

# Should cognitive deficit be a diagnostic criterion for schizophrenia?

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This review examines the question of whether cognitive deficits in schizophrenia are sufficiently reliable, stable and specific to warrant inclusion in the diagnostic criteria for schizophrenia. The literature provides evidence that cognitive deficits are highly prevalent and fairly marked in adult patients with schizophrenia. Similar deficits have been found in children and adolescents with schizophrenia, and in children before they exhibit the signs and symptoms of schizophrenia. These deficits may in fact be central to the pathophysiology underlying the development of overt psychosis in schizophrenia. The deficits appear to be relatively stable across the course of the illness. They are generally more severe in schizophrenia than in affective disorders and may have a relatively specific pattern in schizophrenia. It is concluded that the evidence that cognitive deficits are a core feature of schizophrenia is sufficiently compelling to warrant inclusion of these deficits in the diagnostic criteria for schizophrenia, at least as a nonessential criterion.

Cette étude a cherché à savoir si les déficits cognitifs de la schizophrénie sont suffisamment fiables, stables et spécifiques pour justifier de les inclure dans les critères de diagnostic de cette maladie. La littérature scientifique présente des données probantes indiquant que les déficits cognitifs sont très prévalents et assez marqués chez les patients adultes atteints de schizophrénie. On a constaté des déficits semblables chez les enfants et les adolescents atteints de schizophrénie, ainsi que chez les enfants avant l'apparition des signes et des symptômes de la schizophrénie. Ces déficits peuvent en fait jouer un rôle central dans la pathophysiologie sous-tendant l'apparition de la psychose apparente dans les cas de schizophrénie. Les déficits semblent relativement stables pendant toute l'évolution de la maladie. Ils sont en général plus prévalents dans les cas de schizophrénie que dans les cas de troubles affectifs et peuvent se présenter sous une forme relativement spécifique dans la schizophrénie. On conclut que les données probantes indiquant que les déficits cognitifs constituent une caractéristique fondamentale de la schizophrénie sont suffisamment convaincantes pour justifier d'inclure ces déficits dans les critères de diagnostic de la schizophrénie, au moins comme critère non essentiel.

## Introduction

The presence of cognitive impairment has never been included in any of the major diagnostic criteria systems applied to schizophrenia. Yet cognitive deficits have in the last decade become increasingly accepted as a remarkably robust and core characteristic of the disorder.

Tsuang et al<sup>1,2</sup> have recently suggested that the diagnostic criteria for schizophrenia be reconsidered, because there is an overreliance on psychotic symptoms, which are not specific to schizophrenia. They propose including a new category of "schizotaxia" in the next edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM).<sup>3</sup> They define this term as a state of

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vulnerability or predisposition to develop schizophrenia and propose that DSM criteria for such a category incorporate biologic and neuropsychologic abnormalities. They argue that such abnormalities may be more “proximal” to the disorder’s cause and pathophysiology than are the “distal” psychotic signs and symptoms presently used to make a diagnosis.

In this paper, I will review the evidence for the centrality and specificity of neuropsychologic or cognitive deficits to schizophrenia. I will argue that incorporating these deficits into the diagnostic criteria for the disorder itself (as well as possibly using them to identify vulnerable pre-psychotic children and adolescents) is important for both clinical and educational reasons. Finally, I will suggest a conservative yet flexible way of incorporating cognitive deficits into the existing DSM criteria for schizophrenia.

### Cognitive deficits in adults with schizophrenia

It has been well established that most patients with schizophrenia have cognitive deficits and that these deficits account for much of the impairment in functioning of these patients.<sup>4-8</sup> A large meta-analysis by Heinrichs and Zakzanis<sup>9</sup> of 204 studies of cognitive deficits in schizophrenia found that there are both global and relatively specific cognitive deficits in patients with schizophrenia compared with healthy controls. Altogether, 61%–78% of patients scored below the normal median on all the tests of cognitive function. The largest mean effect sizes that were also supported by adequate numbers of studies were recorded for global verbal memory, performance and full-scale intelligence quotient (IQ), Continuous Performance Test scores (a measure of attention) and word fluency. The authors note that the selectivity of cognitive deficit (i.e., disproportionately greater deficit in the aforementioned domains of cognitive function) occurs against a background of very general impairment, with even the less sensitive tasks yielding effect sizes of 0.5–1.0 on average, and almost 75% of patients scored below the median on global intelligence (Wechsler Adult Intelligence Scale–Revised [WAIS-R] full-scale IQ).

Although informative and persuasive, the limitations of such a meta-analysis should be borne in mind, such as the huge variability in the number of samples from which the data are derived for particular tests and clinical differences in the samples themselves.

Heinrichs and Zakzanis<sup>9</sup> note that a significant mi-

nority of patients with schizophrenia are neuropsychologically healthy, but they postulate that many patients may perform below their premorbid cognitive potential even if they are technically average by normative performance standards. That point had been illustrated in a study of monozygotic twins discordant for schizophrenia, in which the twins with schizophrenia generally performed worse on neuropsychologic measures than their healthy twins, even if their scores fell within the normative range.<sup>10</sup> The fact that cognitive deficit may be relative to the individual’s own premorbid potential may explain the overlap between patients with schizophrenia and healthy controls on cognitive scores. More research is needed, including longitudinal studies, to better understand this phenomenon.

Thus, cognitive deficits are significant and highly prevalent in adults with schizophrenia and may show a prototypical pattern in schizophrenia; however, neuropsychologic testing cannot be expected to completely differentiate patients from healthy controls.

### Cognitive deficits in children and adolescents with schizophrenia

In establishing whether cognitive impairment is a core characteristic of schizophrenia, it is relevant to know whether the impairment can also be identified in younger patients in the early stages of the illness and possibly even in children before they exhibit the signs and symptoms of schizophrenia. Cognitive impairment has in fact been demonstrated in children and adolescents with schizophrenia.<sup>11-19</sup> The cognitive deficits identified by these studies are broadly the same as those found in adult patients.

The domains of cognitive functioning generally found to be impaired in schizophrenia, in both the child and adolescent and the adult literature, are the following:

1. generalized deficits and lowered full-scale IQ (and reduced capacity for information processing, according to Asarnow’s hypothesis, which is discussed later<sup>12,20</sup>);
2. disproportionately greater deficits (over and above the generalized deficits) in:
  - attention and sustained attention/vigilance,
  - memory (global measures — no consensus in the literature on specific types of memory functions),
  - executive functioning (especially working memory and cognitive set-shifting ability).

Salient findings from the literature that help to characterize the deficit in children and adolescents with schizophrenia will be highlighted below.

The question of whether the deficits are a state or trait phenomenon is addressed in the study by Banaschewski et al.<sup>19</sup> These investigators found no correlation between cognitive impairment and any type of psychiatric symptom, for example, positive or negative symptoms, in adolescents with schizophrenia. Adult studies have also observed cognitive deficits to be poorly correlated with psychiatric symptoms in schizophrenia.<sup>8</sup> In the study by Banaschewski et al,<sup>19</sup> premorbid disturbances (language or motor developmental disorders, or both), which were more common in cases of earlier onset, were found to be risk factors for subsequent cognitive impairments, though it should be noted that the premorbid disturbances were assessed retrospectively. These data lend further support for the hypothesis that cognitive deficits are a core and stable characteristic (i.e., trait) of schizophrenia, and that they are independent of psychotic symptoms rather than resulting from them. As a caveat, it needs to be acknowledged that in very acute, profoundly psychotic states, more severe cognitive impairment is bound to occur transiently as a result of the level of disorganization of thought process. This self-evident point is often not made in the research literature, because studies tend to exclude very acutely ill patients. The sample in the study by Banaschewski et al comprised mainly chronically ill patients undergoing rehabilitation.

The relation between the deficits and age or progression of illness is addressed in a study by Bedwell et al.<sup>17</sup> Their study demonstrated that, whereas age-corrected IQ scores declined significantly after the onset of psychosis in patients with childhood-onset schizophrenia compared with healthy controls, raw scores did not. They thus concluded that the postpsychotic decline in IQ represented an inability to acquire new information rather than the loss of previous abilities or knowledge, which would have suggested a dementia. This is consistent with the hypothesis that cognitive impairment is present premorbidly in schizophrenia and that it remains relatively stable as the patient grows older and the illness progresses.

The effects of age on cognition in children and adolescents can be understood in part from a neurodevelopmental perspective; that is, cognitive functions and their neural substrates are in a process of active, ongoing development throughout normal childhood and

adolescence. Casey et al<sup>21</sup> are conducting perhaps the first systematic studies of normative cognition at various ages of childhood and adolescence in relation to structural and functional brain development, using magnetic resonance imaging. They have found, for example, that memory and attention are in a state of continued development throughout childhood and adolescence, associated with prolonged physiologic development and organization of the prefrontal cortex, in which synaptic pruning is thought to play an important role. Thus, what may appear to be a deterioration in cognitive functioning as a child with schizophrenia grows older may in fact be the failure of normal cognitive maturation.

Cognitive functioning in children has been most thoroughly characterized by Robert Asarnow and colleagues in a series of studies.<sup>12</sup> Asarnow et al conclude from their studies that children with schizophrenia perform poorly on tasks that tap executive functions or that place extensive demands on attention and short-term memory, whereas their rote language tasks and simple perceptual functions are not impaired. Thus, they suggest that these children essentially have limitations to their ability to engage in effortful cognitive processing or have impairments to their working memory. Working memory, that is, the manipulation of information held in immediate or short-term memory, is a major aspect of processing capacity. Children with schizophrenia may have a limited processing capacity and/or faulty allocation of processing capacity, namely, they may be limited in how much information processing they can carry out in a given time. Automatized cognitive processes, on the other hand, make minimal demands on processing capacity. Hence, once cognitive processes become automatized, such as language when it is fully acquired, then performance on rote language tasks would be expected to be normal.<sup>12,20</sup>

Some deficits have also been found to be relatively specific to schizophrenia compared with attention-deficit disorder (ADD) and autism. Øie et al<sup>15,16</sup> found children with schizophrenia to be more impaired than children with ADD on tests of working memory with focus on abstraction–flexibility and on measures of visual memory and motor function. Children with ADD, in turn, were relatively more impaired on measures of attention and working memory tests with focus on distractibility, and on measures of verbal memory and learning. Asarnow et al<sup>12</sup> found that increasing the amount of information to be processed had a greater

impairing effect on the rate of visual information processing (span of apprehension task) in children with schizophrenia than in children with ADD. They also found that children with schizophrenia had greater distractibility than nonretarded children with autism.<sup>12</sup>

The literature concerning children and adolescents thus supports the hypothesis that young patients in the early stages of schizophrenia have similar cognitive impairments to those found in adults and that cognitive impairment is a core and stable trait of the disorder. Limitations in information processing may underlie the disproportionate deficits seen in effortful, controlled cognition and may result in difficulties acquiring new information and skills. More research is needed to evaluate the extent to which the pattern of deficits is specific to schizophrenia in children and adolescents, relative to other disorders.

### **Cognitive deficits in children before they exhibit the signs and symptoms of schizophrenia and their relation to the onset of psychosis**

Premorbid cognitive functioning has been studied in children before they exhibit the signs and symptoms of schizophrenia using a variety of study designs, including retrospective case-control studies, prospective follow-up of high-risk individuals and population-based cohort studies. These studies have found that certain cognitive deficits in schizophrenia, particularly attentional deficits, are often detectable before the onset of illness.<sup>22-28</sup>

Asarnow,<sup>20</sup> in reviewing his group's studies of the neurobehavioural antecedents of schizophrenia, notes that the vast majority of children with schizophrenia had significant neurodevelopmental delays beginning early in life. These include gross deficits in early language development, which were found in almost 80% of children who went on to develop schizophrenia. He makes the point, however, that the language delays must be transitory, because it has been established that language skills are in fact remarkably well preserved by the time the clinical features of schizophrenia are established. Asarnow suggests that the problem is essentially a delay in learning a skill, rather than a static neuropsychologic deficit. He suggests that children who develop schizophrenia are most likely to be delayed in acquiring skills that are at the cusp of development, namely, emerging skills that are just coming online developmentally. He hypothesizes that this difficulty is

attributable to these individuals having insufficient information-processing capacity available to handle higher processing demands, resulting in information overload and cognitive impairment.

In keeping with these findings, Keshavan and Hogarty<sup>29</sup> hypothesize that deficits in *social* cognition (related to the construct of emotional intelligence as formulated by Mayer et al<sup>30</sup> and popularized by Goleman<sup>31</sup>) are central to the pathophysiology of schizophrenia. They posit that adolescents who are predisposed to schizophrenia may use prepubertal (concrete, as opposed to abstract) processing styles that are insufficient for the more complex, abstract requirements of adult cognition, thus not getting the gist of the subtleties and nuances of adolescent or adult social interaction. Examples of these more complex social cognition abilities that might be impaired in such individuals include empathy, perspective taking, reflection, appraisal of social contexts, planning and foresight. The psychotic illness might be triggered by the stress caused by age-appropriate psychosocial developmental challenges overwhelming a delayed social cognitive capacity in these neurodevelopmentally compromised individuals. The stress might set off a cascade of neurochemically mediated dysregulations (to which these individuals would also have been predisposed by the early developmental neuronal miswiring, according to the model proposed by Keshavan and Hogarty<sup>29</sup>) that may then manifest as the more familiar symptoms of psychosis. Integrating Keshavan's and Hogarty's model with Asarnow's model, social cognition may be regarded as that cognitive skill set that is at the cusp of development, or coming online, during adolescence, and one may hypothesize that individuals who are vulnerable to schizophrenia cannot cope with the huge information processing capacity that must be involved in acquiring these skills.

Neurocognitive impairments, reflected in the delayed acquisition of certain skills, appear many years in advance of the first onset of psychotic symptoms in individuals who later develop schizophrenia and are present even when psychotic symptoms remit. Asarnow<sup>20</sup> presents a view that is increasingly held by experts in the field, namely, that cognitive impairments antedate, and are developmental precursors of, psychotic symptoms. It is proposed that they may in fact be considered to be the "endophenotype" for schizophrenia.<sup>8,20</sup> An endophenotype is an intermediate expression of the genetic predisposition for a disease that

is expressed endogenously, in contrast to exogenous/observable, that is, behavioural, phenotypes such as psychosis. The finding of cognitive impairments in the unaffected relatives of patients with schizophrenia, even second-degree relatives,<sup>32,33</sup> lends further support to this hypothesis.

The literature concerning children and adolescents thus provides support for the centrality of cognitive deficits to the pathophysiology and essence of schizophrenia.

### **The stability of cognitive deficits in schizophrenia**

The question of whether the cognitive deficits in schizophrenia are static/constant or progressive is an important unresolved question that is of great relevance to the ongoing debate about whether schizophrenia is purely a neurodevelopmental disorder,<sup>34</sup> or whether a neurodegenerative process occurs at the onset of illness.<sup>35</sup> Moreover, if cognitive deficits are stable traits of schizophrenia rather than just transient states associated with psychosis in general, then they would be especially valuable as diagnostic criteria for the disorder. There appears to be an emerging consensus that the cognitive deficits are mostly premorbid and constant (i.e., neurodevelopmental), with a lesser and more variable degree of deterioration (i.e., a neurodegenerative component) possibly occurring in the first few years of illness, perhaps beginning in the prodrome. The influential review by Goldberg and Gold<sup>4</sup> emphasizes the neurodevelopmental view, arguing that the cognitive deficits are already present at the time of first psychotic episode in young patients and that they are approximately equivalent to the deficits seen in chronically ill patients.

Several cross-sectional studies in adults that have compared patients with schizophrenia at different stages of their illness provide supportive evidence for this argument.<sup>36,37</sup> Longitudinal follow-up studies are the best design for assessing the stability of cognitive deficits over time, for differentiating between trait and state deficits, and for minimizing selection bias (because chronically ill patients may a priori represent a more ill group than patients at earlier stages of illness). A review by Rund<sup>38</sup> of 15 studies of neuropsychologic testing in patients with schizophrenia at various stages of illness, which included follow-up of at least 1 year, concluded that the cognitive deficits are relatively stable traits, suggesting that schizophrenia is mostly a static encephalopathy.

Three of the longitudinal studies reviewed by Rund<sup>38</sup> were based on subjects with first-episode schizophrenia.<sup>39-41</sup> Three subsequent longitudinal studies of first-episode schizophrenia<sup>42-44</sup> support Rund's conclusions. Studying patients with schizophrenia at the time of their first psychotic episode is valuable, because the cognitive deficits present may be assumed to be relatively independent of the secondary effects of chronicity and medication. The first-episode studies found that the deficits mostly did not deteriorate significantly, or in some cases even improved slightly (possibly as a result of the patient being in a less severely acute state) over the follow-up period, which ranged from 1 to 5 years.<sup>39,40,42-45</sup> Longitudinal studies of cognitive deficits in younger patients, namely, adolescents with schizophrenia, would be even more helpful in confirming the stability of these deficits in this disorder, but, to this reviewer's knowledge, no such studies have been published.

Three studies<sup>46-48</sup> found that, for the most part, there was no association between cognitive performance and duration of untreated psychosis in young adult patients experiencing their first episode of schizophrenia. This finding lends further support for the stability of the deficits in schizophrenia, because it suggests that cognition does not deteriorate or degenerate as the illness progresses.

### **The relative specificity of cognitive deficits in schizophrenia**

It is important to know how specific the cognitive dysfunction in schizophrenia is, particularly relative to affective disorders, which frequently present with psychosis and may resemble schizophrenia clinically. Contrasts in the severity and pattern of cognitive impairment between patients with schizophrenia and those with affective disorders (unipolar and bipolar) have been studied in adults, but not in children and adolescents. Many studies have found that patients with schizophrenia are more cognitively impaired than patients with affective disorders.<sup>49-69</sup> However, some studies have shown no or relatively limited cognitive differences between these diagnostic groups.<sup>70-82</sup>

Goldberg,<sup>83</sup> in his review of the neurocognitive differences between schizophrenia and affective disorders (mainly bipolar disorder), concludes that the literature, on balance, provides support for the view that patients with bipolar disorder suffer less severe cognitive im-

pairments than do patients with schizophrenia. In general, studies that did find differences in cognitive performance between patients with schizophrenia and those with affective disorder found the schizophrenia group to be more impaired relative to the affective group in global intelligence (IQ) and/or some or all of the domains of attention, memory and executive functioning.<sup>53,60,62,65,67</sup> This seems to suggest a profile or pattern of cognitive impairment specific to schizophrenia relative to affective disorders. When healthy controls were included in studies, the affective group's cognitive performance was generally found to be intermediate between the group with schizophrenia and the healthy controls. This was a consistent trend even in those studies where the differences between the groups did not reach statistical significance.<sup>79,84</sup> Goldberg<sup>83</sup> notes that, whereas some studies had found the groups to be equivalent in impairment, no studies had found patients with bipolar disorder to be consistently more impaired than patients with schizophrenia.

On a cautionary note, as highlighted in the meta-analysis by Heinrichs and Zakzanis,<sup>9</sup> the question of whether the cognitive deficits in schizophrenia are generalized or specific may not yet have been definitively resolved in the literature. One cannot necessarily conclude that the deficits found in studies are qualitative rather than quantitative. Differences in difficulty and complexity levels and other psychometric properties of the neuropsychologic tests make it difficult to draw firm conclusions, and it is possible that certain abilities, or tests for these abilities, may be more sensitive to global nonspecific deficits than are others.<sup>62</sup> It should be noted as well that most of the studies were selective rather than comprehensive in the cognitive domains tested. Statistical issues such as lack of power due to inadequate sample size may also account for some differences in findings. Goldberg and Gold,<sup>4,83</sup> while arguing in favour of differences in cognitive profile between the diagnostic groups, note that there is less consistent support in the literature for such qualitative differences than for quantitative differences.

If the deficits are mainly quantitative, one cannot definitively rule out the possibility that the cognitive differences merely reflect differences in severity of illness. The different diagnostic groups could perhaps be most similar cognitively when acutely psychotic, thus demonstrating a general effect of psychosis, rather than a specific effect of diagnosis (i.e., state rather than trait), on cognitive functioning.<sup>67,85</sup> For example, Hoff et al<sup>71</sup>

and Albus et al<sup>77</sup> did not find a significant difference in cognitive function between acutely ill patients with affective disorder and those with schizophrenia.

Furthermore, the subjects with affective disorder included in some studies did not necessarily have a history of any psychotic symptoms at all. Jeste et al<sup>86</sup> argue that this is a relevant factor, because they found a difference between psychotic and nonpsychotic affective disorders, with the patients with psychotic affective disorder, about half of whom were psychotic at the time of the assessment, resembling the patients with schizophrenia with respect to cognitive profile. Jeste's group also found a similar cognitive profile for schizoaffective disorder and schizophrenia,<sup>87</sup> as did Townsend et al.<sup>88</sup> Jeste and colleagues argue that the neuropsychologic deficits may extend to the full spectrum of psychotic illness, including affective psychosis, rather than being specific to schizophrenia.<sup>87</sup>

On the other hand, in support of the specificity argument proposed by Elvevag and Goldberg,<sup>8,83</sup> other recent studies have provided further evidence that the deficits are specific to schizophrenia relative to psychotic affective disorders and that they are not dependent on the presence or severity of psychosis: Mojtabai et al,<sup>62</sup> in a large study, found consistent differences between schizophrenia and psychotic affective disorders (both bipolar and unipolar). Although the subjects with schizophrenia in this study were on average more psychotic than the subjects with affective disorder at the time of testing, the pattern of significant findings was the same when only the data for the subjects with no current psychotic symptoms were analyzed.<sup>62</sup> Seidman et al<sup>67</sup> found that patients with schizophrenia had more severe cognitive impairment than patients with chronic psychotic bipolar disorder, although they did have similar cognitive profile patterns. The differences were maintained even when IQ was controlled for, which was significantly lower in the schizophrenia group. Virtually all subjects in that study had low-to-moderate levels of psychosis at the time of testing.<sup>67</sup> The argument for cognitive impairment being more the result of psychosis than of schizophrenia is also difficult to reconcile with the fact that severity of positive symptoms has generally been found in most studies to be unrelated to cognitive performance<sup>8,85</sup> and with the stability of the deficits over time described earlier.

There are other clinical factors that could perhaps account for the discrepant results of studies, aside from the severity of psychosis. A very acute affective state

(versus trait) at the time of testing, namely, severe depression or mania, can probably be expected to affect cognitive performance. Severity of affective symptoms is not generally comparable across studies, so one cannot quantify the impact of this variable. Diagnostic uncertainty (discussed later in this paper) is another factor that may contribute to inconsistent results.

Comorbid borderline intellectual functioning, mental retardation and learning disorders would impair cognitive test performance and are, therefore, generally exclusion criteria for most studies. However, the frequent co-occurrence of these cognitive limitations may indeed be integral to the neurodevelopmental basis of schizophrenia, that is, they may in fact be part of the pathology of schizophrenia, particularly early onset schizophrenia. Thus, for example, the study cited earlier by Kumra et al,<sup>18</sup> which compared cognitive deficits in childhood-onset schizophrenia and atypical psychosis, excluded patients with an IQ below 70. A large proportion of their potential subjects (39% of 44 patients with childhood-onset schizophrenia) were excluded from the study based on this criterion. Even within the normal range of IQ, researchers need to be careful not to overcorrect for differences in general intellectual functioning by matching subjects with affective disorder or healthy controls to the subjects with schizophrenia based on IQ or educational attainment, as Seidman et al point out.<sup>67</sup> In their study, they avoided this pitfall by adjusting for parental socioeconomic status instead.

It should be noted that most studies do not control for medication effects, which is a complex and difficult task. Antipsychotics and other psychotropic medications may have variable effects on cognition. Some antipsychotics, particularly the novel or atypical antipsychotics, are said to partially improve aspects of cognition,<sup>89-91</sup> but the different cognitive measures used across studies make it hard to draw definitive conclusions about the beneficial effects of antipsychotics, and some putative positive results may really be method artifacts.<sup>92,93</sup> Many antipsychotics may in fact have a detrimental effect on cognition because of their sedating and anticholinergic properties. Motor (extrapyramidal) side effects of antipsychotics may also affect psychomotor speed, which could affect performance on some cognitive tests. Many patients studied are also on mood stabilizers, most of which are known to impair aspects of cognition, as is the case with anti-parkinsonian drugs, benzodiazepines and some antidepressants.

In summary, the literature on balance does appear to support a conclusion that the deficits are more severe in schizophrenia compared with affective disorder, and that there may be some degree of specificity to schizophrenia in the pattern of deficits. The deficits do not appear to be merely associated with the level of psychotic symptoms, though they should probably be expected to be transiently worsened when the psychotic (or affective) state is very acute and profound. Cognition may be affected by several clinical and treatment variables.

### **Difficulties in making a specific diagnosis in early psychosis on the basis of clinical symptoms alone**

Clinical symptoms are often poorly differentiated in patients in the early stages of a psychotic illness, the more so in youth, making it difficult to distinguish schizophrenia from affective psychosis at initial presentation. This results in a high rate of revision of diagnosis.<sup>94-105</sup> This is despite the demonstrated validity of a diagnosis of schizophrenia in childhood and adolescence and its continuity into adulthood when the diagnosis is made using very standardized, systematic (and time-consuming) methods in highly specialized settings.<sup>106-108</sup> Classic psychotic symptoms, including "Schneiderian" first-rank symptoms, have been shown even in adults to be unreliable in differentiating schizophrenia from affective disorder.<sup>109-111</sup>

There is a need to find more specific measures than traditional symptom-based clinical assessments that could help to differentiate the diagnosis at the time of initial presentation in early psychosis.

### **Adding a criterion for cognitive impairment to the DSM criteria for schizophrenia**

Given that cognitive deficits are a core, relatively stable characteristic of schizophrenia, are largely independent of clinical symptoms and show some degree of specificity relative to affective disorders, consideration should be given to incorporating cognitive deficits into the diagnostic criteria for schizophrenia.

Diagnostic criteria need to be valid, reliable and have predictive utility in order to be useful to clinicians and researchers.<sup>112-115</sup> Although the literature points to cognitive deficit being a valid characteristic of schizophrenia, it is not easy to use as a reliable criterion on ac-

count of the lack of complete specificity relative to normal and other psychiatric conditions and the difficulty in finding a simple, widely available, standardized measure of the deficits.

If cognitive impairment is to be added as a diagnostic criterion, consideration needs to be given as to whether it should be added in a quantitative (neuropsychologic test) or qualitative (clinical impression) form. Tsuang et al,<sup>1,2</sup> in their proposal to incorporate the construct of schizotaxia into the DSM criteria, have put forward fairly stringent quantitative neuropsychologic criteria, which, while well formulated and relatively more reliable, could prove to be unwieldy in most clinical settings.

There are precedents for adding criteria for schizophrenia that have had similar difficulties in being used. For example, formal thought disorder is difficult to define and quantify in a reliable way. The DSM-III-R<sup>116</sup> wording of "incoherence or marked loosening of associations" proved to have extremely variable rates of diagnosis across several large samples of patients with schizophrenia. This wording was therefore replaced in DSM-IV by the more generic term "disorganized speech" and does not include a severity specifier.<sup>113</sup> Similarly, there was difficulty defining negative symptoms, which are harder to assess clinically than the more obvious positive symptoms. There were questions about how much weight to assign negative symptoms in the symptom algorithm versus maintaining the traditional reliance on positive symptoms to make the diagnosis. The final wording was decided based on quantifying the reliability, specificity and base rates of these symptoms in the field trials for DSM-IV.<sup>113</sup> It is also relevant to consider the DSM-IV and ICD-10<sup>117</sup> cri-

teria for cognitive deficit in Alzheimer's type dementia, the prototype cognitive disorder. There is no requirement in those criteria for actual measurement of the cognitive deficit through neuropsychologic testing.

Aside from the aim of utility of diagnostic criteria, DSM-IV makes the point that it serves as an educational tool to teach psychopathology (p. xv).<sup>3</sup> This alone is a compelling reason for the criteria to incorporate all core characteristics of the disease.

The question arises as to whether cognitive impairment should be added as an essential criterion, that is, as a stand-alone criterion. Doing so would effectively raise the threshold for making the diagnosis. In view of our incomplete knowledge regarding the specificity of the deficits to schizophrenia, but the importance of it being included in the definition of the disorder, it is therefore conservatively proposed here that it be added it as a nonessential criterion. This could be achieved by linking the new criterion to an existing criterion by an "or" statement. It is proposed that it be linked to the "social or occupational dysfunction" criterion (Criterion B), because there is a close association between cognitive deficits and level of functioning<sup>5</sup> and because Criterion B is a chronic or enduring criterion (as is cognitive deficit in schizophrenia), whereas the A criteria are acute or episodic.

The proposed additions to these criteria are presented in Table 1. The wording of the proposed cognitive impairment criterion is adapted from the DSM-IV criteria for dementia and from a synthesis of the literature reviewed in this paper.

By incorporating the proposed criterion as a nonessential criterion, it cannot raise the threshold for making a diagnosis of schizophrenia. It is also unlikely

**Table 1: Suggested addition to DSM-IV diagnostic criteria for schizophrenia**

**B. Social/occupational/cognitive\* dysfunction:** For a significant portion of the time since the onset of the disturbance, one or more major areas of functioning such as work, interpersonal relations, self-care, or *cognitive functioning* are markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, failure to achieve expected level of interpersonal, academic, or occupational achievement). *In adults, if cognitive impairment was present premonitory and has not deteriorated since the onset of the disturbance, then cognitive function should be markedly below normal or expected levels.*

*Cognitive impairment in schizophrenia is defined as marked impairment of 2 or more of the following 3 domains of cognitive function:*

1. *attention or vigilance (sustained attention)*
2. *memory (ability to learn new information or to recall previously learned information)*
3. *executive functioning (abstract reasoning, problem solving, planning, initiating, organizing, response inhibition, ability to shift cognitive set, sequencing, evaluating, or working memory, i.e., ability to manipulate information held in immediate memory).*

*These deficits may occur against a background of generalized or global intellectual impairment (e.g., subaverage IQ) and reduced capacity for information processing.*

Note: IQ = intelligence quotient.

\*Italicized text is the proposed addition to the *Diagnostic and statistical manual of mental disorders*, 4th edition (DSM-IV), p. 285.<sup>3</sup>



that the introduction of this criterion will significantly lower the threshold for making a diagnosis, because it sets the threshold for cognitive impairment relatively high, by requiring "marked" impairment in 2 of 3 domains of cognitive functioning. A case could perhaps be made for making the proposed criterion easier to meet, for example, by removing the term "marked" and/or by requiring impairment in only 1 of the 3 domains.

The proposed criterion does not attempt to quantify the degree of impairment (e.g., in terms of standard deviations below normal scores on specific neuropsychologic tests), so as not to make the criteria too stringent or inflexible. Although an argument could be made for requiring quantifiable measures of cognition in order to make the DSM criteria more reliable and valid, at this point data are lacking regarding cut-off scores on cognitive measures that would reliably differentiate most patients with schizophrenia from healthy individuals or from patients with affective disorders. It is proposed at this stage that the revised DSM text could elaborate that the assessment of cognitive impairment in schizophrenia should be based on the clinical impression of the clinician, supplemented, if possible, by neuropsychologic tests or quantified cognitive assessments. The DSM text should also note that the history provided by the patient, family member or other informant may not on its own be reliable in assessing cognitive impairment in schizophrenia, because the impairment tends to have been present premorbidly. The deficit may, therefore, be harder for the patient or family member to recognize, compared with the cognitive impairment of dementia, in which there has been a deterioration from usual functioning.

## Conclusions

Evidence has been presented in this paper that cognitive impairment is a core, stable and relatively specific characteristic of schizophrenia. I have argued that there is a need to enhance the predictive utility of the existing nonspecific DSM criteria for schizophrenia, particularly in early psychosis, in which it is so difficult to make the diagnosis based purely on psychotic symptoms and impairment of functioning. Because there remains some degree of inconclusiveness regarding the specificity and discriminating ability of cognitive deficits for schizophrenia, I have proposed conservatively that cognitive deficit be introduced as a nonessential diagnostic criterion at this stage. If the re-

liability and specificity of the proposed criterion are empirically established through further research, then cognitive deficit might be added as a stand-alone essential criterion in the DSM diagnostic algorithm for schizophrenia. I have argued that incorporating cognitive deficit into the diagnostic criteria at this stage is important not only for clinical diagnostic purposes but also for educational reasons to highlight that cognitive deficits are integral to the definition of schizophrenia. Spurring an understanding of the exact nature, specificity and stability of cognitive deficits in schizophrenia, as well as their relation to functional outcome, is sure to have far-reaching implications for the treatment and rehabilitation of people with the disorder. Finally, because certain cognitive deficits in schizophrenia are often detectable before the onset of illness, it is conceivable that cognitive assessment might also be a potentially valuable screening tool for early intervention programs in high-risk populations, which is one of the ultimate goals of psychiatric treatment. More research is called for, particularly longitudinal studies in young patients, to establish just how stable and specific cognitive deficits are in individuals with schizophrenia and to determine the extent to which the deficits are present before the first clinical manifestations of psychosis and are independent of severity of illness.

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