine n'est probablement pas seule en cause. Dans une étude menée auprès des psychotiques sur l'activité de l'enzyme de synthèse de l'adrénaline, la phényléthanolamine-N- méthyltransférase (PNMT), N Kopp et autres (37) ont constaté il y a quelques années une baisse de l'activité de synthèse de cette enzyme, au niveau du nucleus accumbens et de la substantia perforata posterior. N Kopp et autres faisaient alors le rapprochement avec l'étude de Dide (13) qui découvrait avec les méthodes histologiques classiques une perte de neurones dont la morphologie évoquait à l'auteur une nature sympathique, dans la région interpédonculaire, qui correspond à la substantia perforata posterior et à l'aire A 10 (laquelle est le siège, on le sait maintenant, des corps cellulaires dopaminergiques méso-limbiques). On signalera ici que plusieurs constructions théoriques concernant la schizophrénie ont impliqué les noyaux gris (38,39) ou l'atteinte de circuits les impliquant (40,41). Si aucune ne s'est imposée, n'est-ce pas parce que ces travaux concernaient non pas une réalité clinique limitée, mais un ensemble bien trop vaste et finalement douteux : la « schizophrénie »(42)?

Quant à l'« athymhormie », en tant que noyau symptomatique des formes déficitaires, on ne pourra pas s'empêcher de la rapprocher des symptômes négatifs précédés également du privatif a,isolés par Andreasen et autres (43) au sein des formes déficitaires de la schizophrénie. Ces symptômes : avolition-apathie, anhédonisme-associabilité, alogie et surtout émoussement affectif ne font que reprendre de façon détaillée la notion d'athymhormie. La différence, c'est que Dide et Guiraud avaient l'ambition de découvrir un trouble fondamental, dont tous les autres symptômes de la DP découlaient, et qu'ils proposaient pour ce trouble un site lésionnel. Les classifications en vigueur, DSM IV et CIM 10, ont adopté, on le sait, une position bien différente puisqu'elles sont, en principe, athéoriques et que le diagnostic ne s'appuie plus sur un « maître symptôme », mais sur un ensemble de troubles, peu ou pas hiérarchisés. Ces classifications restent imprégnées d'une conception anorganique des psychoses, puisque leurs critères devraient faire rejeter le diagnostic, quand on découvre une atteinte cérébrale manifeste.

Un même syndrome ?

Mais peut-on utiliser le même mot, athymhormie, pour désigner des troubles psychiatriques et neurologiques dont on affirmerait ainsi l'identité, selon nous? Certes, l'athymhormie des psychiatres et celle des neurologues s'intègrent dans des tableaux cliniques bien différenciés qui correspondent à la distinction classique entre maladies neurologiques et

maladies psychiatriques (44). Toutefois, le schématisme de cette distinction s'estompe avec la mise en évidence ces dernières années, au cours des psychoses, d'anomalies cérébrales morphologiques mais surtout fonctionnelles (dont l'évolution n'est pas toujours réversible), ce qui pourrait constituer à terme le support des aspects déficitaires stables que l'on redécouvre. C'est surtout le renouveau d'une pensée neuropsychiatrique (mouvements de la neurologie des comportements, de la neuropsychiatrie cognitive) qui a démontré tout l'intérêt que représentent certains symptômes (ou syndromes) communs aux deux spécialités, car ils peuvent signifier le déficit de capacités comportementales et fonctionnelles physiologiques, et répondre ainsi à une même pathogénie. Parmi ces syndromes, l'athymhormie occuperait une place de choix, si elle représente bien le déficit d'une fonction précise, qui est celle de la motivation. L'hypothèse selon laquelle il existerait, au cours d'affections démontrées de l'encéphale et au cours de certaines psychoses, une atteinte anatomique lésionnelle ou fonctionnelle d'un circuit neuronal support de la motivation peut déjà faire l'objet de travaux de recherche en imagerie cérébrale. Sur le plan clinique et comportemental, des échelles d'évaluation ont été proposées (45,46) pour mesurer l'intensité des symptômes de l'athymhormie. Enfin, le déficit spécifique de certains neuromédiateurs (ou modulateurs) qui a été supposé (47) ou constaté (35) mériterait d'être vérifié, ce qui permettrait de comprendre l'effet favorable de certaines thérapeutiques et d'en proposer de nouvelles.

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Abstract — The French Concept, "Athymhormie" from 1922 to the present

The psychiatric concept of athymhormie *(loss of vital force "élan vital" and affectivity) was developed at the beginning of the 20th century and used exclusively in France, until it was fortunately adopted by mostly French-speaking neurologists. The authors of the concept, M Dide and P Guiraud, considered the term an independent entity, an heir to* dementia praecox*, for which they did not like to substitute a "schizophrenia" with blurry boundaries. This term then came to mean to them, and to most French psychiatrists to this day, the basic core of psychosis' deficient forms. The word* athymhormie *(or loss of psychic self-activation) is now also used in neurology for the behavioural and emotional consequences of lesions caused on basal nuclei and, more widely, of a circuit associated with the limbic loop. However, the phenomenological reality of the associated disorders and the relevance of the same name can be questioned. This review concludes that although the settings—in a psychiatric or a neurologic clinic—where* athymhormie *takes place are very different, the choice of the same name is justified in a renewed and symptomatic neuropsychiatric approach. The objective of this approach is to explore why some symptoms, common or similar, could represent the lack of a physiological function, presumed here to be motivation.*

BRIEF COMMUNICATIONS

Treating Psychosis: Is There More to Early Intervention than Intervening Early?

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While the possibility of early intervention following the initial onset of psychotic disorders such as schizophrenia is an exciting development, a closer examination of the nature and content, as well as the timing, of treatment is required for this new approach to be successful. Modification and integration of diverse treatments need to be empirically investigated for their potentially greater effectiveness in patients who are, in general, much younger, naive to the mental health system, and possibly capable of full integration into society. Reducing delay in treatment initiation may be complex, may involve adopting early identification strategies, and may pose significant systemic and conceptual challenges. The 2 aspects of early intervention—integration of phase-specific treatments and early case identification—need to go hand in hand to ensure that another opportunity will not be missed in our efforts to improve the outcomes of these most serious of all mental disorders.

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Key Words: *early intervention, psychosis*

Recent years have seen much enthusiasm about the possible benefits of early intervention in cases of psychosis (1–3). This is the result of evidence supporting an association between delayed treatment and such aspects of short-term outcome (at least) as the rate, timing, and level of remission (4), the level of subsequent negative symptoms, and some aspects of quality of life (2). In a recent review of the literature, Norman and Malla (5) concluded that, notwithstanding several methodological issues and several studies reporting negative results (6,7), there is a strong relation between duration of untreated psychosis (DUP) and short-term outcome. An early-intervention strategy is also likely to have been spurred by recent advances in pharmacologic treatment (8), an ever-increasing shift in the care of those with serious mental illness to the community, and an emphasis on disease prevention in general (9).

There are, however, several issues that need close examination when clinicians attempt to incorporate early intervention of psychosis into their practice. Essentially these issues revolve around the question, "Is early intervention anything more than providing treatment earlier than usual?" This question forces us to examine 2 issues central to early treatment of psychosis, namely, its content and timing.

Nature and Content of Treatment

Studies that provide empirical evidence for integration of drug and psychosocial interventions as the preferred approach to treating psychotic disorders (10,11) have been carried out largely in patients who are in later, more chronic stages of illness. It is likely that a combination of novel antipsychotics, which have a significantly lower potential for motor and cognitive side effects, and phase-specific psychosocial interventions (for example, family intervention, cognitive therapy) will prove even more beneficial early in the course of the illness. We now know that substantially lower dosages of medication are needed for optimal efficacy and safety in treating a first episode of the illness (8,12,13). It is also likely that the content of psychological treatments, including family intervention (14), skills training (15), and cognitive therapy (16) will require significant modifications to address the specific needs of relatively younger patients and their families.

Clinical experience and evidence suggest that patients' needs during the early phases of the illness differ from those individuals with longer-standing illness (17,18). The former are generally young, living with their families, attempting to negotiate the normal developmental phases of late adolescence and young adulthood, and using alcohol and drugs socially or

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excessively. Moreover, they are often still dealing with the initial personal trauma of psychosis, with strong hopes of returning to a normal level of functioning, and are more likely to reject a paternalistic approach to medical interventions. The families of such patients are likely younger, with needs and expectations related to the presence of other children of varying ages, higher expectations of treatment benefits, and possibly a higher level of knowledge about psychotic illnesses and their treatment gained through new information systems such as the Internet. If we do not consider these issues when working with patients and their families, we may not fully realize the potential benefits of early intervention.

The above considerations indicate the urgent need to design interventions that address the needs and circumstances specifically associated with early intervention (18–21) and to conduct effectiveness trials of such integrated treatments in first-episode patients. It is essential that the content of such interventions be informed by careful assessment of needs and empirical evidence regarding effectiveness. Otherwise, clinicians are likely simply to employ traditional treatments somewhat earlier on, and we will have missed an important opportunity to significantly alter the course of these serious mental disorders.

There may also be a need to substantially modify our philosophy of treating individuals at early stages of psychotic disorders who are having their first contact with the mental health system. The guarded (sometimes pessimistic) expectations of very limited recovery (22,23), which often characterize approaches to the treatment of longer-term psychosis, must not be allowed to become a self-fulfilling prophecy in early intervention. We may assume too quickly that persons with a first-time diagnosis of a psychotic disorder need social and vocational rehabilitation, thereby overlooking the possibility that they may often be capable of resuming social and economic roles earlier and more readily than we anticipate. We may presume disability as the natural consequence of the diagnosis of schizophrenia or a related disorder—either perpetuated or even inadvertently encouraged by our unnecessarily invoking formal supports or through a paternalistic approach to care.

Timing of Treatment (Intervening Early)

The second equally important objective is to reduce the initial delay in providing effective interventions. The causes of delay in initiating treatment for psychotic disorders are poorly understood. They are likely complex and may include systemic, individual, and family factors, as well as general community attitudes toward mental illness (24,25).

The need to provide treatment early highlights the importance of systemic changes. These include a shift away from a strictly hospital-based emergency and referral-driven clinical practice to assertive community assessment and integrated treatment designed to readily engage ill persons and their families in treatment. Such engagement is more likely to occur if treatment is provided largely within an environment agreeable to individuals and their families—typically using inpatient beds only if and when necessary. Early intervention requires clinicians to shift their conceptualization of psychotic disorders to recognize the limitations of any diagnostic system (26), and to confront the uncertainty inherent in generically diagnosing a psychotic syndrome at the time of the initial assessment. It also requires a better understanding of developmental issues in adolescence and early adulthood and a greater use of various psychotherapeutic interventions, especially cognitively oriented individual and group therapies that incorporate a dynamic understanding of individuals and their families (18,21). Such interventions may have been regarded traditionally as not relevant to schizophrenia and other psychoses.

While case identification requires an operational definition of "caseness," reaching agreement on such definitions and the relevant symptom thresholds may be complex. Clinicians, for example, are more likely to agree about a patient showing evidence of psychotic symptoms (for example, delusions and hallucinations) of moderate intensity present over a certain defined duration (at least 1 week), compared with a patient who shows only thought disorder or bizarre behaviour or only brief episodes of delusions and hallucinations.

Early identification of cases of psychosis may require generic community education campaigns regarding mental illness as well as specific assertive case-detection programs for psychosis. The former are likely to increase a community's knowledge about mental illness, while the latter are more likely to lead to increased case identification (27). Effective and safe case identification will require assertive approaches that not only increase correct identification of early psychosis (increasing the number of true positives and reducing the number of false negatives) but also reduce the risk of incorrectly identifying individuals as suffering from a psychotic disorder (reducing false positives and increasing true negatives).

We must also be cautious about treating individuals in a (putative) prodromal phase of psychosis. There are particular concerns in this respect, given the low predictive validity of putative prodromal symptoms (28–30). Greater utility is likely to be found in the concept of the "at risk mental state," which comprises criteria indicating functional decline within the context of positive family history in a first-degree relative, subthreshold psychotic symptoms, and episodic brief psychotic symptoms lasting 1 or 2 days (31,32). Interventions carried out during periods preceding a syndromal level of psychosis must consider risk–benefit ratio of such treatment. The role of psychological interventions may be even more crucial during the prepsychotic phase and deserve controlled investigations with and without low-dose novel antipsychotic

medications. Given the current state of knowledge, we would not advise incorporating treatment of these prodromal or even "at risk mental states" into everyday clinical practice until the benefits of any such intervention have been well established empirically.

With improved knowledge about psychosis from public education, individuals in the community and their families are more likely to seek professional guidance regarding the risk and presence of a psychotic disorder and expect competent assessment and treatment to be available quickly. Until there are substantial systemic changes in the mental health delivery system, the expectations and hopes raised by community education campaigns could be disappointed. While dissemination of current knowledge about psychosis and its early identification should not be discouraged, such initiatives carried out nationally or provincially must presume adequate service availability in each jurisdiction. Although this is far from being achieved, it could signal the need to establish phase-specific early-intervention assessment and treatment programs as a national and provincial strategy. Several countries, such as Australia, England and Wales, and several of the Scandinavian countries, have already embarked on such initiatives (33–36). We hope that similar strategies will be endorsed in this country, with due attention to details for integrated and phase-specific service, education, and evaluation. This would encourage individual initiatives in various jurisdictions in Canada (37).

In conclusion, 2 essential aspects of early intervention—namely, improved integration of phase-specific medical and psychological treatment and reduced treatment delays following the onset of psychosis (through early recognition)—must go hand in hand. In future, improving both content and timing will be crucial in bringing about better outcomes for those who develop psychotic illnesses. If empirically driven modifications and integration of biological and psychosocial treatments are not achieved as part of an early-intervention strategy, the whole idea is likely to be rejected as a failure, because improving treatment timing alone will bring limited, if any, improvement in outcome.

Clinical implications

- This review will provide some guidelines for pursuing further work in early intervention in psychosis cases.
- This paper may encourage clinicians to integrate new psychological interventions and developments in the pharmacotherapy of early psychosis.

Limitations

- This is not an exhaustive review of the literature on early intervention in psychosis.
- There are no empirical data available on the effectiveness of best practices in treatment of early psychosis.

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Résumé— Traiter la psychose : l'intervention précoce se limite-t-elle à intervenir tôt?

Même si la possibilité d'une intervention précoce suivant la première apparition de troubles psychotiques comme la schizophrénie est un développement très intéressant, il faut un examen plus rigoureux de la nature, du contenu et de la durée du traitement, pour faire de cette approche un succès. Il faut examiner empiriquement la modification et l'intégration de différents traitements en vue de leur plus grande efficacité éventuelle chez les patients qui sont en général plus jeunes, novices du système de santé mentale et possiblement capables d'une intégration complète dans la société. Réduire le délai du début du traitement peut être complexe, obliger à adopter des stratégies d'identification précoce, et présenter des défis systémiques et conceptuels de taille. Les 2 aspects de l'intervention précoce – l'intégration de traitements liés à la phase et l'identification précoce d'un cas – doivent aller de pair pour ne pas rater une autre occasion d'améliorer les résultats de ces troubles mentaux parmi les plus graves.

BRIEF COMMUNICATION

Peritraumatic Dissociation, Acute Stress, and Early Posttraumatic Stress Disorder in Victims of General Crime

Philippe Birmes, MD[1], Didier Carreras, MD[2], Jean-Louis Ducassé, MD[3], Jean-Paul Charlet, MD[4], Barbara A Warner, MD[5], Dominique Lauque, MD[6], Laurent Schmitt, MD[7]

Objective: *To compare the relation between peritraumatic dissociation and acute stress and the early development of posttraumatic stress disorder (PTSD) in victims of general crime.*

Method: *A total of 48 subjects were assessed within 24 hours of the trauma, using the Peritraumatic Dissociative Experiences Questionnaire Self-Report Version (PDEQ-SRV). They were followed longitudinally to assess acute stress (2 weeks after the assault,) using the Standford Acute Stress Reaction Questionnaire (SASRQ), and posttraumatic stress (at 5 weeks), using the Clinician-Administered PTSD Scale (CAPS) and the Impact of Event Scale (IES).*

Results: *Among PTSD subjects mean PDEQ scores were significantly higher (mean 3, SD 0.9) than in those without PTSD (mean 2.3, SD 0.7) (*t *= 2.78, df 46,* P *= 0.007). Among PTSD subjects, mean SASRQ scores were significantly higher (mean 97.9, SD 29.2) than in those without PTSD (mean 54.8, SD 28.2) (*t *= 4.9, df 46,* P *= 0.00007).*

Conclusions: *High levels of peritraumatic dissociation and acute stress following violent assault are risk factors for early PTSD. Identifying acute reexperiencing can help the clinician identify subjects at highest risk.*

(Can J Psychiatry 2001;46:649–651)

Key Words: *acute stress, peritraumatic dissociation, posttraumatic stress disorder, risk factors*

The relation between peritraumatic dissociation (immediate dissociation at the time of the traumatic event) and posttraumatic stress disorder (PTSD) has been studied following combat exposure, disasters, critical incidents, and motor-vehicle accidents. Victims with acute dissociative symptoms appear to be at greater risk for PTSD, independent of trauma exposure level, general dissociative tendencies, and general psychopathology (1–4). Marmar and others (2,3) have proposed a measure of acute dissociative response to trauma: The Peritraumatic Dissociative Experiences Questionnaire (PDEQ). The PDEQ Self-Report Version (PDEQ-10–SRV) contains 10 items that address dissociative experiences at the time of the traumatic event. These include moments of losing track or blanking out; finding the self acting on "automatic pilot"; sense of time changing during the event; the event seeming unreal; feeling as if floating above the scene; feeling of body distortion; confusion about what was happening to the self and others; not being aware of things that happened during the event; and disorientation (3). Some studies have been limited by retrospective design. Prospective and longitudinal assessments provide a powerful methodology to study PTSD risk factors.

Acute stress disorder includes a set of symptoms experienced in the first month following a traumatic event. The victim must present 3 dissociative symptoms and at least 1 each of the following symptoms: intrusion, avoidance, and hyperarousal (5). Recent studies show that acute stress disorder after mass shootings, traffic accidents, and violent crimes strongly predict later PTSD development (6–8).

In the current study, subjects were interviewed within 24 hours of an assault to assess the presence of peritraumatic dissociation. They were reinterviewed after 2 and 5 weeks to assess acute stress and posttraumatic stress. These results compared the predictive powers of peritraumatic dissociation

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and acute stress disorder (with its different clusters) for early PTSD at 5 weeks.

Methods

During a 2-month period, violent assault victims were consecutively recruited in a general hospital emergency department. Exclusion criteria included a head injury with unconsciousness, misuse of alcohol or drugs, suffering from a psychotic disease or from serious physical illness, or a current life-threatening injury. Eligible subjects gave written informed consent and were assessed in the emergency unit with the PDEQ-10–SRV (3). We followed 48 subjects to assess acute stress 2 weeks after the assault, using the Stanford Acute Stress Reaction Questionnaire (SASRQ), and posttraumatic stress 5 weeks after, using the Clinician-Administered PTSD Scale (CAPS) and the Impact of Event Scale (IES) (9–11).

Senior medical doctors who were experienced with the PDEQ-10–SRV provided instructions to each subject. For each of the 10 items, the subject rated a 5-point Likert scale (5 = extremely true, 4 = very true, 3 = somewhat true, 2 = slightly true, 1 = not at all true) (2,3). The PDEQ-10–SRV was scored as the mean item response across all items, and scores ranged from 1.0 to 5.0. The coefficient alpha for these data was 0.79.

Subjects were given the SASRQ at 2 weeks; we used the CAPS at 5 weeks; and we also gave subjects the IES at 5 weeks. The SASRQ was scored as the total sum of raw scores for the scale (with scores ranging from 0.0 to 150.0) and for the following subscales: dissociative (SASRQd); reexperiencing (SASRQr); avoidance (SASRQa); and hyperarousal (SASRQh) (10). A symptom was scored as present if the respondent marked it as occurring at least "sometimes," which includes a response of 3 or higher. An acute stress disorder diagnosis required the presence of at least 3 dissociative symptoms and 1 symptom of reexperiencing, avoidance, marked arousal, and distress or impairment according to DSM-IV (5,10). We used the CAPS to classify the subjects as either having PTSD or non-PTSD status. Similarly, we used the IES to confirm this assessment, with a cut off score of 42. All mean resultant scores are reported with standard deviation. Analyses used *t*-tests to examine a possible association between the presence or absence of PTSD and continuous variables; they used Fischer's exact tests when looking for a possible association between the presence or absence of PTSD and binary variables. The item levels of the Likert scale were compared between the 2 groups, using the Ridit test specifically dedicated to gradient comparisons.

Results

A total of 48 subjects completed the protocol: 21 (43.8%) were men, and most subjects were married (n = 30, 62.5%). The mean age at the time of the assault was 38.2 years (SD 15.8).

All were victims of violent assault: 21 (43.8%) were shot, stabbed, mugged, held up, or threatened with a weapon; and 27 (56.3%) were physically attacked or badly beaten up. All were admitted to an emergency department, and 9 (18.8%) were hospitalized in surgical units.

Diagnoses according to the CAPS and the IES at 5 weeks revealed 16 subjects (33.3%) who met all criteria for PTSD (mean IES scores 53.3, SD 8.2); and 32 (66.6%) did not (mean IES scores 24.8, SD 13.4) (t = 7.77, df 46, $P <$ 0.00001). Neither age nor marital status was related to PTSD status.

The PTSD subjects reported more feeling the self acting on "automatic pilot" (P = 0.022), more feeling of body distortion (P = 0.006), and more disorientation (P = 0.012) than did non-PTSD subjects. The PTSD subjects showed PDEQ-10–SRV scores (mean 3, SD 0.9) which were higher than those of subjects without PTSD (mean 2.3, SD 0.7) (t = 2.78, df = 46, P = 0.007). Of the 48 victims, 28 (58.3%) were diagnosed with an acute stress disorder, without significant correlation to PTSD status. The subjects with PTSD, however, showed SARSQ scores (mean 97.9, SD 29.2) significantly higher than of those subjects without PTSD (mean 54.8, SD 28.2) (t = 4.9, df 46, P = 0.00007). The quantification of the different clusters of acute stress disorder showed equally significant differences between subjects with, and without, PTSD: SASRQd scores, PTSD (mean 26.3, SD 12), non-PTSD (mean 15, SD 11.3) (t = 3.19, df = 46, P = 0.002); SASRQr scores, PTSD (mean 21.9, SD 5.2), non-PTSD (mean 12.4, SD 7.1) (t = 4.7, df 46, P = 0.0001); SASRQa scores, PTSD (mean 20.5, SD 7.9), non-PTSD (mean 11.5, SD 6.7) (t = 4.1, df 46, P = 0.0003); SASRQh scores, PTSD (mean 21.5, SD 5.7), non-PTSD (mean 13, SD 6.7) (t = 4.29, df 46, P = 0.0002).

A logistic stepwise descending regression was used to explain the occurrence of early PTSD. The model started with the 4 SASRQ subscale mean scores as independent variables. The SASRQr was the only independent variable remaining in the process (t = 3.23, df 46, P = 0.002). Having established that the SASRQr was the best subscale of SASRQ for predicting an early PTSD, we tried to improve these findings by introducing the PDEQ-10–SRV mean scores into the model. There was, however, no change, even when we took into account an interaction between the 2 scales (SASRQr and PDEQ-10–SRV).

Discussion

Prospectively assessing early development of PTSD provides a powerful methodology to study traumatized victims meeting criteria in the first month after trauma. These results show that a substantial proportion of traumatized subjects have

reported symptoms of peritraumatic dissociation or acute stress disorder. This study is useful in that it is the only one of general crime victims in which peritraumatic dissociation was measured within 24 hours of the assault (1–4). Data from PDEQ-10–SRV and SASRQ and its subscales obtained after 2 weeks indicate that high levels of peritraumatic dissociation and acute stress seem to be major contributors to the development of PTSD 5 weeks after a violent assault. SASRQr was the most predictive subscale for early PTSD. Diagnoses serve the crucial clinical objective of identifying individuals in need of treatment. In the case of identifying risk factor criteria for developing a later mental disorder, psychometric quantification with reliable instruments might be more sensitive. Other authors have indicated that symptoms of acute stress disorder may simply reflect more severe PTSD, rather than a separate predictive entity (12). Assessing and quantifying acute reexperiencing, however, may help the clinician identify crime victims at highest risk for early development of PTSD.

Clinical Implications

- This study is the only one of peritraumatic dissociation in general crime victims.
- The measure of peritraumatic dissociation was made within 24 hours of the assault.
- This study provides a comparison of predictive powers of peritraumatic dissociation and acute stress for PTSD.

Limitations

- The sample was small.
- The delay between traumatic exposure and assessment of PTSD was 5 weeks.
- This study did not use diagnostic criteria for peritraumatic dissociation.

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Résumé— Dissociation péritraumatique, stress aigu et syndrome précoce de stress post-traumatique chez les victimes de crime en général

Objectif : *Comparer les relations entre la dissociation péritraumatique, le stress aigu et le développement précoce du syndrome de stress post-traumatique (SSPT) chez les victimes de crime en général.*

Méthode : *Un total de 48 sujets ont été évalués dans les 24 heures suivant le traumatisme, à l'aide de la version auto-évaluée du questionnaire sur les expériences de dissociation péritraumatique (PDEQ-SRV). Ils ont fait l'objet d'un suivi longitudinal afin d'évaluer le stress aigu (2 semaines après l'agression) au moyen du questionnaire de Standford sur les réactions au stress aigu (SASRQ), et le stress post-traumatique (à 5 semaines) grâce à l'échelle du SSPT administrée par un clinicien (CAPS) et à l'échelle d'incidence de l'événement (IES).*

Résultats : *Chez les sujets souffrant du SSPT, les scores moyens au PDEQ étaient significativement plus élevés (moyenne 3; écart-type 0,9) que chez ceux sans SSPT (moyenne 2,3; écart-type 0,7) (*t *= 2,78; df = 46;* P *= 0,007). Chez les sujets souffrant du SSPT, les scores moyens au SASRQ étaient significativement plus élevés (moyenne 97,9; écart-type 29,2) que chez ceux sans SSPT (moyenne 54,8; écart-type 28,2) (*t *= 4,9; df = 46;* P *= 0,00007).*

Conclusions : *Les niveaux élevés de dissociation péritraumatique et de stress aigu qui suivent des agressions violentes sont des facteurs de risque de SSPT précoce. L'identification de l'expérience revécue de façon aiguë peut aider le clinicien à détecter les sujets à risque élevé.*

BOOK REVIEWS

Psychopharmacology

Chemicals for the Mind: Psychopharmacology and Human Consciousness. Ernest Keen. Westport (CT): Praeger Publishers; 2000. 142 p. USD59.95.

Review by

Paul Grof, MD, FRCPC
Ottawa, Ontario

This book's title easily generates interest. In recent years, there have been several intriguing multidisciplinary symposia on the nature of consciousness, and the connections with psychopharmacology could be very productive. Thanks to Damasio and others, our grasp of the nature of consciousness has strengthened and is very relevant to the current assumptions so fundamental for psychiatry. Similarly, psychopharmacology has become the cutting-edge tool for our discipline and profession in recent decades.

It is easy to understand Keen's initial concern. He is naturally troubled because many physicians (mostly general practitioners) label normal problems of daily living as depression and prescribe Prozac for them. He would like to see these treated with insight-oriented psychotherapy, presumably offered by psychologists. There is no problem with this view, but the issue could be easily described in a few paragraphs. Keen, however, needs an entire book because he quickly expands his theme from this situation, particularly troublesome in the US, to global statements about psychopharmacology, morality, biological reductionism, and dualism.

Keen's idols are Szasz and Breggin, and all 3 use a similar approach. They start with their ideology and then look for facts that, taken out of context, support their position. Then they generalize their statements: Prozac becomes psychopharmacology, and poor clinical skill is translated into an immoral, violent act against the human being.

Keen arrives at the conclusion that, from the ethical point of view, using medications as part of a reductionistic approach to therapy results in violence to the patient rather than in compassionate psychotherapeutic care. He finds urgent moral issues in psychopharmacology. In his view, we live in a psychopharmacologically incontinent society, and he feels that new drug therapy has "the technological ability to shape ones mind and being" and that "technology morally unchecked may always become violent." He believes that "drugs that change consciousness may impoverish human experience."

To give weight to his points, he explores what he believes are the philosophical roots of the problem and finds them in the unresolved issues of dualism originating with Descartes. For him, biological reductionism "is a psychiatrist's way of avoiding coping with dualism.48 He feels that this reduction then "opens the door to a particular violation of human integrity particular to psychiatry." Whereas a lobotomy was a blatant violation, psychopharmacology is a subtle one. The word "consciousness" appears in the title of the book because Keen sees the inappropriate use of psychopharmacology as an immoral and violent act against "a person's mental life." In the footsteps of Szasz and Breggin, he generalizes that "any experience that is unwanted can be seen as a symptom and it frequently is so labeled." Thus, when "one chemically ablates a personal struggle, one violates "the personal and psychological integrity of the patient." As usual with antipsychiatry, inappropriate use of a particular medication by some becomes a morally despicable act by the whole discipline. Such distortions mean that the original concern, worth mentioning, about the excessive use of Prozac has been lost in the ideological struggle. Thus, the book is not very useful to psychiatrists, even if it were to cost much less than it does.

Schizophrenia

Women and Schizophrenia. David J Castle, John McGrath, Jayashri Kulkarni, editors. New York: Cambridge University Press; 2000. 151p. USD24.95.

Review by

JoAnn Corey, MD
Hamilton, Ontario

This concise, well-written book examines the biological, psychological, and social differences between men and women who suffer with schizophrenia. Although each of the 9 chapters has a different focus, information attended to elsewhere is incorporated to provide a holistic view. This demonstrates the expertise of the 11 contributors. The experience of the editors is also obvious from their introductory caveats concerning the roles women play apart from that of patient. These often-overlooked roles include being the mothers, daughters, wives, informal caregivers, and professional caregivers for people with schizophrenia. Also of credit to the editors is their warning regarding the application of the book's treatment recommendations, given the continually emerging evidence in such a relatively new area of study.

Chapter 2, written clearly by David J Castle, tackles some difficult biological

concepts to explore the sex differences in brain development, organization, and degeneration. This is done on a template of the chronology of a human life. Both strongly supported and less consistently reported evidence of sexual dimorphisms in brain structures are discussed. This includes the fairly recent finding of differences in right–left brain asymmetry, which is important in certain theories of psychosis. Interesting case examples are used to illustrate the process of sexual differentiation of the brain, dependent on the hormonal milieu. Resulting neuropsychological differences are then explored, as are various theories of behavioural differences. The examination of the aging of male and female brains leads into a summative statement of the changes in dopamine receptors over time. This lays the biological foundation on which ensuing chapters are built.

In the third chapter, Dr Castle focuses on debated findings in the epidemiology of schizophrenia. His critical appraisal of the literature identifies the excess of male patients in early-onset illness, a female-preponderant middle-onset peak, and a very late-onset peak that almost exclusively comprises women. Confounding factors that may influence this pattern, such as family history, developmental trajectory, birth complications, and marriage, are identified. The discussion tends to place heavier emphasis on Castle's hypothesis for the findings.

Chapter 4, by Mary V Seeman and Paul Fitzgerald, discusses the differences between men and women in the clinical expression of schizophrenia. They emphasize that women are generally symptomatic longer before first diagnosis than are men. The impediments to early detection that need to be overcome are highlighted; they have important treatment and prognostic consequences. The authors give a thorough and detailed description of symptomatic differences that includes the course of illness, patterns and themes of psychotic symptoms, substance abuse, and triggering life events. (Examples of such life events might have been interesting.) Outcome differences are also thoroughly reviewed and provide a biopsychosocial view of the illness's impact. Once again, the authors connect the chapter to the rest of the book by noting biological and social factors influencing clinical presentation.

Dr Seeman and Dr Fitzgerald also coauthor the chapter on treatment implications. This chapter might have been better-placed after Chapter 4, where it would have followed a natural progression after the discussion of treatment outcome. Again, their writing provides a well-rounded approach to the treatment of women with psychosis. It targets various points in a woman's life that may require special consideration—pregnancy, delivery, postpartum, and menopause. They do not recommend specific medications, but they differentiate between traditional and atypical antipsychotics, particularly in regard to the impact of side effects at each of these phases. Special mention is made of hormonal treatment, which, as a routine adjunct, is still experimental. For example, the authors note the consideration that cyclical variations in estrogen may require cyclical alteration of antipsychotic doses. They also stress the fact that improved representation of women in drug trials is needed to better target responses to treatment, dosing schedules, and safety.

In Chapter 5, Jayashri Kulkarni and George Fink discuss the role that hormones may play in psychosis. They comment on the lack of neuroendocrine studies. A clearly worded explanation of the recently proposed estrogen protection hypothesis is given, along with the supporting evidence from epidemiological, clinical, and biological studies. The authors briefly mention the exciting future possibility of selective estrogen receptor modulators as a treatment adjunct.

Chapter 6, by Joanne Barkla and John McGrath, is a highly effective review of the needs of women with schizophrenia in regard to family planning and antenatal care. It spans the areas of epidemiology, biology, and sociology to impart the realities of pregnancy for these women. The need for preconceptual counselling and antenatal care as a form of primary prevention is stressed. This is something that health professionals believe they are doing but that patients do not perceive them to be doing, according to at least 1 study. The authors bravely address possible reasons for such avoidance—among which are the ethical and legal issues, that may arise. More exploration of this, though requiring another chapter, would have further enhanced the quality of the book.

McGrath and coauthor Jenny Hearle then proceed to illustrate the stress that is added by having a serious mental illness combined with parenting, including the pressures stemming from societal attitudes. Much of the evidence comes from their own recent work, and it might have been useful to corroborate this with data from other sources. They do, however, address issues such as fertility, pregnancy outcome, desire for children, and childcare assistance, including permanent adoption. Through case vignettes they illustrate the impact of compartmentalized services on women, their children, and their families.

The book concludes with a chapter by Jill M Goldstein and Richard RJ Lewine that summarizes the information presented in the rest of the book and links the biology differences to the symptomatic differences gleaned from recent evidence. It identifies questions regarding sex differences in schizophrenia that require further study, as well as methodological issues to consider in such studies. Interestingly, and in contrast to the rest of the book, this chapter does not mention the need for improved awareness of the illness's impact on people's lives and the need for more integrated treatment. These issues should also be incorporated into future clinical studies.

The book is prefaced with a statement of intent to increase mental health workers' awareness of issues pertinent to women with schizophrenia. I believe that the

biopsychosocial overview provided in this clearly written but concise text successfully does this. I would recommend it to any mental health worker.

Child Psychiatry

Diagnostic Assessment in Child and Adolescent Psychopathology. David Shaffer, Christopher P Lucas, John E Richters, editors. New York: Guilford Press; 1999. 398 p. USD45.00.

Review by

Zillah Parker, MB, BS, FRCPC
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"This volume is greatly to be welcomed There is no better account of the 'tools of the trade' that may be used in both clinical research and clinical practice."

These words from the Foreword by Professor Sir Michael Rutter, are a fitting tribute to this book, which will take a rightful place on the shelf of both clinician and researcher. Arranged in 4 parts, the text is well laid out, with clear headings and tables that summarize specific sections. Each chapter is separately and richly referenced. At the graduate level, it could well serve as the core text for psychology and related sciences in a course teaching the fundamental basic science and concepts needed for diagnostic instrument design and the measurement of psychopathology. It explores the particular advantages and disadvantages of various approaches or tests and lays the groundwork for understanding measurable symptomatology and assessing response to treatment. It will be well used and more than repay its cost.

The book concentrates on the DSM-IV categories that form the basis for diagnosis in childhood and adolescence. It does not move into the less well-validated territory and scales used to assess suicide risk, self-esteem, and family relations.

In part 1, "Measures for Assessing General Psychopathology," discussion of respondent-based interviews centres around the Diagnostic Interview Schedule for Children (DISC) and its related instruments, as well as its applications, reliability, validity, and sensitivity. Sources of error are described. Several structured interviews for which psychometric data are available can be used as the basis for diagnosis when they are consistently administered, when terms are well-defined, and when the training of interviewers is standardized. Questions to be considered in selecting a particular interview schedule are outlined and pitfalls described. Various general behaviour rating scales are compared and their applications summarized. They cover a wide variety of behaviours, personality characteristics, and areas of competency, in contrast to the more specific symptom-related diagnoses.

Part 2, "Measures for Assessing Specific Syndromes," focuses on rating scales for the main categories of diagnosis in children and adolescents. The background discussion of disruptive behaviour disorders defines the field to some extent and discusses the role of observation and sources of bias and distortion. The advantages and disadvantages of rating scales are listed, and the benefit of broad vs narrow scales and the effect of ethnicity and culture are outlined. The strengths and limitations of specific scales are reviewed, with helpful notes and comments and a source list for additional information on the included scales. Rating scales for anxiety and mood disorders are discussed and the challenge of identifying and quantifying symptoms and variables taken further than in previous chapters. Selected specific self-rating scales are described and summarized and their role in treatment response and assessment of related constructs evaluated. Pervasive developmental disorders, autistic, and nonautistic syndromes are included among the communication disorders. In clinical practice, a wide variety of skill deficits are seen to have a major impact on language, academic function, relationships, and behaviour; a corresponding range of assessment tools can be useful.

Part 3, "Special Aspects of Assessing Psychiatric Disorders," a most interesting section, covers other approaches to assessing psychopathology and research design. Functional impairment is required for diagnosis with several widely used scales. They all demand some subjective assessment for labelling severity, but they are supported with behavioural examples and strive to define those children and adolescents in need of mental health services—to distinguish a "case" from a "non-case." A subject must have both symptom and impairment to be counted. Family history can provide useful information in retrospective studies or identify a cohort at risk. The application of family history to research design can assist in case selection and in developing control groups. Collecting this information with reliability is influenced by the method, by chance, by cohort, and by life experience. The limitations of retrospective data, the role of memory, motivation for giving or withholding information, and the accuracy of recall are discussed. The last 2 chapters of this section discuss adapting instruments for different ethnic groups and the influence of culture, race, and socioeconomic status on assessment.

The final section, "Biological Measures," provides an up-to-date review of neurophysiology and the basis for assessment in the laboratory. It presents some of the major advances in our knowledge of control systems and neurotransmitters and summarizes information for the reader. There are clear figures illustrating both the control and feedback loops in growth hormone secretion (a model for other neuroendocrine systems) and also the platelet model of 5-HT stimulation. Neurotransmitters are listed and well referenced. This foundation leads to a discussion of application in assessment.

In all, this text comprehensively reviews the best research on assessment tools

and their application to research design and clinical practice. It is well-organized and written by experts in the field. The reference lists are exceptional.

General Psychiatry

Psychological Debriefing: Theory, Practice and Evidence

Beverley Raphael, John P Wilson, editors. Cambridge (UK): Cambridge Press; 2000. 360 p. USD59.95

Review by

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As a relative lay person in the field of psychological debriefing, on reading this edited volume I learned that I had been labouring under an apparently common misapprehension—that psychological debriefing is generally helpful after a wide range of potentially traumatic incidents. Since its first use among US World War II military personnel, the philosophy that reviewing potentially traumatic events proximal to their occurrence and facilitating cognitive restructuring, emotional release, and peer support will promote postincident adaptation and functioning has gained acceptance among participants and practitioners alike. The strength of this book is that it challenges this acceptance using research-based evidence.

The editors have gathered many of the important researchers in the field to present the evidence for and against the utility of psychological debriefing in various circumstances. Raphael is a well-published researcher in many areas of trauma and adaptation to trauma. She and Wilson have collaborated in the past, with Wilson bringing psychotherapeutic perspective to the efforts.

By the end of the book, I felt well informed about the controversies and debates in the field, but the knowledge was not gained easily. The book is physically well designed, with a large, floppy, soft cover that falls open easily and stays at a desired page. The font, however, although reasonably free of typographical errors, is too small to be read comfortably. The 25 chapters are each authored by different researchers in the field, and it seems that they were not given clear instructions regarding format or objectives: there is an annoying amount of repeated historical material and references to various studies, while figures and tables are used inconsistently. The editors have attempted to increase cohesiveness by introducing each chapter with an editorial. These editorials give valuable perspective; without them the unevenness and lack of continuity among the chapters would be difficult to cope with.

The book is divided into 4 sections, but the boundaries between sections are often unclear. The first section "Conceptual Framework of Debriefing," is particularly weak. Whether a debriefing neophyte or experienced in the field, one is generally looking for clear definitions of terms and scope at the beginning of a book. The first 4 chapters review some basic concepts, such as types of debriefing, its historical development, and its differentiation from related interventions. Although the chapters in this section are well written, the authors primarily review their own work at the expense of thoroughness and continuity. Important questions are not presented systematically. Much of what could be considered critical introductory material is gleaned piecemeal from later chapters.

The second section's title, "Debriefing Models, Research and Practice," implies a summary of the different debriefing models currently in use, and the initial chapter, by Mitchell and Everly, succinctly summarizes their popular model, Critical Incident Stress Debriefing (CISD). Other referred-to models, however (for example, those of Dyregrov and Armstrong), are not given equal space. Instead of systematically reviewing the various debriefing models and the research evidence supporting or refuting each, the chapters are divided by type of incident (for example, body recovery and road trauma), with varying emphasis and quality.

Section 3, "Adaptations of Debriefing Models," is strong due to the authors clearly apparent clinical sensitivity; they understand the complexity of the human response to critical events and the necessity of placing those responses in a temporal and social context. It is, however, unclear why some chapters (for example, the chapter on debriefing after traumatic childbirth) are in this section, whereas others with apparently similar focus (for example, the chapter on debriefing after motor vehicle accidents) are in section 2. The authors raise many interesting questions about the usefulness of debriefing in situations other than those including emergency services personnel, for which setting it was originally designed.

In the final section, "Debriefing Overview and Future Directions," the political issues of the debriefing debate are clarified, and this context helps to put the disparate views of previous chapters in perspective. I found myself wishing that some of the messages had been presented earlier in the book—for example, Stuhlmiller and Dunning's assertion that psychological debriefing is improperly founded in assumptions of pathology rather than resilience and Silove's clarification that debriefing is an inappropriate response to disasters in developing countries where the trauma is extended and multiple.

In conclusion, this book is too short on "practice" to be recommended to a reader wanting to learn how to do debriefing. Given the political nature of the content and the lack of well-designed research upon which to comment, this volume will quickly become dated. For readers motivated to understand the state of the field and willing to wade through the inconsistent and discontinuous chapters, the price is unlikely to provide a barrier.

History of Psychiatry

The Father Of Canadian Psychiatry, Joseph Workman.

Christine IM Johnston. Victoria (BC): Ogden Press; 2000. 198 p.

Review by

Peter Faux, MD, FRCP
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Christine Johnston has written the first, and definitive, biography of Dr Joseph Workman, a 19th-century alienist who established psychiatry in Canada as a medical profession within the Provincial Lunatic Asylum in Toronto (and also identified typhoid in its basement sewage cesspool). Is Workman the father of Canadian Psychiatry, as Johnston has proclaimed? After reading her history, I would have to agree that it would be hard to find his equal.

Johnston's work conforms to the tenets of Henry Ellenberger (1): her history of Workman, his family, and his times is notable for its lack of editorializing, and, like Ellenberger's own writing, it will stand as a model for budding historians.

The film noir, *Cause For Alarm* (2), contains the following line: "Men wrapped up in themselves come in small packages." That statement reflects everything that Joseph Workman was not. As described by Johnston, he was a Renaissance man: an Irish immigrant, he graduated in medicine in Upper Canada in 1837 and then proceeded to found a successful hardware business in 1840. He also helped to found the Unitarian Congregation in Toronto and the Board of Trade in 1845, the Canadian Medical Association in 1867, and the Ontario Medical Association in 1881. He was the first chairman of the Board of Education in Toronto in 1850, was appointed to the Provincial Lunatic Asylum in Toronto in 1853, advocated and ensured the completion of the Toronto Asylum in 1870, and successfully battled George Brown, editor of *The Globe*.

As a psychiatric humanitarian he reduced restraint, did away with cupping and bleeding, and conceptualized mental illness as somatic. Throughout his life he maintained an unblemished moral code of the highest standard. He was a moral psychiatrist and a moral human being who achieved Ericksonian altruism.

This biography is well worth reading and is a bargain to boot. Whether Workman is indeed the "father" of Canadian psychiatry will be debated. What will never be questioned is that his life was exemplary and his example worth following to this day.

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LETTERS TO THE EDITOR

Moving From the Clinic to the Community: The Alone Mothers Together Program

Dear Editor:

Health promotion among lone-mother families should be a priority because they constitute a large segment of the population, experience extraordinarily high poverty rates, and are at elevated risk of depression and other mental health problems. Moreover, their health care use is suboptimal. These considerations stimulated our move from clinic-based support and education groups for lone mothers to community-based groups. The pilot study of community-based support and education groups for poor lone mothers of preschoolers focused on identifying and enlisting lone mothers, retaining them in the groups, completing evaluations, and designing acceptable evaluation tools.

In the planning period, we identified the need to collaborate with key community agencies, to get broad community exposure, and to provide a high-quality, structured children's program. More intensive advertising (for example, flyers and information sessions) was done in areas where groups were organized. Interested mothers registered by telephone or in person at information sessions. Enlistment was aided by providing an Ontario Works credit for participation, as well as snacks, transportation, and a clothing exchange. To sustain group involvement, mothers received telephone reminders and transportation assistance.

To facilitate evaluation, we provided flexible times, sites, and compensation for questionnaire completion. The evaluations indicated that measures were acceptable in terms of length and language. Self-report (1–5) and observer-rated (6) measures assessed maternal well-being and parenting. Evaluations were carried out both before and after the group sessions. During the pilot study, we organized 2 group interventions, each 8 weeks long. Community-based support and education groups run within subsidized housing projects met weekly for 1½ hours in sessions led by 2 experienced group leaders. Recurrent discussion themes included issues common to lone mothers—the experiences of poverty and social isolation—and child-rearing issues (for example, concerns about child development and behaviour management). A parallel children's program focusing on social, language, and motor skills was provided to the preschoolers. We developed a manual for each group.

Advertising efforts attracted 20 lone mothers who expressed interest, of whom 10 agreed to participate in the intervention. Eight of the 10 completed more than one-half the group sessions, and 6 of the 10 completed the postgroup evaluation.

We successfully achieved our pilot goals (that is, we identified and enlisted lone mothers into the groups, we maintained a satisfactory participation level, and we developed acceptable evaluation tools). This provided the basis for proceeding to a randomized controlled trial evaluating the effectiveness of these groups in the community. Despite strong expressed community support, recruiting mothers was an intensive process that attracted only small numbers. Readiness to change and to engage in treatment activities appears to be much lower in the community, compared with clinic settings, where mothers have been mobilized to ask for help with specific problems. Accordingly, those developing community-based programs must pay close attention to the tradeoffs between the intervention program's acceptability and attractiveness and the burden of participation defined by the intervention's intensity, duration, and structure.

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Citalopram Overdose, Serotonin Toxicity, or Neuroleptic Malignant Syndrome?

Dear Editor

We would like to comment on the letter "Neuroleptic Malignant Syndrome Due to Citalopram Overdose" (1): we question the authors' assertion that they present the first case of neuroleptic malignant syndrome (NMS) due to citalopram use. The reported case contains a few features that are possibly consistent with NMS, but it occurred after an overdose of an agent that is much more likely to cause serotonin toxicity. Serotonin toxicity is far more likely to occur with selective serotonin reuptake inhibitors (SSRIs) than is NMS, and we believe that this is what has been described.

The diagnostic criteria for NMS usually include 4 principal features: autonomic lability, hyperthermia (pyrexia) without other cause, extrapyramidal syndrome (commonly cog-wheel or lead pipe rigidity), and encephalopathy (2). In the patient described, there was no evidence provided of autonomic lability—only the initial heart rate and blood pressure were reported. The patient had a low-grade temperature on admission, but no other temperature measurements were reported. Only mild rigidity (of unclear type) of the limbs and abdominal musculature was described. Finally, the encephalopathy of NMS cannot be determined in a comatose patient (coma of itself is very unusual in cases of NMS). Thus, the patient had none of the major features of NMS at the time of the diagnosis. A raised creatine kinase (CK) is not diagnostic of NMS (2) and, in this patient, could have resulted from numerous other causes that were not excluded.

The patient was described as regaining consciousness 6 hours after bromocriptine administration. No details are provided, however, concerning sedation given or paralysis used while the patient was ventilated. Regaining consciousness was more likely to be part of the clinical course or the result of any sedation wearing off. Administering bromocriptine in cases of NMS will lead to resolution of muscle rigidity, autonomic lability, and hyperthermia; it will not particularly increase the level of consciousness alone.

We believe that the condition described in this case is more likely to have been serotonin toxicity, which is a recognised complication of SSRI overdose (3). Usually referred to as the serotonin syndrome, the condition is better described as a spectrum of toxicity (4). Although Sternbach's criteria are the most commonly cited for diagnosis (5), serotonin toxicity is more clearly characterized by a triad of clinical features: autonomic features, neuromuscular changes, and altered mental status (3).

Although there are similarities to NMS, serotonin toxicity is a distinct condition (6,7). NMS is an idiosyncratic reaction to therapeutic dosages of neuroleptic agents, while serotonin syndrome is a toxic reaction due to overstimulation of 5-HT_{2a} receptors in the central nervous system (6,8). Distinguishing features include bradykinesia and lead pipe rigidity in NMS, compared with hyperkinesia and clonus in serotonin toxicity, and autonomic instability in NMS, compared with presence of a serotonergic agent in serotonin toxicity (6,7). In the case described, there is evidence of serotonin toxicity; however, there are insufficient clinical details to confirm the diagnosis.

Over the last 2 years, there have been 46 citalopram overdose presentations to our unit. There was moderate-to-severe serotonin toxicity in 7 cases, and in 4 of these, citalopram was the only drug ingested. All 7 met Sternbach's criteria (5). The dosage ingested by the 4 patients using citalopram alone ranged from 280 mg to 3000 mg daily. There were no cases of NMS. This suggests that serotonin toxicity is not uncommon with citalopram overdose. There is 1 report in the literature of serotonin syndrome caused by an infusion of 20 mg of citalopram (9).

Because the described clinical features did not satisfy the diagnostic features of NMS (2), other differential diagnoses should have been considered by the authors—in particular serotonin toxicity. It is our contention that the authors need to make an unusually strong argument for the case of citalopram-induced NMS: the patient they describe had a citalopram overdose with very few features of NMS. We believe this is not a case of NMS due to citalopram overdose; it is more likely to be the expected serotonin toxicity.

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Reply: Citalopram Overdose, Serotonin Toxicity, or Neuroleptic Malignant Syndrome?

Dear Editor:

We are grateful to your correspondent for opening the debate on our letter.

Neuroleptic malignant syndrome (NMS)—like the encephalopathy that develops in association with the use of antidepressants—indicates that both it and serotonin syndrome are spectrum disorders induced by drugs with both antidopaminergic and serotonergic effects (1). Medications that affect brain dopamine and serotonin levels are occasionally associated with neurotoxic behavioural and autonomic syndromes, variously described as NMS and toxic serotonin syndromes (TSS). Based on the drugs' presumed brain mechanisms of action, different corrective interventions have been recommended. Moreover, Fink has claimed that NMS and TSS are not specific syndromes but, rather, examples of a nonspecific generalized neurotoxic syndrome and that they are

subtypes of catatonia (2). For these reasons, NMS and TSS may be confused—or they may be presentations of different aspects of the same etiologic cause (dopamine–serotonin imbalance).

Although NMS is described as having 4 classic signs, no agreed-upon criteria exist for the diagnosis of the syndrome in terms of severity or combination of these signs, and milder or incomplete varieties have been detected and included with the full-blown cases (3). In our patient, there was no autonomic lability, but there was coma, subfebrile fever, and rigidity on admission. In NMS, mental status changes, coma, and catatonia are common (4). Although creatine kinase (CK) is not a specific indicator of NMS, it may be important for the early detection of the syndrome (3). Muscle isoenzyme of CK may be raised by intramuscular injections, hyperactivity, and catatonia (5); it may also be raised in medically ill patients taking neuroleptics (6). In our case, there were no such conditions.

No sedation or paralysis agents were used while the patient was ventilated. Therefore, consciousness was not regained as the result of any sedation wearing off. Otherwise, regaining of consciousness was not the part of the clinical course, it was a secondary event to bromocriptine administration. We observed no change during a 7-day period of supportive treatment, but the clinical course improved completely within 24 hours of bromocriptine administration. In one review, it has been stated that, when compared with supportive measures alone, bromocriptine in particular shortened the time to the resolution of NMS symptoms (7). Since there was neither hyperkinesia nor clonus in our case, we did not suspect serotonin toxicity.

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Lycanthropy: 2 Case Reports

Dear Editor:

According to Keck and others (3), lycanthropy can be diagnosed when a patient reports in a lucid moment, or retrospectively, feeling, or having felt, sensations of being an animal. Similarly, it can be diagnosed when a patient displays animal-like behaviour (for example, howling or crawling). It does not form a separate syndrome but is a symptom of different psychiatric diseases (2). We report 2 cases of patients with schizophrenia who felt they had been transformed into a frog and a bee, respectively.

Case Report 1

A 34-year old woman suffering from schizophrenia came to the emergency department. At first, she was mutistic; later she seemed agitated and tense. Suddenly, she started moving like a frog, jumping around, making frog-like noises, and showing her tongue as though intending to catch a fly. We found out that she had taken part in a workshop about fairy tales prior to becoming symptomatic. An organic cause was excluded, and no drug intake was found.

Case report 2

A 24-year-old woman suffering from schizophrenia reported a strange feeling that could not be properly described, together with the feeling that she was becoming a bee and getting smaller and smaller. She also felt a burning sensation in her thighs. She attributed her metamorphosis to her being stung by bees as a child and the "splashes of bee" that had touched her. In this case, it is interesting to note that she described herself as caring for her mother like a "working bee" and that she could not really develop autonomy. No organic causes could be found, and drug screening tested negative.

Discussion

To date, the metamorphosis into a frog or a bee has not been described in the medical literature. Both patients suffered from schizophrenia, and the lycanthropy was accompanied by other psychotic symptoms. Psychodynamically, lycanthropy could be an attempt to delegate affects to the animal. Lycanthropy in our cultural context seems bizarre and strange, appears suddenly, and does not seem to be understood rationally. In his discussion of countertransference phenomena induced by lycanthropy, Knoll points out that its strange and often threatening aspect may lead therapists to neglect it (3). This might explain why the phenomenon is rarely described in psychiatric literature. Focusing on the patient's subconscious choice of animal species may hint at the patient's subconscious conflicts and might be helpful in psychotherapy of the mental disease.

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Re: Weight Change With Antipsychotic Use

Dear Editor:

Weight increase with the use of antipsychotics, especially novel antipsychotics, has been of significant concern to patients suffering from psychotic disorders. There are several psychosocial issues associated with weight gain (for example, issues of self-esteem, discrimination, and medical morbidity). Studies and clinical practice involving antipsychotic use consistently indicate weight gain as a potentially serious side effect.

It has been stated that weight gain and loss may not be dosage-related. In my clinical observation on 2 different occasions, however, dosage appeared to be related to weight loss after initial weight gain. I present here the cases of 2 patients who lost weight, following initial gain.

Case Report 1

A 25-year-old man suffering from schizophrenia was prescribed olanzapine 20 mg daily (monotherapy). His weight was recorded at 100 kg. He was clinically stable. The dosage was reduced to 17.5 mg daily, and then to 15 mg daily, with no rebound increase in psychosis. His recorded weight showed a reduction of 7 kg, accompanied by more self-confidence in the patient. There was no other change in his medication regimen, diet, medical condition, lab values, or daily routine.

Case Report 2

A 37-year-old lady suffering from schizoaffective disorder with manic and psychotic symptoms had a trial of several mood stabilizers, typical antipsychotics, long-acting antipsychotics, and clozapine, with multiple subsyndromal to full-blown symptoms and frequent hospitalisations. She was treated with 75 mg of topiramate and was started on olanzapine with dose titration and final increase to 20 mg daily. She experienced a weight gain of 11 kg in 3 months. This combination of mood stabilizers and olanzapine led to significant improvement in her mental state. Initially, it was a daily struggle keeping her on olanzapine, because she was extremely weight-conscious. With improvement in her clinical condition, olanzapine dosage was decreased to 17.5 mg daily, and then to 15 mg daily, with weight reduction of 3 kg. She was much more accepting of olanzapine and was discharged.

Based on these observations, it is not possible to determine the mechanism of action leading to weight loss. Olanzapine dosage reduction—wherever possible at the earliest opportunity—may, however, be a useful strategy to reduce weight following initial gain.

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Misleading Drug Advertising

Dear Editor:

It must be evident to all psychiatrists of my vintage (over age 65 years) that pharmaceutical company advertising has changed remarkably in the last 40 years. In the 1960s, battling the hegemony of psychodynamically oriented psychiatry, the drug companies timidly promoted their products (sometimes, even the neuroleptics) as adjuncts to psychotherapy (1).

Now, we seem to be in an era wherein the aim of drug advertising is to devalue a competitor's product in the hope of casting a favourable light on your own. For example, we have the altruistic folks at the Lilly Company informing us in a 2-page advertisement (2) of the dangers of prolonging the QTc interval, possibly in anticipation of ziprasidone's introduction by a competitor. We also have the thoughtful people at Janssen telling us about the high incidence and dangers of type 2 diabetes, an increased risk for those treated with olanzapine (3).

There is nothing wrong with receiving factual information. Every clinician knows that the use of any drug for a particular patient requires the exercise of sound judgement and a careful balancing of advantages against disadvantages. When drug companies try to deceive us, however, they lose credibility; unfortunately, their spokespersons may also lose credibility. The following 2 examples are illustrative.

The March 2001 issue of *Physician Perspective*, a newsletter sent through the mail by Eli Lilly Canada Inc, contained the "viewpoint" of Canadian physician, Joel Raskin, entitled "The New Atypicals and Abnormalities in Glucose Metabolism: Real or Exaggerated Risk?" Dr Raskin, who just happens to be the vice-president of clinical research for Lilly, states: "Risperidone and ziprasidone were designed to behave like haloperidol, but without giving rise to extrapyramidal symptoms." Anyone aware of the pattern of receptor blockade of these 2 drugs, and that of haloperidol (and others, for example see [4] and [5]), will find this statement grossly misleading. A recent article by Kapur and Seeman (6) is also relevant.

A second example: A friend of mine recently picked up a pamphlet in her family doctor's office. *Anxiety—It's Not Just In Your Head* was authored by Michael Evans, MD, and Richard Swinson, MD. Its publication was "supported by an educational grant from Wyeth-Ayerst Canada Inc," the makers of Effexor (venlafaxine). The pamphlet is structured as a series of questions the prospective patient may have. Question 13 reads as follows: "Are the medications addictive? Antidepressants are not addictive in the same way that cigarettes or benzodiazepines are." To suggest that the addictive potential of benzodiazepines is comparable with that of

nicotine (cigarettes) is deceitful and serves only to frighten unnecessarily those patients who have been helped by benzodiazepines.

Can drug companies not do better than this? Psychiatrists are not stupid.

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