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The World Psychiatric Association (WPA)

The WPA is an association of psychiatric societies aimed to increase knowledge and skills necessary for work in the field of mental health and the care for the mentally ill. Its member societies are presently 123, spanning 106 different countries and representing more than 150,000 psychiatrists. The WPA organizes the World Congress of Psychiatry every three years. It also organizes international and regional congresses and meetings, and thematic conferences. It has 55 scientific sections, aimed to disseminate information and promote collaborative work in specific domains of psychiatry. It has produced recently several educational programmes and series of books. It has developed ethical guidelines for psychiatric practice, including the Madrid Declaration (1996). Further information on the WPA can be found in the website www.wpanet.org.

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Mental patients in prisons: punishment versus treatment?

AHMED OKASHA

President, World Psychiatric Association

One issue that has attracted the attention of our profession for decades is the struggle against political abuse of psychiatry, i.e. preserving political opponents or people otherwise breaking the law from being kept in mental hospitals under the pretext of mental illness. Investigations and efforts to monitor and prevent such violation included visits to mental hospitals and attempts to ensure that no person is being kept there for reasons other than his or her suffering from a mental disorder. The scope of such initiatives has been widely covered in the literature. The Hawaii and Madrid Declarations, which set guidelines and regulations to prevent abuse of our profession, have been a valuable product of this concern.

The other side of the coin, i.e. the incarceration of mental patients or their neglect in prisons, should not receive lesser attention and concern. Despite efforts over the last 30 years to promote diversion from jail for individuals with serious mental illness who have engaged in criminal behavior, few jail diversion programs have been adequately implemented. A sobering Guardian article on March 3, 2003 reports 300,000 mentally ill people to be held in US prisons. The US Bureau of Justice reports that an estimated 16% of the two million prisoners in the US are mentally ill, "often because there is nowhere else for them to go. So serious is the problem that one jail in Los Angeles has become in effect the biggest mental institution in the country". The situation has been exacerbated by the closure of many mental institutions: between 1982 and 2001, the numbers of public hospital beds available for the mentally ill decreased by 69% in the US. According to Oscar Morgan, a senior consultant at the National Mental Health Association (NMHA) and a former mental commissioner for the state of Maryland, this was a major issue for the prison service and "it is acknowledged now that many people in the prison system could, with proper treatment, be elsewhere".

The high numbers reported from the US are probably because of the transparency of the

subject and its coverage in the media. The same phenomenon is to be expected in many other countries: a recent report from India revealed equally high figures of mental patients in prisons. In several countries mental patients are in prisons because they did not have the chance to be examined before being convicted of a crime. In many countries which adopted the deinstitutionalization policy, the community care system is not reliable and is lacking in both financial and trained human resources to be able to provide the necessary service for mental patients, which contributes to the increased number of mental patients in prisons. It is also noteworthy that many Ministries of Finance did not channel the budgets of closed mental hospitals into other forms of mental health care services.

One disorder that stands out in this respect is substance abuse, which has been reported to have a high association with violence. Illicit drug abuse requires access to the black market; from there the road of substance abusers frequently leads into prison rather than into substance abuse rehabilitation centers (1). Other symptoms/disorders showing various degrees of association with criminal behavior include delusions, organic brain disorder, major affective disorder and antisocial personality disorder. Investigations of representative samples of US prison inmates (2-5) and Canadian penitentiary inmates (6) have revealed higher prevalence rates of mental disorders, particularly of the major mental disorders such as schizophrenia and major affective disorders, within these facilities than in the general population. Most of the major mental disorders were present before the current period of incarceration (7).

Offenders in prison experienced more social maladjustment than offenders in drug addiction treatment, they were less preoccupied by their drug consumption and less motivated to change (8). If anything, this should call for a treatment environment that responds more to rehabilitative needs than to punitive ones. Implications of jail diversion services for mental health professionals include learning how to collaborate with

law enforcement personnel, integrating mental health and substance abuse services into the criminal justice system despite segregated funding streams, and ensuring that clients who are intensively monitored are also provided with adequate treatment.

Some WPA member societies have expressed their concern regarding the incarceration of mental patients in prisons and especially in the US. The President of WPA brought these concerns to the attention of the WPA/APA leadership meeting in San Francisco in May 2003. Furthermore the President referred the issue to the WPA Review Committee, which promised a full report as how to best address this problem.

The presence of mental patients in prisons does not only deprive them of their right to proper treatment and care, but also leads to possible maltreatment and stigmatization. It is an ethical obligation to stop both. The UN resolution 1991 on the human rights of mental patients requires that they should be treated in adequate facilities, preserving their dignity. The Madrid Declaration states that mental patients should be treated by the least restrictive methods. Incarcerating mental patients is a violation of both.

As long as the budget of mental health is treated as the Cinderella of health services, mental patients will continue to be deprived of their right to be managed in mental

health premises rather than in prisons and other incarcerating places.

References

1. Arseneault L, Moffit TE, Caspi A et al. Mental disorders and violence in a total birth cohort. *Arch Gen Psychiatry* 2000;57:979-86.
2. Collins JJ, Schlenger WE. The prevalence of psychiatric disorder among admissions to prison. Presented at the 35th Annual Meeting of the American Society of Criminology, Denver, November 1985.
3. Daniel AE, Robins AJ, Reid JC et al. Lifetime and six month prevalence of psychiatric disorders among sentenced female offenders. *Bull Am Acad Psychiatry Law* 1988;16:333-42.
4. Hyde PS, Seiter RP. The prevalence of mental illness among inmates in the Ohio prison system. Department of Mental Health and the Ohio Department of Rehabilitation and Correction Interdepartmental Planning and Oversight Committee for Psychiatric Services to Corrections, 1987.
5. Neighbors HW, Williams DH, Gunnings TS et al. The prevalence of mental disorder in Michigan prisons. Final report submitted to the Michigan Department of Corrections, 1987.
6. Hodgins S, Cote G. The prevalence of mental disorders among penitentiary inmates. *Canada's Mental Health* 1990;38:1-5.
7. Teplin LA. The prevalence of severe mental disorder among urban jail detainees: comparison with the epidemiologic catchment area program. *Am J Public Health* 1990; 80:663-9.
8. Teplin LA. Criminalizing mental disorder: the comparative arrest rate of the mentally ill. *Am Psychol* 1984;39:794-803.

Mental health: scarce resources need new paradigms

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Mental disorders represent a major challenge to global development worldwide. Effective (and in some cases cost-effective) interventions are available for almost all of them. However, these interventions are often not implemented. Unknown variables, quite similar among themselves across the world, can have a strong influence in increasing the service quality in spite of the differences in resources and technologies. These unknown variables often result from the adoption of paradigms which are relatively independent from resources and technologies. We examine here four such paradigms.

Key words: Mental disorders, effective interventions, resources, service quality, paradigms

There is a rapid rise in the incidence of mental disorders. They represent a major challenge to global development. The rise in this burden will be relatively higher in developing countries, which have the least resources to respond. 450 million people are affected at any given time. Put in another way, one in four families has at least one member with a mental disorder. No group is immune to mental disorders, but the risk is higher among the poor, children and adolescents, abused women, the unemployed, persons with low education, the neglected elderly, victims of violence, migrants and refugees.

Effective (and in some cases cost-effective) interventions are available for almost all mental disorders. Often interventions do not cure the disorders but substantially improve symptoms or decrease relapses or lead to social (not clinical) recovery or improve quality of life.

Nevertheless, cost-effective interventions are often not implemented and there is a huge gap between treated and untreated. Indeed, several barriers prevent people from receiving appropriate treatment. These barriers are of different natures.

Stigma. Around the world, many people with mental disorders are victimized for their illness and become the targets of unfair discrimination. Access to housing, employment, and other normal societal roles are often compromised.

Discrimination in coverage for mental disorders. In many countries, mental disorders are not covered by health insurance schemes, and many people cannot afford treatment. One-quarter of all countries do not provide disability benefits to patients with mental disorders. One-third of the world's population – 2 billion people - live in countries that spend less than 1% of their health budgets on mental health.

Lack of drugs. Though 85% of countries have an essential drugs list that countries use as a basis for procuring therapeutic drugs, almost 20% of countries do not have at least one common antidepressant, one antipsychotic, and one antiepileptic in primary care.

Wrong priorities. Too many countries (mostly developed countries) still spend most of their resources on a few large mental asylums, which not only focus on a small fraction of those who need treatment but provide poor quality and often inhumane care.

Lack of skills at the primary health care level. Too few doctors and nurses know how to recognize and properly treat mental disorders. Forty-one per cent of countries do not have any mental health training programme for primary health care professionals.

Lack of rational and comprehensive mental health policies and legislation. Worldwide, 40% of countries do not have a mental health policy; 25% of countries do not have mental health legislation; 30% of countries do not have a national mental health programme.

Closing this gap is a clear obligation; otherwise no discourse around new classifications, concern about more sophisticated diagnosis, or development of innovative psychopharmacological research can be credible.

LESSONS LEARNED

As a conclusion of World Health Organization (WHO)'s global activities over the last two years, we can say that our key words are: more resources and more technologies and knowledge. However, reality seems more complex.

We have learned interesting lessons when providing policy assistance to countries like Brazil, Russia, Mexico, Mozambique, Israel, Fiji, Mongolia, Sri Lanka etc., and these lessons give us the sense of a more complex picture.

Lesson one. The quantity of resources available for psychiatric services provision is not in proportion to the quality of the system (there is of course a minimum critical mass of resources below which quality is simply impossible but, above this, quality is not granted by quantity).

Lesson two. Technologies available to the service provision system (diagnostic and therapeutic skills, special-

ization of services, professional diversification) are not proportionate to the system's quality (again, as before, there is of course a minimum critical mass of resources below which quality is simply impossible but, above this, quality is not granted by quantity). In other words, the equation R (resources) plus T (technologies) to be equal to Q (quality) requires one or more unknown variables.

In the Patagonian province of Rio Negro, the psychiatric hospital has been replaced by home visits, vocational rehabilitation, general hospital interventions, community care relying on an extended network of effective informal agents: the community is used as a resource.

In the Brazilian city of Santos, the psychiatric hospital is provided through a rich and articulated network of professionals who enjoy a rather unique popularity among the population. They use as additional resources patients themselves! The "cost" becomes the resource.

In the Indian Southern state of Tamil Nadu, the lack of solid public mental health service is replaced by a strong network of non-governmental organizations of professionals working in close collaboration with family members: the informal provider becomes a formal partner.

These three examples show informal resources and informal technologies working as the unknown variables resulting in quality.

Lesson three. Differences in systems are much less than one would expect. In spite of obvious huge differences in the availability of technologies and resources, problems and solutions (including bad solutions) remain rather similar even if nations, cultures and economies are diverse.

Following the lessons learned, we can say that unknown variables, quite similar among themselves across the world, can have a strong influence in increasing or decreasing the service quality in spite of the difference in resources and technologies.

These unknown variables often result from the adoption of paradigms which are relatively independent from resources and technologies. These paradigms are core assumptions, which affect the implementation of cost-effective interventions for mental disorders, making resources useless or powerful. Core assumptions essentially reflect "values".

Let us examine four paradigms we have identified as factors reducing or enhancing quality.

PARADIGMS

Exclusion versus inclusion

The "exclusion approach" is not focused on patient's experience/need but rather on environment's perception and need. This approach results in an overemphasis on security issues, including an overestimate of dangerousness.

We have noticed that systems where security preoccupations prevail are generally of worse quality than those where patients' understanding is prevailing.

When social organizations are extremely vertical (State is prevailing through a closed system of values), they tend to invest in control rather than in inclusion. Usually these social organizations identify exclusion with large institutions. On the other hand, when social organizations are very horizontal and the State is almost absent, they tend to organize exclusion in "diffuse institutions" where asylums' logic and rules remain in spite of the absence of visible walls: homelessness or abandonment to a wild private system of care with no quality control.

On the contrary, when a social organization starts addressing a patient's needs and looking at his/her pathology without denying the patient's enjoyment of full citizenship, the necessity of exclusion decreases. Shifting the paradigm from exclusion to inclusion has obviously enormous consequences in terms of investment in hospitals and beds.

Deinstitutionalization is therefore much more than simple de-hospitalization. It has to do with a radical assumption of the meaning of the patient's experience.

It is interesting how the notion of bed is still prevailing in mental health service planning language. It is interesting to notice how in the world there are either too many beds or, when there are too few, the only innovative idea is generally the one of creating new beds. Beds are not the solution but rather the illusion of responding in a simple (simplistic) way to a complex demand, namely better care, effective rehabilitation, more enjoyment of full citizenship.

Short-term care versus long-term care

A radical shifting of the care paradigm is required. Health systems are conceived and organized to respond to acute cases (hospital model). After the acute phase is resolved, the patient enters a limbo of infrastructures, human resources, skills, responsibilities. At that point the primary health care system appears as the *deus ex machina* that in ancient Greek tragedies used to solve everything. The key question, instead, is "how the entire health system can serve the needs of the patient when he/she requires long-term care (keeping the enjoyment of his/her full citizenship)?" And this is not just for mental disorders, but for many chronic conditions requiring long-term care (HIV/AIDS or tuberculosis, for example). Indeed, asylum temptation is there as the simplest and most devastating answer!

In other words, we need a radical shifting from a model centred on *space* location of the *provider* (hospitals, ambulatory, clinic and so on) to one centred on a *time* dimension of the *client*. Acute and chronic are *conditions*, not *places*, and we can conceive managing acute as well as chronic conditions at a community level more than at the hospital level.

Health systems tend to put resources and competencies in the short-term care (namely, hospitals) and to ignore or

delegate long-term care. As a consequence of that, a parallel system is created, which is characterized by poor resources and competencies. Instead, long-term care should imply means and strategies much beyond those associated with a medical model: diversity in people, competencies, places, social actors becomes a necessity. Comprehensiveness becomes the key word. Taking long-term seriously and avoiding shortcomings (institutionalisation, abandonment) requires a serious analysis of the real natural and social history of severe mental disorders.

Biopsychosocial approach versus biomedical approach

The social dimension of mental illness should be an intrinsic component of intervention and not just a concession in etiological modelling. The social dimension of mental illness requires a social dimension of treatment; neurosciences have provided an extraordinary contribution to understanding the brain but very few practical solutions. This statement has dramatic implications, because the emphasis of intervention should be moved from symptoms to functioning and disability.

Here the issue is the relationship between medical and social as two aspects of the same comprehensive approach or as two independent dimensions. Indeed, where health and social care are run by different political or administrative systems, there is a risk of failure: in fact, patients may be shunted from one service to another with obvious negative consequences on the continuity of care. In some cases the health sector is left just with the medical component of treatment. The result of this approach is a mental health service reduced to bio-medical treatment in hospitals for short period of time, on the one hand, and, on the other, a miserable rehabilitation service left in an isolated basket of generic and usually miserable welfare interventions. Or, similarly, where there are rigid boundaries between primary, secondary and tertiary care or between parts of the service, patients are either shunted around or simply drop out of the services.

The lack of the social dimension leads to inappropriate long-term care provision: entertainment instead of rehabilitation. Psychosocial rehabilitation is not entertainment for patients organized by second class staff or by specialists in the most bizarre techniques (painting, acting, playing, singing, sculpturing, etc.); it is re-construction of citizenship through everyday life skills. Indeed, rehabilitation can be a most sophisticated care provision for the severely mentally ill, but it requires interdisciplinary and intersectoral strategies and alliances.

The need for a much more radical permeability of disciplines takes us to our last paradigm: morbidity versus co-morbidity.

Morbid versus co-morbid

The distance between a DSM/ICD diagnosis and a real clinical case is huge. The “noise” disturbing the clean case is simply reality and psychiatrists often have the tendency of looking at reality as a dangerous noise making their laboratory so chaotic. However, real patients are more complex than pure diagnosis. For example, real patients have co-morbid diseases. Co-morbidity can occur within psychiatry or across different medical disciplines: e.g., cardiology and oncology. Co-morbidity can be also inter-human; namely, within a microenvironment like a family (alcohol abuse - domestic violence - depression - enuresis) or even in a macroenvironment (post-conflict, refugees, severely underprivileged urban settings).

Shifting the paradigm from vertical/mono-morbid interventions to co-morbidity approaches enhances effectiveness and adherence; furthermore, a matrix approach can avoid the under-utilization or mis-utilization of human resources. A mono-morbid paradigm will lead to vertical programmes where effectiveness is dispersed and expenditure is increased. A co-morbidity approach will instead facilitate the links between treatment of mental disorders and consequent enhancing of compliance and adherence to treatments for co-morbid physical diseases.

CONCLUSION

In 2001, the WHO made an exception to its usual politically correct language, saying “Stop Exclusion, Dare to Care”.

The WHO recognizes exclusion of people with mental disorders as a universal phenomenon to be fought. Fighting exclusion is a pre-condition (stop comes before dare) for care. No credible care can be framed within exclusion. Care should be dared (therefore, care exists), but why to dare? We need a courageous connection between scientific, empirical and ethical knowledge, if we want to offer answers which are humane, ethically acceptable, culturally adapted and cost-effective.

To do so, we need to build up networks of knowledge, people and institutions, otherwise we will offer simplistic answers to complex questions.

Human suffering is not linear, why should the response be linear?

Functional neuroimaging in mental disorders

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Recent advances in functional neuroimaging allow us to map neural activity in the living human brain with precise spatial and temporal resolution and provide an unprecedented opportunity to examine the neurocognitive components of mental disorders. In this article we aim to summarize the main functional neuroimaging findings in the major psychiatric disorders and the different methodological approaches that have been used to study them. We will discuss studies of the resting state and of activation during the performance of cognitive tasks, and studies focused on specific psychiatric symptoms. We will also review work on functional connectivity, discuss future directions in the field and consider how functional neuroimaging may contribute to clinical practice.

Key words: Functional neuroimaging, functional magnetic resonance imaging (fMRI), positron emission tomography (PET), mental disorders

Functional neuroimaging techniques – such as single photon emission computed tomography (SPECT), positron emission tomography (PET), functional magnetic resonance imaging (fMRI) – allow mapping of the physiology of the brain by measurement of blood flow, metabolism, and receptor-ligand binding. Research applying these techniques to mental illness has grown rapidly over the last two decades and has improved our understanding of the mechanisms underlying psychiatric disorders. In this article, we review functional neuroimaging studies of blood flow and glucose metabolism in mental illness. Neurochemical imaging and spectroscopy studies are not included.

RESTING STATE STUDIES

Early functional neuroimaging studies investigated brain activity in patients who were in the 'resting state'. The most robust finding in studies of resting cerebral blood flow (CBF) or metabolism in schizophrenia was decreased activity in frontal cortex (termed 'hypofrontality') relative to controls. However, some studies did not find differences between patients and controls in resting frontal activity, and others reported 'hyperfrontality' (1,2). Analogous findings have been described in depressive disorder, with several studies reporting decreased frontal activity, particularly in the dorsolateral prefrontal and anterior cingulate cortex, although again these results have not been consistent. Discrepant findings across resting state studies may be attributable to clinical heterogeneity: patients may differ in symptom profile, symptom severity, the duration of illness, and medication status. Another potential factor is that 'rest' may comprise a diversity of emotional and cognitive states in different subjects and across different studies.

One way of addressing the issue of the heterogeneous character of the resting state is to examine the correlation between regional CBF (rCBF) and symptom dimensions. Liddle et al (3) reported that each of three symptom

dimensions in schizophrenia (negative symptoms; formal thought disorder; delusions and hallucinations) was associated with a specific pattern of rCBF. Bench et al (4) employed the same approach with three symptom factors in depressive disorder (anxiety; psychomotor retardation; cognitive performance) and found that each was associated with a particular pattern of resting blood flow. While this approach has proved useful, activity measured during scanning is related to clinical ratings made outside the scanner. There is no means of controlling or measuring cognitive or emotional processes during the scan itself.

COGNITIVE ACTIVATION STUDIES

If subjects carry out a cognitive task during scanning, the cognitive and emotional processes that are active during the scan in different subjects are more likely to be similar than if subjects are scanned at 'rest'. Moreover, by selecting tasks which engage specific cognitive or emotional processes, the investigator can focus on functions that are thought to be particularly relevant to a given disorder. Thus, tasks involving 'executive' functions have been extensively examined in schizophrenia. As in resting state studies, many of these investigations have detected abnormal prefrontal responses in patients relative to controls. While 'hypofrontal' activation has often been reported, recent work has indicated that the nature of the activation can depend on which cognitive task is used, the level of task difficulty, and whether patients perform it as well as controls (5). Thus, using fMRI, Curtis et al (6,7) found that while patients with schizophrenia showed less prefrontal activation than controls during verbal fluency, activation in the same groups did not differ when they performed a semantic decision task. Using a graded memory task, Fletcher et al (5) demonstrated that patients with schizophrenia showed normal prefrontal activation until the demands on working memory were high and their performance deteriorated. There is also evidence that prefrontal activation can vary with the mental state of the

patient at the time of scanning: Fu et al (8) found that the degree to which prefrontal activation was reduced in patients with schizophrenia was related to the severity of positive symptoms of psychosis. Some authors have thus concluded that the term ‘hypofrontality’ is of limited utility (9).

Measuring brain activity while subjects perform cognitive tasks has also been used to investigate the biological bases of specific symptoms (as opposed to a disorder). For example, Kircher et al (10) employed a sentence completion task in conjunction with fMRI to examine semantic processing in patients with schizophrenia who exhibited formal thought disorder. They found that the activation in right temporal cortex, that was normally evident in controls and in non thought-disordered patients, was significantly attenuated in patients with thought disorder. Using joystick movement tasks during PET scanning, Spence et al (11) examined motor processing in schizophrenic patients with passivity phenomena. The latter engaged the right inferior parietal cortex more than patients with no passivity phenomena, and failed to show this when they were scanned again after remission. The same approach has been used to examine patients who have a trait vulnerability for a specific symptom but are not expressing this at the time of scanning. Using PET, McGuire et al (12) studied schizophrenic patients with and without history of auditory hallucinations while they were performing a task that engaged the monitoring of inner speech. Although the patients were asymptomatic at the time of study, those with a strong history of auditory hallucinations showed reduced activation in areas implicated in inner speech monitoring compared with patients with no history of hallucinations and controls. These findings have since been replicated and extended using fMRI (13).

STUDIES MEASURING SYMPTOMS ON-LINE

A relatively direct way of exploring the relationship between psychopathology and brain activity is to scan patients while they are actually experiencing a given symptom. In schizophrenia, this approach has been used in several studies of auditory hallucinations. Studies using SPECT (14), PET (15,16) and fMRI (17,18) have tried to capture the pattern of brain activity while patients were perceiving auditory hallucinations. While initial studies highlighted the involvement of different areas, such as the left inferior frontal cortex (14), the anterior cingulate gyrus (15), the lateral temporal cortex (17), and subcortical nuclei (16), recent work suggests that auditory hallucinations are mediated by a distributed network of areas that includes all of these regions (18). Because fMRI permits the acquisition of large numbers of images in a single patient, it is possible to study two different symptoms occurring at different times in the same individual. Thus Shergill et al (19) studied a patient with schizophrenia who was experiencing both auditory and tactile hallucina-

tions and showed that the former were associated with activity in the lateral temporal cortex whereas the latter were correlated with activation in the somatosensory and posterior parietal cortex (Figure 1).

While the above studies examined patients with ‘spontaneous’ hallucinations, symptoms can also be studied following their experimental provocation. This approach has been often applied in studies of anxiety disorders. Thus, obsessive-compulsive symptoms have been provoked in the scanner by presenting patients with obsessive-compulsive disorder (OCD) with potential contaminants (that elicit handwashing), and have been associated with activation in orbitofrontal and cingulate cortex, and in the striatum (20-22). Words, pictures, and sounds redolent of traumatic events have been employed to provoke symptoms in patients with post-traumatic stress disorder (PTSD) (23-25), which have been associated with decreased medial prefrontal and inferior frontal activation. Rauch et al (26) analyzed pooled PET data from symptom provocation paradigms in OCD, simple phobia and PTSD, and suggested that activation in inferior frontal and orbitofrontal cortices, insula, basal ganglia and brain stem were common across different anxiety disorders.

Symptom provocation has also been used in depression. Liotti et al (27) studied transient sadness, provoked by autobiographical memory script, in remitted depressive patients and actively depressed patients, using PET. They found that mood challenge in the remitted patients produced rCBF decrease in medial orbitofrontal cortex, which was also evident in active depressive patients but not in the healthy controls, consistent with a trait marker of depression. In schizophrenia, formal thought disorder

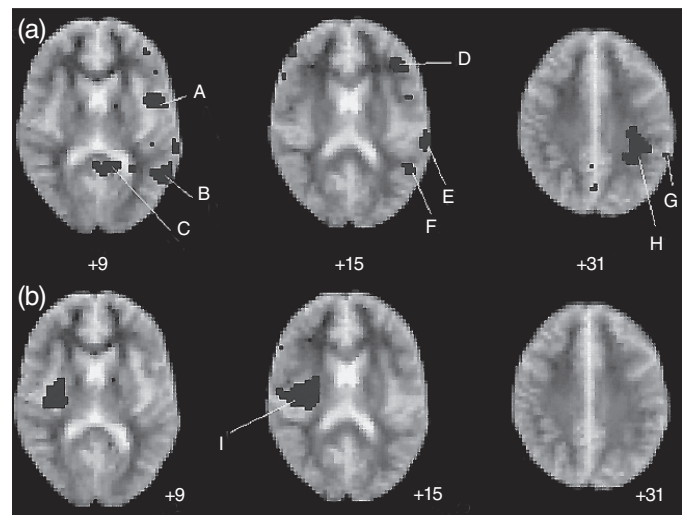


Figure 1 - Brain areas active during different types of hallucination in a patient with schizophrenia. Black voxels in row (a) indicate foci of activation associated with somatic hallucinations (A to H). Those in row (b) correspond to auditory hallucinations (I). The left side of the brain is shown on the right side of each image. The axial level (z coordinate in Talairach and Tournoux space) is shown below each slice. Adapted from Shergill et al (19).

has been induced by asking patients to interpret ambiguous pictures. Both McGuire et al (28), using PET, and Kircher et al (29), using fMRI, found that the severity of formal thought disorder was inversely correlated with activity in the left superior temporal cortex (Figure 2).

TREATMENT STUDIES

Functional imaging provides an opportunity to assess the effects of clinical treatments on brain function. Patients can be scanned before and after treatment, and changes in brain activity pattern may be related to improvements in symptoms and/or cognitive function within the same subjects.

Using a variety of interventions – including antidepressant drugs, electroconvulsive therapy, transcranial magnetic stimulation, sleep deprivation, and psychotherapy – functional imaging studies have examined activity before and after the treatment of major depressive disorder (MDD). The most robust finding is a normalization of resting frontal hypometabolism after treatment, while findings in other regions are inconsistent. However, recent studies suggest that various factors – including medication type (30), duration of treatment (31), symptom profile (32), treatment modality (medication vs. psychological treatment) (33,34), and the placebo effect (35) – can affect the pattern of brain activity change in MDD.

Changes in resting activity have also been reported after treatment with selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapy (CBT) in OCD. Successful treatment of OCD with SSRIs was associated with a decrease in caudate metabolism (36,37), as was successful CBT (36,38). Recently, Saxena et al (39) reported that the regional metabolic response to treatment with SSRIs was different in OCD to that in MDD. In OCD, symptomatic improvement was associated with decreased

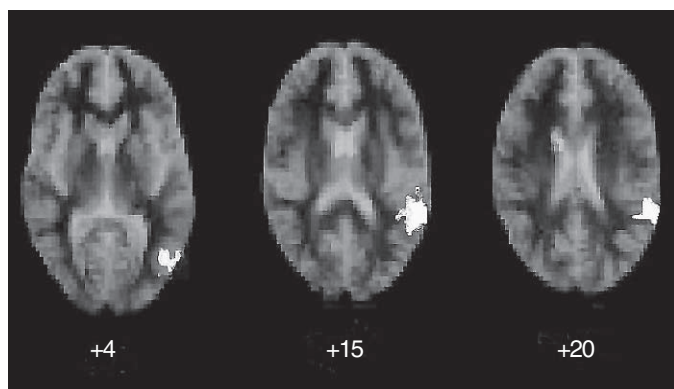


Figure 2 - Neural correlates of formal thought disorder in schizophrenia. When patients were talking there was an inverse correlation between the severity of thought disorder and activity in the left superior temporal gyrus (white voxels). The left side of the brain is shown on the right side of each image. The axial level (z coordinate in Talairach and Tournoux space) is shown below each slice. Adapted from Kircher et al (10).

metabolism in right caudate, right putamen, right ventrolateral prefrontal cortex, bilateral orbitofrontal cortex, and thalamus, but these changes were not evident in MDD. It should be noted, however, that treatment study findings in OCD have not been entirely consistent (40).

In schizophrenia, the effects of treatment with typical and atypical antipsychotics on neural activity have been compared. A PET study found that haloperidol treatment was associated with decreased resting CBF in frontal regions but increased CBF in the basal ganglia compared to risperidone, whereas risperidone treatment was associated with decreased rCBF in cerebellar regions compared to haloperidol (41). Using fMRI, Honey et al (42) found that after substitution of risperidone for typical antipsychotics, schizophrenic patients showed increased activation during a working memory task in the right prefrontal cortex, supplementary motor area, and posterior parietal cortex. The effects of psychological interventions in schizophrenia, such as CBT, remain largely uninvestigated.

FUNCTIONAL CONNECTIVITY

Theoretical models suggest that psychiatric disorders involve a disruption of the normal integration of different cognitive processes and activity in different brain regions. Functional connectivity refers to the temporal relationship between activity (as measured using functional imaging) in topographically distinct areas. Using PET data, Friston and Frith (43) found that healthy subjects, when performing a verbal fluency task, showed an inverse correlation between activity in prefrontal and superior temporal cortex. This correlation was absent in patients with schizophrenia. A similar difference in fronto-temporal correlations during verbal fluency task was described by Fletcher et al (44). Using the same task, Spence et al (45) found disturbed correlation between activity in left prefrontal and cingulate cortex in patients with schizophrenia, while Shergill et al (46) reported differences in the correlation between frontal and temporal activity in an fMRI study of covert verbal generation. These studies indicate that the correlation between activity in different regions in schizophrenia is perturbed, but cannot show whether there is a causal relationship between them. Path analysis can provide further information about the direction of the putative interactions between regions. Jennings et al (47) used this approach to examine PET data from a semantic processing task and found that schizophrenic patients showed a negative connection from left inferior frontal to left temporal cortex, which was positive in the controls, and a positive connection from the right frontal pole to the anterior cingulate cortex, which was negative in the controls (Figure 3). While these studies have yielded promising results, some studies have failed to detect differences in functional connectivity between patients and controls (48), and because this approach is relatively new, further research is required to develop the methodology.

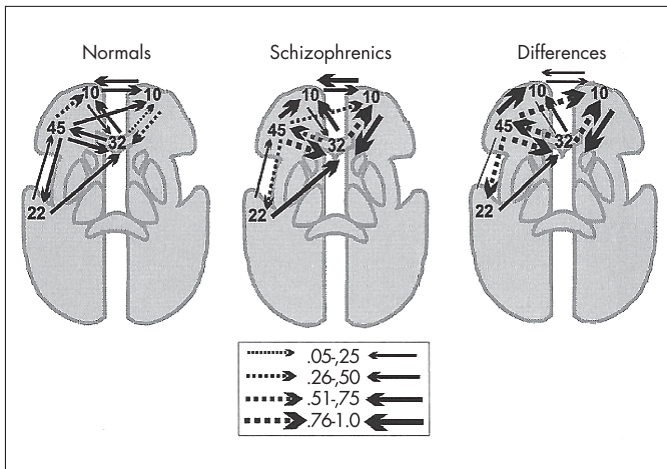


Figure 3 - Functional networks as defined by path analysis of functional imaging data from patients with schizophrenia and controls. The width of the arrow indicates the strength of the connection between each pair of areas. Positive path coefficients are represented as solid black arrows, and negative as dashed arrows. This particular analysis suggests that there are differences in functional connectivity between patients and controls. Adapted from Jennings et al (47).

Most of these studies have been carried out in schizophrenia, but there has been analogous work on other disorders. For example, Shaw et al (49) recently applied another analytic method (canonical variates analysis) to PET data from a working memory task in PTSD patients.

FUTURE DIRECTIONS

Integration of functional imaging and other data

To date, most neuroimaging studies in psychiatric disorders have involved a single type of scan: thus functional and structural imaging studies have usually been carried out separately. More recently, particularly with the increasing availability of MRI (which permits the acquisition of functional, volumetric and spectroscopic data with the same camera), investigators have been collecting different types of imaging data from the same subjects. Partly because the methodological problems in integrating data from distinct imaging modalities are not trivial, few such studies have been completed, but these are likely to emerge in the near future. Integrating the findings from functional imaging in psychiatric disorders with those from structural imaging would significantly advance our understanding of their pathophysiology. For example, some functional imaging studies suggest that there is a disruption of functional connectivity in schizophrenia. However it is unclear whether this reflects an underlying abnormality of the anatomical connections between cortical areas. This issue can be addressed using diffusion tensor imaging (DTI), an MRI technique which permits assessment of the integrity of white matter tracts. Initial applications of DTI in schizophrenia indicate that there may be abnormalities in cortico-cortical connections (50),

and it would be particularly interesting to examine how such changes are related to disturbances in functional connectivity in the same patients.

It may also be useful to integrate functional imaging data with information from non-imaging techniques. Transcranial magnetic stimulation (TMS) is a newly developed technology that can be used to noninvasively stimulate or inhibit selected regions of cerebral cortex. Combining TMS with functional neuroimaging makes it possible to examine the effects of modulating activity in a given region on the activity in other areas, particularly those it is connected to (51). One limitation of fMRI/PET techniques is their relatively low temporal resolution. Electroencephalography (EEG)/magneto-encephalography (MEG) signals have a higher temporal resolution but poorer spatial resolution. Integration of information from fMRI/PET and EEG/MEG has the potential to combine their respective advantages and provide data with high spatial and temporal resolution. Although collecting both types of data in the scanner is technically difficult, such studies are beginning to be done. For example, Mathiak et al recently carried out simultaneous recording of fMRI and MEG data while subjects were performing a mismatch paradigm (52). Applying such combined paradigms in patients (as opposed to volunteers) will present an additional challenge.

Clinical applications

While there has been a great deal of research using functional imaging in mental disorders, to date there has been relatively little use of functional imaging for purely clinical purposes. At present, diagnosis and assessment of prognosis and effectiveness of treatments are largely dependent on the clinical history and current psychopathology. Neuroimaging has yet to play a significant role in these areas, but there are signs that this is a possibility. For example, there is some evidence that the treatment of OCD and depression can normalize increased regional brain metabolism. Moreover, the severity of pretreatment abnormalities in these disorders can help to predict which patients will respond to treatment (30,53,54). In schizophrenia there is evidence that the severity of volumetric abnormalities (gray matter volume) in first episode patients are associated with a relatively poor prognosis (55). Other work with structural MRI suggests that subjects with prodromal signs of psychosis who later develop psychosis differ from subjects who do not, in having reduced gray matter volume in the prefrontal, cingulate, and medial temporal cortex (56). Since perturbations of regional brain function may be evident before macroscopic loss of gray matter, functional imaging may be a more powerful means of detecting differences of these types than structural imaging. However, to date there have been few studies of this type and further work is needed to explore this.

A key issue with all of the above is that the functional

imaging differences are quantitative rather than qualitative, and evident at the group level rather than the individual level. A key challenge for future work is to develop means of using data from a single patient to inform clinical assessment and management.

References

- Szechtman H, Nahmias C, Garnett ES et al. Effect of neuroleptics on altered cerebral glucose metabolism in schizophrenia. *Arch Gen Psychiatry* 1988;45:523-32.
- Ebmeier KP, Blackwood DH, Murray C et al. Single-photon emission computed tomography with 99mTc-exametazime in unmedicated schizophrenic patients. *Biol Psychiatry* 1993;33:487-95.
- Liddle PF, Friston KJ, Frith CD et al. Patterns of cerebral blood flow in schizophrenia. *Br J Psychiatry* 1992;160:179-86.
- Bench CJ, Friston KJ, Brown RG et al. Regional cerebral blood flow in depression measured by positron emission tomography: the relationship with clinical dimensions. *Psychol Med* 1993;23:579-90.
- Fletcher PC, McKenna PJ, Frith CD et al. Brain activations in schizophrenia during a graded memory task studied with functional neuroimaging. *Arch Gen Psychiatry* 1998;55:1001-8.
- Curtis VA, Bullmore ET, Brammer MJ et al. Attenuated frontal activation during a verbal fluency task in patients with schizophrenia. *Am J Psychiatry* 1998;155:1056-63.
- Curtis VA, Bullmore ET, Morris RG et al. Attenuated frontal activation in schizophrenia may be task dependent. *Schizophr Res* 1999;37:35-44.
- Fu CH, Suckling J, Williams S et al. Effects of psychotic state and task demand on prefrontal function in schizophrenia: an fMRI study of overt verbal fluency. Submitted for publication.
- Gur RC, Gur RE. Hypofrontality in schizophrenia: RIP. *Lancet* 1995;345:1383-4.
- Kircher TT, Bullmore ET, Brammer MJ et al. Differential activation of temporal cortex during sentence completion in schizophrenic patients with and without formal thought disorder. *Schizophr Res* 2001;50:27-40.
- Spence SA, Brooks DJ, Hirsch SR et al. A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain* 1997;120:1997-2011.
- McGuire PK, Silbersweig DA, Wright I et al. Abnormal monitoring of inner speech: a physiological basis for auditory hallucinations. *Lancet* 1995;346:596-600.
- Shergill SS, Bullmore E, Simmons A et al. Functional anatomy of auditory verbal imagery in schizophrenic patients with auditory hallucinations. *Am J Psychiatry* 2000;157:1691-5.
- McGuire PK, Shah GM, Murray RM. Increased blood flow in Broca's area during auditory hallucinations in schizophrenia. *Lancet* 1993;342:703-6.
- Cleghorn JM, Franco S, Szechtman B et al. Toward a brain map of auditory hallucinations. *Am J Psychiatry* 1992;149:1062-9.
- Silbersweig DA, Stern E, Frith C et al. A functional neuroanatomy of hallucinations in schizophrenia. *Nature* 1995;378:176-9.
- Dierks T, Linden DE, Jandl M et al. Activation of Heschl's gyrus during auditory hallucinations. *Neuron* 1999;22:615-21.
- Shergill SS, Brammer MJ, Williams SC et al. Mapping auditory hallucinations in schizophrenia using functional magnetic resonance imaging. *Arch Gen Psychiatry* 2000;57:1033-8.
- Shergill SS, Cameron LA, Brammer MJ et al. Modality specific neural correlates of auditory and somatic hallucinations. *J Neurol Neurosurg Psychiatry* 2001;71:688-690.
- McGuire PK, Bench CJ, Frith CD et al. Functional anatomy of obsessive-compulsive phenomena. *Br J Psychiatry* 1994;164:459-68.
- Rauch SL, Jenike MA, Alpert NM et al. Regional cerebral blood flow measured during symptom provocation in obsessive-compulsive disorder using oxygen 15-labeled carbon dioxide and positron emission tomography. *Arch Gen Psychiatry* 1994;51:62-70.
- Breiter HC, Rauch SL, Kwong KK et al. Functional magnetic resonance imaging of symptom provocation in obsessive-compulsive disorder. *Arch Gen Psychiatry* 1996;53:595-606.
- Bremner JD, Staib LH, Kaloupek D et al. Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder: a positron emission tomography study. *Biol Psychiatry* 1999;45:806-16.
- Shin LM, McNally RJ, Kosslyn SM et al. Regional cerebral blood flow during script-driven imagery in childhood sexual abuse-related PTSD: a PET investigation. *Am J Psychiatry* 1999;156:575-84.
- Lanius RA, Williamson PC, Boksman K et al. Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2002;52:305-11.
- Rauch SL, Savage CR, Alpert NM et al. The functional neuroanatomy of anxiety: a study of three disorders using positron emission tomography and symptom provocation. *Biol Psychiatry* 1997;42:446-52.
- Liotti M, Mayberg HS, McGinnis S et al. Unmasking disease-specific cerebral blood flow abnormalities: mood challenge in patients with remitted unipolar depression. *Am J Psychiatry* 2002;159:1830-40.
- McGuire PK, Quedest DJ, Spence SA et al. Pathophysiology of 'positive' thought disorder in schizophrenia. *Br J Psychiatry* 1998;173:231-5.
- Kircher TT, Liddle PF, Brammer MJ et al. Neural correlates of formal thought disorder in schizophrenia: preliminary findings from a functional magnetic resonance imaging study. *Arch Gen Psychiatry* 2001;58:769-74.
- Ketter TA, Kimbrell TA, George MS et al. Baseline cerebral hypermetabolism associated with carbamazepine response, and hypometabolism with nimodipine response in mood disorders. *Biol Psychiatry* 1999;46:1364-74.
- Mayberg HS, Brannan SK, Tekell JL et al. Regional metabolic effects of fluoxetine in major depression: serial changes and relationship to clinical response. *Biol Psychiatry* 2000;48:830-43.
- Brody AL, Saxena S, Mandelkern MA et al. Brain metabolic changes associated with symptom factor improvement in major depressive disorder. *Biol Psychiatry* 2001;50:171-8.
- Brody AL, Saxena S, Stoessel P et al. Regional brain metabolic changes in patients with major depression treated with either paroxetine or interpersonal therapy: preliminary findings. *Arch Gen Psychiatry* 2001;58:631-40.
- Martin SD, Martin E, Rai SS et al. Brain blood flow changes in depressed patients treated with interpersonal psychotherapy or venlafaxine hydrochloride: preliminary findings. *Arch Gen Psychiatry* 2001;58:641-8.
- Mayberg HS, Silva JA, Brannan SK et al. The functional neuroanatomy of the placebo effect. *Am J Psychiatry* 2002;159:728-37.
- Baxter LR Jr., Schwartz JM, Bergman KS et al. Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. *Arch Gen Psychiatry* 1992;49:681-9.
- Saxena S, Brody AL, Maidment KM et al. Localized orbitofrontal and subcortical metabolic changes and predictors of response to paroxetine treatment in obsessive-compulsive disorder. *Neuropsychopharmacology* 1999;21:683-93.
- Schwartz JM, Stoessel PW, Baxter LR Jr. et al. Systematic changes in cerebral glucose metabolic rate after successful behavior modification treatment of obsessive-compulsive disorder. *Arch Gen Psychiatry* 1996;53:109-13.
- Saxena S, Brody AL, Ho ML et al. Differential cerebral metabolic changes with paroxetine treatment of obsessive-compulsive disorder vs major depression. *Arch Gen Psychiatry* 2002;59:250-61.

40. Rubin RT, Ananth J, Villanueva-Meyer J et al. Regional ^{133}Xe cerebral blood flow and cerebral $^{99\text{m}}\text{Tc}$ -HMPAO uptake in patients with obsessive-compulsive disorder before and during treatment. *Biol Psychiatry* 1995;38:429-37.
41. Miller DD, Andreasen NC, O'Leary DS et al. Comparison of the effects of risperidone and haloperidol on regional cerebral blood flow in schizophrenia. *Biol Psychiatry* 2001;49:704-15.
42. Honey GD, Bullmore ET, Soni W et al. Differences in frontal cortical activation by a working memory task after substitution of risperidone for typical antipsychotic drugs in patients with schizophrenia. *Proc Natl Acad Sci USA* 1999;96:13432-7.
43. Friston KJ, Frith CD. Schizophrenia: a disconnection syndrome? *Clin Neurosci* 1995;3:89-97.
44. Fletcher P, McKenna PJ, Friston KJ et al. Abnormal cingulate modulation of fronto-temporal connectivity in schizophrenia. *Neuroimage* 1999;9:337-42.
45. Spence SA, Liddle PF, Stefan MD et al. Functional anatomy of verbal fluency in people with schizophrenia and those at genetic risk. Focal dysfunction and distributed disconnectivity reappraised. *Br J Psychiatry* 2000;176:52-60.
46. Shergill SS, Fukuda R, Brammer M et al. Impaired monitoring of inner speech in schizophrenia. *Br J Psychiatry* (in press).
47. Jennings JM, McIntosh AR, Kapur S et al. Functional network differences in schizophrenia: a rCBF study of semantic processing. *Neuroreport* 1998;9:1697-700.
48. Welchew DE, Honey GD, Sharma T et al. Multidimensional scaling of integrated neurocognitive function and schizophrenia as a disconnection disorder. *Neuroimage* 2002;17:1227-39.
49. Shaw ME, Strother SC, McFarlane AC et al. Abnormal functional connectivity in posttraumatic stress disorder. *Neuroimage* 2002;15:661-74.
50. Lim KO, Hedehus M, Moseley M et al. Compromised white matter tract integrity in schizophrenia inferred from diffusion tensor imaging. *Arch Gen Psychiatry* 1999;56:367-74.
51. Paus T, Castro-Alamancos MA, Petrides M. Cortico-cortical connectivity of the human mid-dorsolateral frontal cortex and its modulation by repetitive transcranial magnetic stimulation. *Eur J Neurosci* 2001;14:1405-11.
52. Mathiak K, Rapp A, Kircher TT et al. Mismatch responses to randomized gradient switching noise as reflected by fMRI and whole-head magnetoencephalography. *Hum Brain Mapp* 2002;16:190-5.
53. Mayberg HS, Brannan SK, Mahurin RK et al. Cingulate function in depression: a potential predictor of treatment response. *Neuroreport* 1997;8:1057-61.
54. Brody AL, Saxena S, Schwartz JM et al. FDG-PET predictors of response to behavioral therapy and pharmacotherapy in obsessive compulsive disorder. *Psychiatry Res* 1998;84:1-6.
55. Zipursky RB, Zhang-Wong J, Lambe EK et al. MRI correlates of treatment response in first episode psychosis. *Schizophr Res* 1998;30:81-90.
56. Pantelis C, Velakoulis D, McGorry PD et al. Neuroanatomical abnormalities before and after onset of psychosis: a cross-sectional and longitudinal MRI comparison. *Lancet* 2003;361:281-8.

Body dysmorphic disorder: recognizing and treating imagined ugliness

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Body dysmorphic disorder (BDD), also known as dysmorphophobia, is a severe psychiatric disorder that occurs around the world. However, the diagnosis is usually missed in clinical settings. It is important to recognize and diagnose BDD, because this disorder is relatively common and causes significant distress and impairment in functioning. It is also associated with markedly poor quality of life. Although research on effective treatment is still limited, serotonin reuptake inhibitors (SRIs) are currently considered the medication treatment of choice. For symptoms to improve, a relatively high SRI dose and at least 12 weeks of treatment is often needed. The psychosocial treatment of choice is cognitive behavioral therapy, consisting of elements such as exposure, response prevention, behavioral experiments, and cognitive restructuring. Although knowledge of BDD is rapidly increasing, further research is needed on all aspects of this disorder, including treatment studies, epidemiology studies, and investigation of its cross-cultural features and pathogenesis.

Key words: Body dysmorphic disorder, dysmorphophobia, delusional disorder, somatoform disorders

Body dysmorphic disorder (BDD), also known as dysmorphophobia, is an underrecognized yet relatively common and severe mental disorder that occurs around the world. Patients with BDD believe they look ugly or deformed (thinking, for example, that they have a large and ‘repulsive’ nose, or severely scarred skin), when in reality they look normal. As a result of their appearance concerns, they may stop working and socializing, become housebound, and even commit suicide (1,2).

Enrico Morselli, a psychiatrist in Italy, first described BDD more than 100 years ago (3), noting that “The dysmorphophobic, indeed, is a veritably unhappy individual, who in the midst of his daily affairs, in conversations, while reading, at table, in fact anywhere and at any hour of the day, is suddenly overcome by the fear of some deformity ... (which) may reach a very painful intensity, even to the point of weeping and desperation”. Other authors, including Kraepelin (4) and Janet (5), have described BDD over the past century, referring to it with terms such as ‘dermatologic hypochondriasis’, Schönheitshypochondrie (‘beauty hypochondria’), and Hässlichkeitskümmerer (‘one who is worried about being ugly’) (1).

DSM-IV classifies BDD as a separate disorder, defining it as a preoccupation with an imagined defect in appearance; if a slight physical anomaly is present, the person’s concern is markedly excessive (6). The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, and it cannot be better accounted for by another mental disorder, such as anorexia nervosa. DSM-IV classifies BDD as a somatoform disorder, but classifies its delusional variant as a psychotic disorder (a type of delusional disorder, somatic type). (However, delusional patients may be diagnosed with both BDD and delusional disorder, reflecting clinical impressions and empirical evidence that delusional and nondelusional

al BDD are probably the same disorder, which spans a spectrum of insight [7].) ICD-10 also groups BDD with the somatoform disorders, but unlike DSM-IV classifies BDD as a type of hypochondriasis (8); it classifies delusional BDD as a type of ‘other persistent delusional disorders’.

CLINICAL FEATURES

Individuals with BDD obsess that there is something wrong with how they look, even though the perceived appearance flaw is actually minimal or nonexistent (1,2,9-14). They may describe themselves as looking unattractive or deformed, or even hideous or like a monster. Concerns most often focus on the face or head (e.g., acne or skin color, balding, or head size) but can include any body area or the entire body, and concern with multiple body areas is typical. The appearance preoccupations are difficult to resist or control, and on average consume 3 to 8 hours a day. They are often associated with fears of rejection and feelings of low self-esteem, shame, embarrassment, unworthiness, and being unlovable. Insight is usually poor, and nearly half of patients are delusional (i.e., completely certain that they look abnormal and that their view of the ‘defect’ is accurate) (2,7). In addition, a majority have ideas or delusions of reference, thinking that others take special notice of the ‘defect’, perhaps staring at it, talking about it, or mocking it.

Most patients perform repetitive, compulsive behaviors aimed at examining, improving, or hiding the ‘defect’ (1,2,9-14). Common behaviors include mirror checking, comparing with others, excessive grooming (e.g., applying makeup, hair styling), camouflaging (e.g., with a hat, clothes, or makeup), frequent clothes changing, reassurance seeking, skin picking, and eating a restricted diet.

These behaviors typically occur for many hours a day and are difficult to resist or control.

Some studies report an approximately equal gender ratio (15), whereas others report a preponderance of men (11) or women (12,16) (although referral biases are evident in some reports). A majority of patients have never been married, and a relatively high proportion are unemployed (7,13). The disorder's clinical features appear generally similar in women and men, although several differences are apparent (15,17).

BDD usually begins during early adolescence and can occur in childhood. Although there is a dearth of research in this age group, BDD's clinical features in children and adolescents appear similar to those in adults (18). Prospective studies of BDD are lacking, but available data indicate that the disorder is typically chronic, often with waxing and waning symptoms (10).

Most BDD patients seen in psychiatric settings have other mental disorders. Most studies have found that major depression is the most common comorbid disorder, with the largest study (n=293) reporting a current rate of 58% and a lifetime rate of 76% (19). In this study, onset of major depression most often occurred after onset of BDD, consistent with clinical impressions that depression is often (although not always) secondary to BDD. Substance use disorders, social phobia, obsessive compulsive disorder (OCD), and personality disorders (most often, avoidant) also commonly co-occur with BDD (10,19).

IMPAIRMENT IN FUNCTIONING, DISTRESS, AND QUALITY OF LIFE

Although level of functioning varies, BDD nearly always causes impaired functioning - often to a marked degree - as well as other complications (1,2,7,9,13,18). Social impairment is nearly universal. Individuals with BDD may have few or no friends, and may avoid dating and other social interactions. Most patients also have impaired academic, occupational, or role functioning. BDD obsessions, behaviors, or self-consciousness about being seen often diminish concentration and productivity. Patients not uncommonly drop out of school or stop working. In one series, nearly 30% had been completely housebound for at least one week, more than half had been psychiatrically hospitalized, more than two thirds had experienced suicidal ideation due to BDD, and nearly 30% had attempted suicide (7). A study of dermatology patients who committed suicide reported that most had acne or BDD (20).

BDD patients experience unusually high levels of perceived stress (21) and markedly poor quality of life. In a study that assessed health-related quality of life with the Short Form Health Survey (SF-36), outpatients with BDD (n=62) scored notably worse in all mental health domains than norms for the general US population and for patients with depression, type II diabetes, or a recent myocardial

infarction (22). More severe BDD symptoms were associated with poorer mental health-related quality of life.

CULTURAL ASPECTS OF BDD

BDD has been reported in numerous countries and continents around the world - not only the US, Canada, Australia, and many countries in Eastern and Western Europe, but also China, Japan, the former Soviet Union, South America, Africa, and others (e.g., 1, 23-27). However, the largest systematic phenomenology studies of BDD, to my knowledge, are from the US (n=293 [20] and n=50 [11]), Italy (n=58) (13), and England (n=50) (12). Thus, reports from these countries have shaped much of our knowledge of BDD's clinical features.

Only one cross-cultural study has been done, which compared BDD's prevalence in nonclinical samples of American (n=101) and German (n=133) students, finding similar rates in the two groups (4.0% of Americans and 5.3% of Germans) (28). No cross-cultural studies have compared BDD's clinical features in community or clinical samples. Nonetheless, published case reports and series from around the world suggest that BDD's clinical features are generally similar across cultures, but that culture may produce nuances and accents on an apparently invariant, or universal, expression of BDD. For example, case series from Japan suggest that BDD's clinical features in that country are generally similar to those in other countries; however, concern with the eyelids and with causing others displeasure (by appearing unattractive) may be more common than in Western cultures.

Questions have been raised as to whether koro is related to BDD. Koro, a culture-related syndrome occurring primarily in Southeast Asia, is characterized by a preoccupation that the penis (labia, nipples, or breasts in women) is shrinking or retracting and will disappear into the abdomen, resulting in death (29). While koro has similarities to BDD, it differs in its usually brief duration, different associated features (usually fear of death), response to reassurance, and occasional occurrence as an epidemic.

PREVALENCE

Although large epidemiologic surveys of BDD's prevalence have not been done, studies to date indicate that BDD is relatively common in both nonclinical and clinical settings (14). Studies in community samples have reported current rates of 0.7% and 1.1%, and studies in nonclinical student samples have reported rates of 2.2%, 4%, and 13% (14). A study in a general inpatient setting found that 13% of patients had BDD (30). Studies in outpatient settings have reported rates of 8%-37% in patients with OCD, 11%-13% in social phobia, 26% in trichotillomania, 8% in major depression, and 14%-42% in atypical major depression (14). In one study of atypical depression, BDD was more than twice as common as OCD (31), and

in another (32) it was more common than many other disorders, including OCD, social phobia, simple phobia, generalized anxiety disorder, bulimia nervosa, and substance abuse or dependence. In a dermatology setting, 12% of patients screened positive for BDD, and in cosmetic surgery settings, rates of 6%-15% have been reported (14).

BDD is underdiagnosed, however. Two studies of inpatients (2,30), as well as studies in general outpatients (33) and depressed outpatients (31), systematically assessed a series of patients for the presence of BDD and then determined whether clinicians had made the diagnosis in the clinical record. All four studies found that BDD was missed by the clinician in every case in which it was present. Thus, underdiagnosis of BDD appears very common.

DIAGNOSING BDD

BDD may be difficult to diagnose because many patients are too ashamed to reveal their symptoms, fearing that their concerns will be trivialized or considered vain (9). Unless BDD is specifically asked about, the diagnosis is easily missed. Not diagnosing BDD is problematic because treatment may be unsuccessful, and the patient may feel misunderstood and inadequately informed about the diagnosis and treatment options. BDD can be diagnosed using the following questions (9), which reflect its DSM-IV criteria:

1) Are you very worried about your appearance in any way? (*OR*: Are you unhappy with how you look?) *If yes*: what is your concern?

2) Does this concern preoccupy you? That is, do you think about it a lot and wish you could worry about it less? How much time do you spend thinking about (*fill in body areas of concern*)?

3) What effect has this preoccupation with your appearance had on your life? Has it:

- Significantly interfered with your social life, school work, job, other activities, or other aspects of your life?
- Caused you a lot of distress?
- Affected your family or friends?

BDD is diagnosed in people who are 1) concerned about a minimal or nonexistent appearance flaw, 2) preoccupied with the perceived flaw (think about it for at least an hour a day), and 3) experience clinically significant distress or impaired functioning as a result of their concern.

BDD should be inquired about when patients have referential thinking, are housebound, have unnecessary surgery or dermatologic treatment, or present with social anxiety, depression or suicidal ideation.

To diagnose BDD, ICD-10 and certain diagnostic instruments require that patients refuse to accept the advice and reassurance of one or more doctors. This requirement will result in underdiagnosis of BDD, because many patients, despite having severe symptoms,

do not seek medical help or reveal their symptoms because of shame, limited access to health care, or other reasons. Furthermore, screening measures for the somatoform disorders that are based on the presence of physical symptoms are also likely to underdiagnose BDD, because BDD only rarely presents with physical symptoms typical of other somatoform disorders. In fact, preliminary data suggest that BDD patients do not have elevated levels of somatization (31).

Patients may present to clinicians revealing only anxiety, depression, or suicidal ideation (9). Consequently, BDD may be misdiagnosed as social phobia or agoraphobia (due to secondary social anxiety and isolation) or as panic disorder (because situational panic attacks may occur, for example, when looking in the mirror). Often, BDD is missed in depressed patients, in whom only depression is diagnosed. BDD is commonly misdiagnosed as OCD (because both disorders are characterized by obsessions and compulsive behaviors) and may also be misdiagnosed as trichotillomania (in patients who cut or pluck their hair to improve their appearance). Delusional BDD is sometimes misdiagnosed as schizophrenia or psychotic depression.

TREATMENT

Although treatment research is still limited, serotonin reuptake inhibitors (SRIs) and cognitive-behavioral therapy (CBT) are currently the treatments of choice (34,35). Available data indicate that SRIs, but not other medications or electroconvulsive therapy, are often efficacious for BDD, even for delusional patients (34). Following reports of cases that responded to SRIs (36), a largely retrospective study of 30 patients found that 58% responded to SRIs compared to only 5% for other medications (2); an expansion of this study (n=130) yielded similar findings (34). Another retrospective study (n=50) similarly found that SRIs were more effective than non-SRI tricyclics (37). Two prospective open-label studies of the SRI fluvoxamine found that two thirds of patients responded (38,39). In a prospective study of the SRI citalopram, 11 of 15 patients responded; functioning and quality of life, as well as BDD symptoms, significantly improved (40).

Only two controlled pharmacotherapy studies have been done; additional controlled studies are needed. In a double-blind cross-over trial (n=29 randomized patients), the SRI clomipramine was more effective than the non-SRI antidepressant desipramine (41). In the only placebo-controlled study (n=67 randomized patients), the SRI fluoxetine was more effective than placebo (42). Of note, available data consistently indicate that SRIs are effective even for delusional BDD (7,39,41,42), whereas delusional BDD does not appear to respond to antipsychotics alone (34).

Although dose-finding studies are lacking, BDD appears to often require higher doses than typically used

for depression. In a chart-review study (n=90), the mean SRI doses were 66.7 ± 23.5 mg/day of fluoxetine, 308.3 ± 49.2 mg/day of fluvoxamine, 55.0 ± 12.9 mg/day of paroxetine, 202.1 ± 45.8 mg/day of sertraline, and 203.3 ± 52.5 mg/day of clomipramine (43). Some patients respond only to doses higher than the maximum recommended dose (e.g., 80-100 mg/day of citalopram or paroxetine). In most studies, which used fairly rapid dose titration, the average time required for BDD to respond was 6-9 weeks, with some patients requiring 12 or even 14 weeks (34). It is therefore recommended that patients receive an SRI for at least 12 weeks before switching to another SRI, and that the highest SRI dose recommended by the manufacturer (if tolerated) be reached if lower doses are ineffective. Long-term treatment appears often necessary (34).

There are only limited data on SRI augmentation strategies (34). Adding buspirone (40-90 mg/day) or combining clomipramine with an SSRI may be helpful (although clomipramine levels must be monitored). Adding an antipsychotic to an SRI is worth considering for delusional patients, although this strategy has received limited investigation. Agitated or highly anxious patients often benefit from a benzodiazepine in addition to an SRI. Patients who fail one adequate SRI trial may respond to another SRI or venlafaxine. If none of these strategies is effective, an MAO inhibitor may be worth trying.

Although psychotherapy research is also limited, CBT appears to often be effective (35). Most studies have combined cognitive components (e.g., cognitive restructuring aimed at challenging faulty appearance-related beliefs) with behavioral components, consisting mainly of exposure and response prevention (ERP) to reduce social avoidance and repetitive behaviors (such as mirror checking and excessive grooming). Early case reports indicated a successful outcome with exposure therapy (44) and cognitive plus behavioral techniques (45). In a subsequent series of 17 patients who received 4 weeks of daily individual 90-minute CBT sessions (20 total sessions), BDD symptom severity significantly decreased (46). In an open series of 13 patients treated with group CBT, BDD significantly improved in twelve 90-minute group sessions (47). In a study of 10 participants who received thirty 90-minute individual ERP sessions without a cognitive component, plus 6 months of relapse prevention, improvement was maintained at up to 2 years (48).

Two wait-list controlled studies have been published. In a randomized pilot study of 19 patients, those who received 12 weekly sessions of 60-minute individual CBT improved significantly more than those in a no-treatment wait-list control condition (49). In another study (n=54), women randomized to cognitive therapy plus ERP (provided in 8 weekly 2-hour group sessions) improved more than those randomized to a no-treatment wait-list control condition (16) (However, patients appeared to have relatively mild BDD, and most had body weight and shape concerns, making it difficult to determine the applicability

of the results to more severely ill patients with more typical BDD symptoms.)

The above findings are very promising, but more rigorously controlled studies are needed. Also requiring investigation are the optimal number, duration, and frequency of sessions as well as the relative efficacy of group versus individual treatment. It is not known whether behavioral treatment (ERP) alone is usually effective or whether cognitive restructuring and behavioral experiments are a necessary treatment component because of the poor insight and depression so often characteristic of BDD. A broadly applicable treatment manual is not available and is needed. It is also not known whether SRIs or CBT is more effective, or whether their combination is more effective than either treatment alone. However, for patients with severe BDD, especially very depressed or suicidal patients, an SRI is recommended, as partial response may make CBT more tolerable and enable patients to participate in CBT treatment.

Before instituting an SRI and/or CBT, it is important to provide psychoeducation on BDD. Many patients appreciate referrals to books or websites (e.g., 9,50). For patients who are reluctant to accept the diagnosis and treatment (e.g., delusional patients), it can be helpful to emphasize that treatment is likely to decrease their suffering and improve functioning.

Research on insight-oriented and supportive psychotherapy is extremely limited but suggests that BDD symptoms - especially severe symptoms - are unlikely to significantly improve with these treatments alone (2). However, they can be helpful for other problems the patient may have and may be a useful adjunct to CBT and/or an SRI.

A majority of patients with BDD seek and receive surgery or nonpsychiatric medical (e.g., dermatologic) treatment. Some, in desperation, even do their own surgery - for example, attempting a facelift with a staple gun or trying to replace their nose cartilage with chicken cartilage in the desired shape (9,51). Although prospective studies are lacking, such treatments appear to usually be ineffective. In the largest study (n=250 adults from a psychiatric setting), only 7% of all nonpsychiatric treatments led to improvement in both concern with the treated body area and BDD more generally (52). Systematic treatment outcome studies of patients who clearly have BDD have not been done in nonpsychiatric settings, but observations in the dermatology and surgery literature generally indicate that the outcome of such treatments tends to be poor (53,54). Occasional dissatisfied patients commit suicide or are violent toward the treating physician (1).

CONCLUSIONS

BDD is a severe and relatively common psychiatric disorder that occurs around the world. However, it usually goes undiagnosed in clinical settings. It is important to

diagnose BDD, as it causes significant impairment in functioning and is associated with markedly poor quality of life. SRIs and CBT are currently considered the treatments of choice. However, studies of all aspects of BDD are needed - in particular, treatment studies, epidemiology studies (in which BDD symptoms are specifically inquired about and differentiated from other disorders such as hypochondriasis and OCD), cross-cultural studies, and investigation of BDD-related disability and the disorder's cost and burden to society. Research is also needed on whether BDD may be more closely related to social phobia, OCD, or depression than to most of the other somatoform disorders with which it is classified. Research on BDD's pathogenesis, including its underlying neurobiology, has just begun; such work may ultimately lead to more effective treatments and prevention of this severe mental disorder.

References

- Phillips KA. Body dysmorphic disorder: the distress of imagined ugliness. *Am J Psychiatry* 1991;148:1138-49.
- Phillips KA, McElroy SL, Keck PE Jr et al. Body dysmorphic disorder: 30 cases of imagined ugliness. *Am J Psychiatry* 1993;150:302-8.
- Morselli E. Sulla dismorfofobia e sulla tafefobia: due forme non per anco descritte di Pazzia con idee fisse. *Boll R Accad Genova* 1891;6:110-9.
- Kraepelin E. *Psychiatrie*, 8th ed. Leipzig: Barth, 1909-1915.
- Janet P. *Les obsessions et la psychasthenie*. Paris: Felix Alcan, 1903.
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*, 4th ed. Washington: American Psychiatric Association, 1994.
- Phillips KA, McElroy SL, Keck PE Jr et al. A comparison of delusional and nondelusional body dysmorphic disorder in 100 cases. *Psychopharmacol Bull* 1994;30:179-86.
- World Health Organization. *The ICD-10 classification of mental and behavioural disorders*. Geneva: World Health Organization, 1992.
- Phillips KA. *The broken mirror: understanding and treating body dysmorphic disorder*. New York: Oxford University Press, 1996 (revised and expanded edition, in press).
- Phillips KA. Body dysmorphic disorder. In: Phillips KA (ed). *Somatoform and factitious disorders*. Washington: American Psychiatric Publishing, 2001.
- Hollander E, Cohen LJ, Simeon D. Body dysmorphic disorder. *Psychiatr Ann* 1993;23:359-64.
- Veale D, Boocock A, Gournay K et al. Body dysmorphic disorder: a survey of fifty cases. *Br J Psychiatry* 1996;169:196-201.
- Perugi G, Giannotti D, Frare F et al. Prevalence, phenomenology, and comorbidity of body dysmorphic disorder (dysmorphophobia) in a clinical population. *Int J Psychiatry Clin Pract* 1997;1:77-82.
- Phillips KA, Castle DJ. Body dysmorphic disorder. In: Castle DJ, Phillips KA (eds). *Disorders of body image*. Hampshire: Wrightson Biomedical, 2002:101-20.
- Phillips KA, Diaz S. Gender differences in body dysmorphic disorder. *J Nerv Ment Dis* 1997;185:570-7.
- Rosen JC, Reiter J, Orosan P. Cognitive-behavioral body image therapy for body dysmorphic disorder. *J Consult Clin Psychol* 1995;63:263-9.
- Perugi G, Akiskal HS, Giannotti D et al. Gender-related differences in body dysmorphic disorder (dysmorphophobia). *J Nerv Ment Dis* 1997;185:578-82.
- Albertini RS, Phillips KA. 33 cases of body dysmorphic disorder in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 1999;38:453-9.
- Gunstad J, Phillips KA. Axis I comorbidity in body dysmorphic disorder. *Compr Psychiatry* 2003;44:270-6.
- Cotterill JA, Cunliffe WJ. Suicide in dermatological patients. *Br J Dermatol* 1997;137:246-50.
- DeMarco LM, Li LC, Phillips KA et al. Perceived stress in body dysmorphic disorder. *J Nerv Ment Dis* 1998;186:724-6.
- Phillips KA. Quality of life for patients with body dysmorphic disorder. *J Nerv Ment Dis* 2000;188:170-5.
- Korkina MB. The syndrome of dysmorphomania (dysmorphophobia) and the development of psychopathic personality. *Zh Nevropatol Psikhiatr* 1965;65:1212-7.
- Ung EK, Fones CSL, Ang AWK. Muscle dysmorphia in a young Chinese male. *Ann Acad Med Singapore* 2000;29:135-7.
- Turkson SNA, Asamoah V. Body dysmorphic disorder in a Ghanaian male: case report. *East Afr Med J* 1999;76:111-4.
- Yamada M, Kobashi K, Shigemoto T et al. On dysmorphophobia. *Bull Yamaguchi Med School* 1978;25:47-54.
- Fontenelle LF, Mendlowicz MV, Muzzi TC et al. The man with the purple nostrils: a case of rhinotrichotillomania secondary to body dysmorphic disorder. *Acta Psychiatr Scand* 2002;106:464-6.
- Bohne A, Keuthen NJ, Wilhelm S et al. Prevalence of symptoms of body dysmorphic disorder and its correlates: a cross-cultural comparison. *Psychosomatics* 2002;43:486-90.
- Chowdhury AN. The definition and classification of koro. *Cult Med Psychiatry* 1996;20:41-65.
- Grant JE, Won Kim S, Crow SJ. Prevalence and clinical features of body dysmorphic disorder in adolescent and adult psychiatric inpatients. *J Clin Psychiatry* 2001;62:517-22.
- Phillips KA, Nierenberg AA, Brendel G et al. Prevalence and clinical features of body dysmorphic disorder in atypical major depression. *J Nerv Ment Dis* 1996;184:125-9.
- Perugi G, Akiskal HS, Lattanzi L et al. The high prevalence of "soft" bipolar (II) features in atypical depression. *Compr Psychiatry* 1998;39:63-71.
- Zimmerman M, Mattia JI. Body dysmorphic disorder in psychiatric outpatients: recognition, prevalence, comorbidity, demographic, and clinical correlates. *Compr Psychiatry* 1998;39:265-70.
- Phillips KA. Pharmacologic treatment of body dysmorphic disorder: review of the evidence and a recommended treatment approach. *CNS Spectrums* 2002;7:453-60.
- Neziroglu F, Khemiani-Patel S. A review of cognitive and behavioral treatment for body dysmorphic disorder. *CNS Spectrums* 2002;7:464-71.
- Hollander E, Liebowitz MR, Winchel R et al. Treatment of body-dysmorphic disorder with serotonin reuptake blockers. *Am J Psychiatry* 1989;146:768-70.
- Hollander E, Cohen L, Simeon D et al. Fluvoxamine treatment of body dysmorphic disorder. *J Clin Psychopharmacol* 1994;14:75-7.
- Perugi G, Giannotti D, Di Vaio S et al. Fluvoxamine in the treatment of body dysmorphic disorder (dysmorphophobia). *Int Clin Psychopharmacol* 1996;11:247-54.
- Phillips KA, Dwight MM, McElroy SL. Efficacy and safety of fluvoxamine in body dysmorphic disorder. *J Clin Psychiatry* 1998;59:165-71.
- Phillips KA, Najjar F. An open-label study of citalopram in body dysmorphic disorder. *J Clin Psychiatry* 2003;64:715-20.
- Hollander E, Allen A, Kwon J et al. Clomipramine vs desipramine crossover trial in body dysmorphic disorder: selective efficacy of a serotonin reuptake inhibitor in imagined ugliness. *Arch Gen Psychiatry* 1999;56:1033-9.
- Phillips KA, Albertini RS, Rasmussen SA. A randomized placebo-

- bo-controlled trial of fluoxetine in body dysmorphic disorder. *Arch Gen Psychiatry* 2002;59:381-8.
43. Phillips KA, Albertini RS, Siniscalchi JM et al. Effectiveness of pharmacotherapy for body dysmorphic disorder: a chart-review study. *J Clin Psychiatry* 2001;62:721-7.
 44. Marks I, Mishan J. Dysmorphophobic avoidance with disturbed bodily perception: a pilot study of exposure therapy. *Br J Psychiatry* 1988;152:674-8.
 45. Cromarty P, Marks I. Does rational role-play enhance the outcome of exposure therapy in dysmorphophobia? A case study. *Br J Psychiatry* 1995;167:399-402.
 46. Neziroglu F, McKay D, Todaro J et al. Effect of cognitive behavior therapy on persons with body dysmorphic disorder and comorbid axis II diagnoses. *Behav Ther* 1996;27:67-77.
 47. Wilhelm S, Otto MW, Lohr B et al. Cognitive behavior group therapy for body dysmorphic disorder: a case series. *Behav Res Ther* 1999;37:71-5.
 48. McKay D. Two-year follow-up of behavioral treatment and maintenance for body dysmorphic disorder. *Behav Modif* 1999;23:620-9.
 49. Veale D, Gournay K, Dryden W et al. Body dysmorphic disorder: a cognitive behavioral model and pilot randomized controlled trial. *Behav Res Ther* 1996;34:717-29.
 50. <http://www.BodyImageProgram.com>
 51. Veale D. Outcome of cosmetic surgery and DIY surgery in patients with body dysmorphic disorder. *Psychiatr Bull* 2000;24:218-21.
 52. Phillips KA, Grant J, Siniscalchi J et al. Surgical and nonpsychiatric medical treatment of patients with body dysmorphic disorder. *Psychosomatics* 2001;42:504-10.
 53. Cotterill JA. Body dysmorphic disorder. *Psychodermatology* 1996;14:457-63.
 54. Fukuda O. Statistical analysis of dysmorphophobia in out-patient clinic. *Jap J Plast Reconstruct Surg* 1977;20:569-77.

Psychiatric comorbidity: is more less?

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With each successive revision of the DSM and ICD, psychiatric comorbidity has become more prevalent. The 'atheoretical' approaches of the DSM and ICD explicitly encourage multiple diagnoses with few exclusionary hierarchies, in the hope that all clinically relevant information will be captured. However, the current strategy of diagnosing 'maximal' comorbidity may not reflect 'optimal' comorbidity. Many clinicians and health information systems, particularly those in developing countries, have a limited capacity for capturing this diagnostic information, and fail to characterize additional diagnoses that are present. This article will address the evolution of our current diagnostic system as a way of understanding the emergence of comorbid psychiatric diagnoses. Alternative diagnostic approaches (a dimensional system, diagnostic hierarchies, and mixed diagnostic categories) that could be used to address the emergence of comorbid psychiatric diagnoses are considered. Future challenges for the next evolution of DSM and ICD are presented.

Key words: Psychiatric diagnosis, comorbidity

Since the revision of the DSM-III, there has been an apparent significant increase in the prevalence of comorbid psychiatric diagnoses. In the literature, much attention has been given to the co-occurrence of mood and anxiety disorders, psychosis and substance use disorders, as well as among the Axis II diagnoses, to name a few.

This article will examine the evolution of our current diagnostic system as a way of understanding the emergence of comorbid psychiatric diagnoses. Using clinical examples, we will explore several alternate strategies for reducing psychiatric comorbidity that could be implemented in place of the current one, and consider strengths and weaknesses of each.

The concept of comorbidity certainly is not unique to psychiatry. Feinstein (1) coined the term comorbidity as 'any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study'. A simple example would be a patient with chronic obstructive pulmonary disease and diabetes mellitus.

In psychiatry, when distinct symptoms like anxiety and depression co-occur, whether they indicate the presence of two distinct clinical entities or whether they are two components of a single disorder is mostly a matter of speculation, as we know little about the etiology and pathophysiology

interrelationship of mental illnesses. For example, does the patient who binges and purges and also abused alcohol really have two distinct mental illnesses (what we might view as 'true comorbidity', as defined by Feinstein), or are they both the manifestations of a primary disorder of impulse control?

In recognition of this lack of knowledge, the diagnostic systems that are in current use (DSM-IV and ICD-10) are broad, descriptive, and have relatively few exclusionary hierarchies (whereby one disorder is assumed to be responsible for, and therefore supercedes the diagnosis of, another).

The DSM-IV employs the basic strategy that, given the absence of knowledge about the underlying nature of psychiatric disorders, clinicians should convey the maximum amount of descriptive information possible. According to the DSM-IV, a patient who drinks heavily, has severe and recurrent depressive episodes, binges and purges, and has panic attacks, would be assigned four separate Axis I diagnoses (as opposed to a single, all-encompassing diagnosis like "severe neurotic disorder" that might incorporate all of these symptoms). In the above case, giving four diagnoses might emphasize that each of these problems needs to be addressed in the treatment plan. This fact might be obscured if a single less descriptive diagnosis was imposed.

At its best, our current diagnostic system has the potential to communicate large amounts of detailed clinical information about patients with complex problems, allowing for targeted treatments and more precisely defined study populations. At its worst, it can be overwhelming to clinicians and health information systems, and obscure the focus of our treatments by 'losing the forest for the trees'.

HISTORICAL CONTEXT

With each successive revision from DSM-I to DSM-IV, psychiatric comorbidity has become more prevalent. The reason lies in the design of the diagnostic system itself: the DSM-IV is a descriptive, categorical system that splits psychiatric behaviors and symptoms into numerous distinct diagnoses, and employs few exclusionary hierarchies to eliminate multiple diagnoses.

The original version of the DSM was a descriptive system that incorporated many of the concepts and the structure of Emil Kraepelin's classifications of mental disorders. Compared to subsequent revisions, the DSM-I and DSM-II followed a "one disease-one diagnosis" model in which the clinician strived to assign a single all-encompassing diagnosis, using qualifying phrases (such as: "with neurotic reaction" and "with psychotic reaction") to describe complex cases.

Qualifying phrases from other categories could be applied to any of the major diagnoses to 'lump' symptoms into fewer, broader categories. For example, 'schizophrenic reaction, hebephrenic type, with neurotic reaction' might be used to describe a patient with a primary diagnosis of schizophrenia who also shows clinically significant depressive symptoms. In another example from DSM-I, a patient with different types of anxiety symptoms is given a single diagnosis of 'phobic reaction, manifested by claustrophobia, with obsessive-compulsive symptoms, counting and recurring thoughts'.

The DSM-III, however, took a different approach of cutting the diagnostic pie into small slices, adding a large number of relatively narrowly defined psychiatric diagnoses, and supplied operationalized diagnostic criteria for each. For example, the DSM-III split the single DSM-II category 'phobic neurosis' into five DSM-III categories: agoraphobia with panic, agoraphobia without panic, social phobia, simple phobia, and separation anxiety disorder. Not surprisingly, the number of distinct psychiatric diagnoses described in the DSM-IV is nearly double that of the DSM-II.

Because of the potential for spurious comorbidity that might result from the increase in the overall number of categories, exclusionary criteria were sometimes added to reduce comorbidity in those cases where it was believed that a symptom presentation that met criteria for one disorder was really 'due to' another disorder. For example, the criteria for agoraphobia indicate that the diagnosis should not be given if the characteristic avoidant behavior is really due to obsessive-compulsive disorder and a diagnosis of panic disorder is not given if the panic attacks are really due to major depression. However, the use of the phrase 'due to' in these exclusion criteria forced the clinician to determine when a symptom was attributable to one disorder versus another, a decision based on assumptions about causality that are not

empirically-based. Resulting partly from research conducted by Boyd (2) in the 1970s and 1980s, many of the underlying assumptions about the relationship between mood and anxiety symptoms came into question. In subsequent revisions of the DSM, starting with the DSM-III-R, an increasing recognition that the existing diagnostic hierarchies were not based on empirical data led to the removal of many (though not all) exclusion criteria.

The ultimate result of this combination of widespread diagnostic splitting and the spare use of diagnostic hierarchies is the common occurrence that patients qualify for multiple diagnoses. In a study involving 500 subjects presenting for intake in a general psychiatric clinic, Zimmerman and Mattia (3), using semi-structured clinical interviews, noted that more than a third of the patients qualified for three or more Axis I disorders.

Psychiatric comorbidity is, in fact, explicitly encouraged in the DSM-IV. According to the 'Use of the Manual' section of the DSM-IV, 'the general convention in DSM-IV is to allow multiple diagnoses to be assigned for those presentations that meet criteria for more than one DSM-IV disorder'. The strategy is to encourage the clinician to record the maximum amount of diagnostic information, as a way of characterizing the complexity of clinical presentations.

Unfortunately, many clinicians and health information systems have a limited capacity for actually capturing this diagnostic information, and instead fail to characterize additional diagnoses that are present (4). Furthermore, recording 5 or 6 diagnoses on a patient's chart may obscure the intended focus of treatment. Many health information systems, particularly those in developing countries, only allow for coding a single diagnosis, with the result that any comorbid diagnoses are ignored. Analysis of diagnostic data collected from such systems may lead to erroneous assumptions being made (e.g., about treatment utilization). For example, consider three patients carry-

ing a primary diagnosis of severe, recurrent, major depressive disorder, each of whom has a different comorbid diagnosis (e.g., obsessive-compulsive disorder; alcohol dependence; gender identity disorder). A system that allows for the recording of only major depression would imply that these three patients were diagnostically homogeneous, when in fact their disparate comorbid diagnoses suggest otherwise.

APPRAISAL OF DSM

Since the ICD-10 diagnostic criteria for research were largely modeled on the DSM-III-R system, with a few key exceptions (i.e., the inclusion of several mixed diagnostic categories like hyperkinetic conduct disorder), the ICD-10 follows the DSM convention regarding psychiatric comorbidity.

The DSM-IV/ICD-10 approach to diagnostic comorbidity has several advantages from a clinical utility perspective. It maximizes the communication of diagnostic information and helps insure that all clinically important aspects of the patient's presentation are addressed. For example, when a patient has symptoms that meet criteria for "major depressive disorder, recurrent, moderate", we can consider specific, evidence-based interventions such as selective serotonin reuptake inhibitor (SSRI) therapy, perhaps combined with individual or group psychotherapy. The specifier "moderate" suggests that the impairment is likely not so severe as to warrant inpatient treatment. If the patient also has symptoms that meet criteria for panic disorder, we might also consider use of a short course of a benzodiazepine until the SSRI takes effect. Going further, if the patient's pattern of substance use also meets criteria for alcohol abuse or dependence, this may give us pause in the use of benzodiazepines to treat her anxiety, as well as cause us to question how much of her depression may be related to her drinking. Lastly, if our assessment reveals that this patient's longstanding pattern of relating to

other people is characteristic of borderline personality disorder, this may help the clinician to form a better sense of long-term prognosis, symptom chronicity, or suggest specific psychotherapies (such as dialectical behavioral therapy).

This clinical example demonstrates how each facet of the diagnostic formulation (reflected in its inclusion in the list of comorbid diagnoses) theoretically can provide a more complete appreciation of the complexity of the patient's clinical presentation, which has the potential to result in more appropriate treatment planning and prognosis.

There is evidence, however, to suggest that much of this diagnostic complexity is not being adequately captured in clinical practice. Zimmerman and Mattia (4) have summarized how several US studies demonstrated that clinicians routinely under-detect psychiatric comorbidity as compared to research assessments using structured diagnostic interviews. One such study found that five times as many comorbid diagnoses were made when using semi-structured interviews as opposed to the clinicians' assessments alone (5). The reasons underlying this discrepancy are probably complex and multifactorial. Many have speculated that, with shrinking reimbursement and more rapid patient flow, psychiatrists lack sufficient time to perform complete diagnostic assessments. It is worth noting that, in Basco's study, the added diagnostic information provided by semi-structured interviews led to a change in patient care in approximately one half of the patients at one month follow-up. This suggests that the comorbid diagnoses were clinically relevant enough to justify a change in the treatment plan.

While it makes intuitive sense that more comprehensive diagnostic information would result in improved patient outcomes, we do not have studies that have investigated this issue. It is likely that psychiatrists in clinical practice are simply choosing not to make certain diagnoses

because they do not consider them to be a clinically relevant focus of treatment. For example, the Epidemiological Catchment Area Study, which used a structured interview for its diagnostic assessments, indicated that specific phobia is the most common psychiatric disorder occurring in the US general population (6). Although using structured diagnostic interviews would increase the detection of specific phobia in patients as a comorbid diagnosis, this added information is not likely to be clinically relevant since this is typically not the reason patients seek psychiatric care (4).

Unfortunately, due to our field's limited grasp of how mental disorders interrelate and affect each other's emergence, treatment, and prognosis, we have very little evidence to guide clinicians in how to prioritize multiple co-occurring diagnoses, other than to direct clinicians to use their judgment. For example, DSM-IV provides the following statement: "When (as is often the case) an individual's pattern of behavior meets criteria for more than one Personality Disorder, the clinician should list all relevant Personality Disorder diagnoses in order of importance". But where is the data to guide clinicians in determining which diagnoses are relevant, or which ones are most important? Without such guidance, clinicians must prioritize diagnoses on a case-by-case basis subject to their own clinical judgment. In effect, clinicians apply their own (potentially idiosyncratic) hierarchical rules, resulting in a potential loss of diagnostic reliability and validity.

CLINICAL EXAMPLE: PERSONALITY DISORDERS

A prominent example of excessive comorbidity in the DSM-IV resulting in widespread dissatisfaction among clinicians is in the area of personality disorders (7,8). Using personality disorder diagnosis as a focus, we will describe three alternative approaches to dealing with the problem of comorbidity.

There are 10 distinct personality disorders described in the DSM, with no hierarchical system provided to reduce comorbidity. According to this system, when an individual's pattern of behavior meets criteria for more than one personality disorder, all diagnoses should be listed in order of clinical significance. Widiger (8) has pointed out that certain psychiatric inpatients can be found to meet criteria for 3 to 5, and in some cases up to 7 personality disorders. Using pooled data consisting of 1116 inpatients and outpatients at multiple sites, Stuart et al (9) found that, among those subjects that met criteria for at least one personality disorder, the average number of personality disorder diagnoses was 2.7 (with nearly 10% of subjects meeting criteria for 4 or more). In a study using two separate semi-structured interviews on 100 patients in long-term inpatient treatment, fewer than 15% had only a single personality disorder diagnosis (10).

Despite this, clinicians tend to diagnose personality disorders reductively, even when the patient has sufficient criteria to qualify for two or more specific diagnoses. In a study in which psychiatrists were given case histories on 46 patients that met criteria for 4 personality disorders (for example: borderline, narcissistic, histrionic, and antisocial), two-thirds diagnosed only one, a quarter diagnosed two, and no psychiatrist diagnosed all four (11).

Dimensional approach

An alternative approach to the DSM-IV/ICD-10 categorical method for diagnosing personality disorders is to adopt a dimensional model that depicts the patient's psychopathology as points on a series of fundamental dimensions of psychopathology. The five factor model (FFM) of personality is one externally validated dimensional system that has been proposed as an alternative to the DSM-IV categorical system (12,13). Rather than applying distinct criteria to distinguish 'case' from 'non-case' in each of

a series of personality diagnoses, the clinicians using the FFM rate patients on five major dimensions: neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. Within each dimension, there are six ‘facets’ on which a patient can be rated from ‘very high’ to ‘low’. By using the dimensional model, the FFM system is able to characterize patients in complex ways, without applying multiple labels that imply distinct disorders, each with their own presumed etiology, pathophysiology, and clinical course.

For example, a patient with psychopathology suggestive of borderline, antisocial, and narcissistic personality disorders in the DSM-IV system would instead be characterized as high in ‘neuroticism’ (with corresponding subscales reflecting propensity for anger, irritability, stress-tolerance, etc.), low in ‘agreeableness’ (antagonistic), and high in ‘openness to experience’ (exaggerated mood states, preoccupation with fantasy). Unlike the DSM-IV categorical system that would assign three personality disorder diagnoses to this patient, the FFM method avoids such comorbidity by providing instead a profile indicating patient’s place on the continuum of the FFM dimensions.

Furthermore, in contrast to the DSM categorical approach which can suffer from poor diagnostic reliability, especially in cases where patient’s personality disorder item count straddles the boundary between case and non-case (e.g., 4 or 5 out of 9 for borderline personality disorder), a personality formulation using the FFM is likely to be more reliable. However, the lack of discrete categories does not lend itself to the study of (ostensibly) distinct clinical populations, and also does not provide a straightforward answer to the question ‘does this patient have a personality disorder?’. Providing such a categorical answer to this question is important both for treatment planning (which often requires a categorical judgment of whether or not to treat the patient),

and for practical concerns such as determining eligibility for disability. Moreover, most health information systems (e.g. for clinical information or insurance) are not equipped to incorporate dimensional approaches.

Diagnostic hierarchies

Another diagnostic strategy that would reduce comorbidity, alluded to earlier in this article, is to impose additional diagnostic hierarchies. For example, consider the situation where a patient’s symptoms meet criteria for both disorder A and disorder B. In the DSM-IV, the patient would receive two diagnoses, A and B. However, if disorder B contained an exclusionary criterion that states it cannot be diagnosed if criteria are also met for disorder A, then only disorder A would be diagnosed. Exclusionary hierarchies are based upon the concept that a single pre-eminent disorder takes precedence over one or more subordinate diagnoses. The underlying assumption is that the symptoms of the subordinate diagnosis are associated features of the primary disorder and thus do not warrant an additional psychiatric diagnosis.

For example, a patient who has suffered from six months of auditory hallucinations combined with persecutory delusions in the absence of mood symptoms is assigned a diagnosis of schizophrenia. The six-month period of non-bizarre delusions also fits the pattern of delusional disorder. However, a diagnostic hierarchy that has schizophrenia take precedence over a diagnosis of delusional disorder prevents a comorbid diagnosis of delusional disorder from also being given. While this hierarchy has not been disputed, others have drawn more criticism, particularly with regard to the relationship between mood and anxiety disorders. Given the dissatisfaction with the excessive comorbidity of personality disorder diagnoses in the DSM, some have recommended that some axis II diagnoses, such as borderline personality disorder, take precedence over other subordinate

diagnoses, like dependent or histrionic personality disorder (14).

The problem with imposing diagnostic hierarchies is that they imply certain knowledge of symptom attribution that is at odds with the descriptive approach that is the cornerstone of the DSM. Even some of the remaining hierarchies, such as the exclusion of generalized anxiety disorder in the setting of comorbid major depressive disorder, have continued to draw criticism (15). While expanding the number of diagnostic hierarchies in DSM in order to eliminate all (or virtually all) co-occurring diagnoses would certainly reduce comorbidity, such steps would only be valid if we had an understanding of the etiologies of mental illnesses. Furthermore, such a radical reduction in the number of reported diagnoses risks losing clinically relevant distinctions in complex cases.

Mixed diagnostic categories

Another strategy that has been employed to address comorbidity has been the development of ‘mixed’ diagnostic categories, which lump together categories that have been separated in the DSM. Research has attempted to identify cases in which apparently co-occurring disorders (by our current diagnostic strategy) may actually reflect a single distinct diagnostic entity, with its own pathogenesis, treatment strategy, and prognosis (16,17). For example, Taylor et al (18) presented data on children referred to a clinic for disruptive or antisocial behavior. They identified that a subset that might otherwise have been characterized as having hyperkinetic syndrome and conduct disorder (by ICD-9 criteria) actually had distinct symptom onset, IQ ratings, neurological exam, and medication responsiveness when compared to the remaining clusters of patients. They used these findings to propose a new diagnosis of ‘hyperkinetic conduct disorder’ which would be added along with the existing conduct disorder and hyperkinetic categories. Another example includes the

suggestion to combine major depressive disorder and generalized anxiety disorder into a single category, called mixed anxiety/depression (19). One drawback of combining single categories into combined categories based on known co-occurring syndromes is that the number of possible combinations and permutations of categories (in a mathematical sense at least) could easily reach into the thousands.

Another way of combining separate categories that reduces diagnostic comorbidity is to lump categories together into higher order constructs. One example of a 'lumping' diagnostic strategy already in use in the DSM-IV is the combining of the 10 specific personality disorders into three personality disorder 'clusters' based on presumed common characteristics. Clinicians commonly incorporate these clusters into diagnostic formulations, such as 'personality disorder not otherwise specified, with cluster B traits', and researchers have used these mixed categories to delineate patient populations in studies of Axis I comorbidity, treatment responsiveness, and prognosis (20).

The co-occurrence of personality disorders within each cluster is generally higher than between-cluster comorbidity. For example, Stuart et al (9), using data collected in multiple sites on 1116 subjects, found that 73% of patients diagnosed with narcissistic personality disorder also met criteria for histrionic personality disorder, and many met criteria for a third and fourth diagnosis. Under the cluster system, comorbidity of personality disorders is reduced because instead of noting one, two, or three specific personality disorders from cluster B, the clinician would simply note 'cluster B personality disorder' regardless of the number of specific disorders actually present. However, currently, there is insufficient research to justify lumping the personality disorders into clusters for all diagnostic purposes. Furthermore, the widespread introduction of mixed categories whenever certain comorbid combinations are common is unwieldy, given the poten-

tially large number of combined categories that might result.

CONCLUSIONS

More research is needed into the etiology and interrelationship of psychiatric syndromes if we are to understand the full clinical significance of psychiatric comorbidity. In a seminal paper, Robins and Guze (21) posited that diagnostic validity could be improved through increasingly precise clinical description, delineation of syndromes, demographic and biological correlates, and treatment response profiles. Central to this concept was the assertion that empirical evidence would become the mainstay of psychiatric diagnosis. More than three decades later, however, our understanding of the etiology and pathogenesis of mental illness is still very limited. The research that will fully illuminate our understandings of mental illness is still several decades down the road.

As a field, we in psychiatry need to address the implications of our lack of understanding about pathophysiology on the use of our current diagnostic system. The 'atheoretical' approach of the DSM and ICD explicitly acknowledges our incomplete understanding and encourages multiple diagnoses with few exclusionary hierarchies in the hope that all clinically relevant information will be captured.

Unfortunately, this feature also makes the current system quite cumbersome to use as it was intended. Clinicians and health information systems intrinsically place certain information at a higher order of importance than other. Many information systems (especially in developing countries) do not have the capacity for incorporating all comorbidities. Individuals use clinical judgment to prioritize diagnoses, and may fail to account for diagnostic complexity where it exists. However, there is not much guidance for how clinicians make such determinations. As a result, the application of these systems in the real world has veered

away from consistency toward more idiosyncratic use. More research is needed if we as a field aspire to accuracy and uniformity in diagnosis. The ultimate goal is to increase the clinical utility of the DSM and ICD in providing better case-conceptualization, communication, and accuracy in prognosis (22).

In another strategy to limit comorbidity, the next editions of the DSM and ICD might add a provision that only those diagnoses that are clinically relevant and that are included as target symptoms in the current treatment plan (or which are useful with respect to prognosis, education, and treatment) should be listed. Again, how to make such differentiations in a consistent manner is not immediately clear and requires future investigation.

In summary, the current strategy of diagnosing 'maximal' comorbidity may not result in 'optimal' comorbidity in terms of best clinical practice. The practice of listing multiple diagnoses has the power to both enhance and obfuscate important clinical information. The next evolution of the DSM needs to balance the current 'rule-based' system with diagnostic strategies that depend on clinical judgment. As the DSM-V and ICD-11 are developed, opportunities for reducing comorbidity by 'lumping' diagnoses (e.g., replacing the 8 specific paraphilias with a single paraphilic disorder with subtypes to indicate specificity, e.g., 'paraphilia, pedophilic and sadistic type') or formalizing a convention for omitting diagnoses from the diagnostic list that are not clinically relevant, should be explored. However, as we move forward in addressing these challenges, we will need to provide explicit decision rules based on systematic assessment of the best data available, or risk reverting to the subjective and impressionistic formulations that were the state of the art fifty years ago with the DSM-I.

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References

1. Feinstein AR. The pre-therapeutic classification of co-morbidity in chronic disease. *J Chronic Dis* 1970;23:455-68.
2. Boyd JH, Burke JD Jr, Gruenberg E et al. Exclusion criteria of DSM-III: a study of co-occurrence of hierarchy-free syndromes. *Arch Gen Psychiatry* 1984;41:983-9.
3. Zimmerman M, Mattia JI. Psychiatric diagnosis in clinical practice: is comorbidity being missed? *Compr Psychiatry* 1999;40:182-91.
4. Zimmerman M, Mattia JI. Principal and additional DSM-IV disorders for which outpatients seek treatment. *Psychiatr Serv* 2000;51:1299-304.
5. Basco MR, Bostic JQ, Davies D et al. Methods to improve diagnostic accuracy in a community mental health setting. *Am J Psychiatry* 2000;157:1599-605.
6. Regier DA, Burke JD. Jr. Psychiatric disorders in the community: the Epidemiological Catchment Area Study. In: Hales RE, Frances AJ (eds). *American Psychiatric Association annual review*, Vol. 6. Washington: American Psychiatric Association, 1987:610-24.
7. Maser JD, Kaelber C, Weise RE. International use and attitudes toward DSM-III and DSM-III-R: growing consensus in psychiatric classification. *J Abnorm Psychol* 1991;100:271-9.
8. Widiger TA, Frances AJ, Pincus HA et al (eds). *DSM-IV sourcebook*, Vol. 4. Washington: American Psychiatric Press, 1998.
9. Stuart S, Pfohl B, Battaglia M et al. The cooccurrence of DSM-III-R personality disorders. *J Personal Disord* 1998;12:302-15.
10. Oldham JM, Skodol AE, Kellman HD et al. Diagnosis of DSM-III-R personality disorders by two structured interviews: patterns of comorbidity. *Am J Psychiatry* 1992;149:213-20.
11. Adler DA, Drake RE, Teague GB. Clinicians' practices in personality assessment: does gender influence the use of DSM-III axis II? *Compr Psychiatry* 1990;31:125-33.
12. O'Connor BP, Dyce JA. A test of models of personality disorder configuration. *J Abnorm Psychol* 1998;107:3-16.
13. Widiger TA, Costa PT. Jr, McCrae RR. A proposal for Axis II: diagnosing personality disorders using the five-factor model. In: Costa PT Jr, Widiger TA (eds). *Personality disorders and the five-factor model of personality* (2nd ed.) Washington: American Psychological Association, 2002:431-56.
14. Gunderson JG. Diagnostic controversies. In Tasman A, Riba MB (eds). *Review of psychiatry*, Vol. 11. Washington: American Psychiatric Press, 1992:9-24.
15. Zimmerman M, Chelminski I. Generalized anxiety disorder in patients with major depression: is DSM-IV's hierarchy correct? *Am J Psychiatry* 2003;160:504-21.
16. Caron C, Rutter M. Comorbidity in child psychopathology: concepts, issues and research strategies. *J Child Psychol Psychiatry All Discipl* 1991;32:1063-80.
17. Rutter M. Comorbidity: concepts, claims and choices. *Criminal Behav Ment Health* 1997;7:265-85.
18. Taylor EA, Schachar R, Thorley G et al. Conduct disorder and hyperactivity: I. Separation of hyperactivity and antisocial conduct in British child psychiatric patients. *Br J Psychiatry* 1986;149:760-7.
19. Tyrer P. The case for cothymia: an open verdict? *Br J Psychiatry* 2002;180:380-1.
20. Gude T, Vaglum P. One-year follow-up of patients with cluster C personality disorders: a prospective study comparing patients with 'pure' and comorbid conditions with cluster C, and 'pure' C with 'pure' cluster A or B conditions. *J Personal Disord* 2001;15:216-28.
21. Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *Am J Psychiatry* 1970;126:983-6.
22. First MB, Pincus HA, Levine JB et al. Using clinical utility as a criterion for revising psychiatric diagnoses. *Am J Psychiatry* (in press).

Disorders are different from diseases

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Pincus et al correctly point out that what is often called “the co-morbidity problem” is unavoidable, because it is simply a fact of life in clinical psychiatry. They provide a useful discussion of the topic, but the very use of the conventional term ‘co-morbidity’ serves to hide the real nature of the problem. This is because ‘morbid’ means disease, and to have a disease is conceptually very different from suffering from a disorder. Strictly speaking, the terms ‘diagnosis’ and ‘disease’ are both best avoided in psychiatric discourse unless they are completely justified. Clinical psychiatrists make few diagnoses in the sense of identifying known abnormalities which underlie the presenting symptoms. Instead, for most patients they have to make do with identifying disorders by assessing the number and severity of individually non-diagnostic symptoms from an agreed list. Most currently recognised disorders are no more than symptom clusters, and there is no particular reason why most patients should be expected to have only one of these. Viewed in this way, it is clear that it would be more honest for psychiatrists to use other terms, such as ‘co-existing disorders’ or ‘multiple disorders’.

On the basis of the points just made, it is natural to wonder why the inappropriate term ‘co-morbidity’ has become accepted usage. Probably it is a hang-over effect from the vitally important general medical training that all psychiatrists undergo, during which it is easy to develop the expectation that most patients have only one diagnosable disease. But things are different in psychiatry, and surely

it is best to use more realistic terms that are a constant reminder that our knowledge of the nature of psychiatric illnesses is rather superficial.

Two other associated issues are: first, the frequent lack of clarity in clinical work about the purposes for which the information is being recorded, and second, the special needs of researchers.

In the patient’s clinical case record, good practice requires that as many disorders should be recorded as are needed to describe the complete state of the patient, and this instruction is common to both ICD-10 and DSM-IV. In addition, clinicians should be encouraged always to give an order of priority for the disorders present, the reasons for this order, and the actions required by each disorder. If for some reason only one disorder can be recorded, it is up to those requiring the information to make clear the main purpose for which it will be used. Recording systems are now far more powerful than they were even a few years ago, and the old administrator’s plea of ‘no room on the form’ has become a weak excuse.

The needs of researchers are often different from those of clinicians, since most types of research involve the restrictive selection of groups of individuals whose symptoms and other characteristics resemble each other in clearly stated ways. Whether or not it is appropriate to include patients with more than one disorder will be determined by the type and purposes of the study, and so a more flexible approach to exclusion criteria is needed in research than is the case for ordinary clinical recording. This is why, for ICD-10, the Diagnostic Criteria for Research (1) are published separately from the Clinical Descriptions and Diagnostic Guidelines. While on this

topic of criteria for research, it needs to be pointed out that the comment of Pincus et al that “the ICD-10 diagnostic criteria for research were largely modelled on the DSM-III system” is a somewhat approximate précis of a long and complicated process. The many similarities between ICD-10 and DSM-IV in both general style and detailed content is the purposeful end result of an initiative started as long ago as 1980 by Gerald Klerman and Norman Sartorius, in the form of a ‘Joint Project’. The final manifestation of this was a series of meetings around 1990 between World Health Organization (WHO) advisors and the Chairpersons of the Task Forces for DSM-IV at which many harmonising changes were agreed to the drafts of both the classifications.

As a thought for the future, new ways of recording multiple disorders should be tried out as new versions of the classifications are developed, rather than leaving the process of recording as an afterthought to be worked out only when the classifications have been finished.

References

1. World Health Organization. The ICD-10 classification of mental and behavioural disorders. Diagnostic Criteria for Research. Geneva: World Health Organization, 1993.

The syndrome – an antidote to spurious comorbidity?

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Pincus et al identify correctly the proliferation of comorbid diagnoses in psychiatry as a by-product of the

current classification systems, especially since the abandonment of the hierarchical rules which had been introduced in DSM-III. If we adhere to the original definition of comorbidity as co-occurrence of clinically independent conditions, the reported prevalence of multiple comorbid psychiatric disorders - over a third of all cases in the population (1) - strains credibility and reflects adversely on the conceptual basis of the current classifications. Either the nature of psychiatric illnesses is such that they always tend to occur in clusters, or the diagnostic classification fails to discriminate between spurious comorbidity (mistaking facets of the same clinical entity for independent disorders) and true comorbidity.

The problem seems to be unique to psychiatry among the medical disciplines. An individual can, of course, harbour two or more diseases and, consequently, be a member of two or more categories in medical classifications, but such multiple membership can only be based on distinct and independent sets of characteristics. Current psychiatric classifications allow multiple category membership on the basis of the same set of data since their categories are not mutually exclusive. For example, an individual can meet the diagnostic criteria of both dysthymia and major depressive disorder on the basis of essentially the same symptoms, depending on their intensity, duration and sequence.

Part of the problem stems from the fact that DSM-IV and ICD-10 evade the difficult problem of defining the nature of the entities that are being classified, and instead adopt as the currency unit in psychiatric classification the term 'disorder' (first introduced in DSM-I in 1952), which has no clear correspondence with either the concept of disease or the concept of syndrome in medical classifications (2). The ambiguous status of the classificatory unit of 'disorder' has two corollaries which create conceptual confusion and hinder the advancement of knowledge: a) the 'reification fallacy' - the tendency to view the

DSM-IV and ICD-10 'disorders' as quasi-disease entities; and b) the fragmentation of psychopathology into a large number of 'disorders', of which many are merely symptoms. This blurs the distinction between true and spurious comorbidity, and masks the presence of complex but essentially unitary *syndromes*, such as Bonhoeffer's "exogenous reaction types" (3) or the recently revived "general neurotic syndrome" (4). It is not surprising, therefore, that 'disorders', as defined in the current versions of DSM and ICD, have a strong tendency to co-occur, which suggests that "fundamental assumptions of the dominant diagnostic schemata may be incorrect" (5).

In contrast, syndromes are basic concepts for most clinicians, and much of their clinical knowledge is cognitively stored in this format. These are good reasons for reinstating

the syndrome, as a "real-world correlational structure" in psychopathology (6), to its rightful place as the basic Axis I unit in future classifications.

References

1. Wittchen HU. What is comorbidity - fact or artefact? Br J Psychiatry 1996;168 (Suppl. 30):7-8.
2. Jablensky A, Kendell RE. Criteria for assessing a classification in psychiatry. In: Maj M, Gaebel W, López-Ibor JJ et al (eds). Psychiatric diagnosis and classification. Chichester: Wiley, 2002:1-24.
3. Bonhoeffer K: Zur Frage der exogenen Psychosen. Zentralblatt für Nervenheilkunde 1901;32:499-505.
4. Andrews G, Stewart GW, Morris-Yates A et al. Evidence for a general neurotic syndrome. Br J Psychiatry 1998;157:6-12.
5. Sullivan PF, Kendler KS. Typology of common psychiatric syndromes. Br J Psychiatry 1998;173:312-9.
6. Rosch E. Cognitive reference points. Cogn Psychol 1975;7:532-47.

State-of-the-art psychiatric diagnosis

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A major challenge for those revising the DSM-IV and ICD-10 will be to assess the feasibility of using pathophysiology, genetic vulnerability, and environmental exposure variables as components of diagnostic criteria. Our current use of clearly defined descriptive syndromes represented a major scientific advance in 1980, when the DSM-III was adopted as a national standard in the US, and subsequently was utilized as a model for the ICD-10, which was internationally adopted in 1992. Introducing explicit descriptive criteria, which could be reliably replicated by multiple clinicians, DSM-III replaced previous nosologies that relied on unproven theoretical formulations promulgated by leading psychi-

atrists in various countries or in psychoanalytic 'schools of psychiatry'. This seemingly modest advance in classification was supported by the demonstration that previous wide discrepancies in cross-national diagnostic practice could be readily reduced if common diagnostic criteria and assessment interviews were used (1). Given the absence of any etiological, anatomical, or pathophysiological criteria in DSM-III through DSM-IV over the past 25 years, the advances in research and clinical practice made possible simply by reliable criteria, independent of any validity indicators, are truly remarkable (2). A cumulative research field has developed on the basis that scientists and clinicians around the world use a common set of criteria and the same diagnostic language to describe the mental disorders they are studying and treating.

The current descriptive approach

affords great precision in differentiating specific syndromes (as symptoms that cluster together in patients seen in clinical practice or research settings). One consequence of this advantage, however, is an expansion in the number of diagnoses, some of which may be only slightly different expressions of common but currently unknown underlying pathological processes. Various critics of the current diagnostic system have characterized the expansion of diagnostic categories as a 'guild' attempt to justify payment for any condition that a psychiatrist might see in practice, or as fabrications of the pharmaceutical industry to justify the sale of their products (3,4). While one should never discount the power of financial incentives for driving human behavior, it would be difficult to demonstrate that such incentives were the driving force for the international adoption of the ICD-10 – a decision that required many countries to abandon disorders or classification systems that often were closely tied to strongly held nationalistic traditions (5). Rather, in the absence of clear etiological factors, anatomical abnormalities, or demonstrable pathophysiology, the advance from symptoms to syndromes to disorder or disease states is the scientific course that classification has taken in all fields of medicine (6).

As the scientific basis for psychiatric diagnosis and practice continues to expand, we should expect to find some of the multiple syndromes now defined as separate diagnoses to be linked to more parsimonious common underlying processes. Until the discovery of the spirochete, the multiple dermatological, peripheral nervous system, and central nervous system manifestations of syphilis confounded physicians relying on symptom-based syndrome diagnoses. However, when it was found that the multiple (co-morbid) syndromes of syphilis all responded to the same antibiotic treatment, the 'syndromes' were understood to be manifestations of different organ systems rather than discrete diseases. In psychiatry, we already have generated some preliminary evidence that the fre-

quently 'co-morbid' syndromes of generalized anxiety disorder and major depression appear to share a common genetic basis, with differences in expression possibly due to variations in environmental exposures (7). The finding that both disorders appear to respond to selective serotonin reuptake inhibitors (SSRIs) may eventually lead us to an understanding of common processes in an underlying genetically directed pathophysiology.

As we consider the possibility that the next revisions of the DSM and ICD might be able to combine a descriptive syndrome approach with information regarding genetic and environmental risk factors, a temporary increase in the number of diagnoses and co-morbidity rates is distinctly possible. This is illustrated most dramatically by the recent findings of Caspi et al (8), who demonstrated that seemingly minor differences in the serotonin transporter gene alleles were correlated with markedly different resilience or vulnerabilities to major depression, when exposed to equivalent stresses over a lifetime. Since major depression occurred in both genetic types, one could now make a case for further subdividing major depression into serotonin transporter short-arm and long-arm allele forms of the disorder.

As psychiatry's science base progresses to a better understanding of pathophysiology and etiology, we can expect greater predictive validity for our diagnoses and greater specificity for our treatments. We may well move from the aspirin-equivalent treatment for fever syndromes to the antibiotic-equivalent treatment for specific causal agents. However, as the neurosciences mature and our understanding of the interactions between brain and environment expands, we likely will find additional areas where malfunctions and pathophysiology in the brain are possible. One need only look at the infectious diseases, where new conditions such as severe acute respiratory syndrome (SARS) continue to emerge despite the maturity of microbiological science. Clearly, the development of a better classification system for all men-

tal disorders will be an uneven and iterative scientific process, in which etiological and pathophysiological hypotheses are tested before they replace the more conservative and atheoretical descriptive criteria that represent the current state-of-the-art.

References

1. Cooper JE, Kendell RE, Gurland BJ et al. *Psychiatric diagnosis in New York and London*. London: Oxford University Press, 1972.
2. Regier DA. Mental disorder diagnostic theory and practical reality: an evolutionary perspective. *Health Aff* 2003;22: 21-7.
3. Shorter E, Tyrer P. Separation of anxiety and depressive disorders: blind alley in psychopharmacology and classification of disease. *Br J Psychiatry* 2003;327:158-60.
4. First MB, Regier DA. Separation of anxiety and depressive disorders: new tools will lead to more valid classification system. *Br J Psychiatry* 2003;327:869-70.
5. Sartorius N, Jablensky A, Regier DA et al (eds). *Sources and traditions of classification in psychiatry*. Toronto: Hogrefe and Huber, 1990.
6. Aronowitz RA. When do symptoms become a disease? *Ann Int Med* 2001; 134:803-8.
7. Kendler KS. Major depression and generalized anxiety disorder. Same genes, (partly) different environments - revisited. *Br J Psychiatry* 1996;168(Suppl. 30): 68-75.
8. Caspi A, Sugden K, Moffitt TE et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 2003;301:291-3.

Comorbidity and Chairman Mao

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As Pincus et al observe, comorbidity among psychiatric conditions is rampant. This fact has led many authors to propose solutions for limiting the extent of comorbidity. Nevertheless, most of these solutions, such as the hierarchical exclusion rules enshrined in recent editions of the

DSM, are based more on 'common sense' or clinical lore regarding the presumed etiological relations among comorbid conditions than on data.

Most proposals for decreasing comorbidity place the cart before the horse. We still do not know which, if any, of the more than 300 conditions in the DSM-IV are dimensional as opposed to taxonic. By a taxon, we mean a category that exists in nature rather than solely in the minds of clinicians (1). Although some DSM-IV categories, such as schizophrenia (2), may be underpinned by genuine categories, others, like anorexia and bulimia nervosa (3), may merely represent the product of scientifically arbitrary cutting points on one or more continuously distributed personality traits (e.g., neuroticism, introversion) (4).

The concept of comorbidity may be meaningful only when discussing taxonic conditions (5). As Pincus et al note, Feinstein (6) defined a comorbid condition as a 'distinct additional clinical entity' that coexists with another condition. If comorbid conditions merely represent the confluence of extreme scores on one or more dimensions, they would be neither distinct nor qualitatively different from normal functioning. Moreover, the extent of comorbidity among dimensional conditions would be driven by scientifically arbitrary decisions, such as the cut-off points for demarcating pathology from normality.

Psychologists and statisticians have developed a number of useful methods for detecting and/or validating taxa underlying psychological disorders. These methods include the taxometric techniques developed by Meehl and his colleagues (7), admixture models (8), molecular genetic studies (9), and multivariate behavior genetic studies (10). Although none of these methods by itself can provide definitive confirmation of taxa, consistent findings of taxonicity across multiple methods offer converging evidence that a psychiatric condition is categorical at a latent level (11).

Most proposals for constraining comorbidity may serve only to mask a

fundamental problem with the DSM, namely the possibility that many of its categories reflect not true taxa but the intersection of high scores on continuous traits. Rather than impose hierarchical exclusion rules in the absence of compelling research evidence and thereby impose a premature 'band-aid' solution to widespread comorbidity, it may be preferable to, in Mao Tse Tung's words, "let a thousand flowers bloom" – that is, freely permit comorbidity to exist unless or until there is some strong empirically driven reason not to. Such an approach, although perhaps more confusing for clinicians, is consistent with the splitting preference embodied in recent editions of the DSM (12). In an early stage of scientific development, splitting is generally preferable to lumping given that the relation between splitting and lumping is asymmetrical. One can always split first and lump later if the etiological data indicate that two or more comorbid conditions should be housed under the same diagnostic roof; but once one has lumped it can be extremely difficult to split later. By minimizing comorbidity in the absence of data, we may never discover whether two conditions believed to be either isomorphic or closely related are actually "distinct", to use Feinstein's term.

Pincus et al note that dimensional models may meet with resistance from many clinicians. In part, this is probably because of 'categorical thinking': the pronounced tendency of humans to conceptualize the natural world in terms of categories even when such categories do not exist (13). The great American psychologist Gordon Allport observed that "the human mind must think with the aid of categories...We cannot possibly avoid this process. Orderly living depends on it" (14). Categorical thinking is typically adaptive, as Allport pointed out, but it often leads us to oversimplify the world. If studies demonstrate that most conditions in the DSM are dimensional rather than taxonic at an underlying level, we should revise our

psychiatric classification system to mirror that fact even if clinicians find it difficult to think in dimensional terms. The DSM should reflect the state of nature, not merely how clinicians think about the state of nature.

References

1. Meehl PE. Factors and taxa, traits and types, differences of degree and differences in kind. *J Personality* 1992;60:117-74.
2. Lenzenweger MF, Korfine L. Confirming the latent structure and base rate of schizotypy: a taxometric analysis. *J Abnorm Psychol* 1992;101:567-71.
3. Tylka TL, Subich ML. Revisiting the latent structure of eating disorders: taxometric analyses with nonbehavioral indices. *J Counsel Psychol* 2003;50:276-86.
4. Lilienfeld SO, Waldman ID, Israel AC. A critical examination of the term and concept of 'comorbidity' in psychopathology research. *Clin Psychol Sci Pract* 1994;1: 71-83.
5. Meehl PE. Comorbidity and taxometrics. *Clin Psychol Sci Pract* 2001;8:507-19.
6. Feinstein AR. The pre-therapeutic classification of co-morbidity in chronic illness. *J Chronic Dis* 1970;23:455-68.
7. Meehl PE, Golden RR. Taxometric methods. In: Kendall PC, Butcher JN (eds). *Handbook of research methods in clinical psychology*. New York: Wiley, 1982: 127-81.
8. Cloninger CR, Sigvardsson S, von Knorring A-L et al. An adoption study of somatoform disorders: II. Identification of two discrete somatoform disorders. *Arch Gen Psychiatry* 1984;41:863-71.
9. Waldman ID, Rowe DC, Abramowitz A et al. Association and linkage of the dopamine transporter gene (DAT1) and attention deficit hyperactivity disorder in children. *Am J Hum Genet* 1998;63: 1767-76.
10. Kendler KS, Neale MC, Kessler RC et al. Major depression and generalized anxiety disorder: same genes, (partly) different environments? *Arch Gen Psychiatry* 1992;49:716-22.
11. Waldman ID, Lilienfeld SO. Applications of taxometric methods to problems of comorbidity: perspectives and challenges. *Clin Psychol Sci Pract* 2001;8: 520-7.
12. Lilienfeld SO. Comorbidity between and within childhood externalizing and internalizing disorders: reflections and directions. *J Abnorm Child Psychol* 2003;31: 285-91.
13. Macrae CN, Bodenhausen GV. Social cognition: thinking categorically about others. *Ann Rev Psychol* 2000;51:93-120.
14. Allport, GW. *The nature of prejudice*. Reading: Addison-Wesley, 1954.

Different reasons for comorbidity require different solutions

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Pincus et al have provided a thoughtful discussion of the challenges posed by the high rates of psychiatric comorbidity observed using the current diagnostic classification system. In this commentary, I will elaborate on their discussion of alternative approaches to dealing with comorbidity: mixed diagnostic categories, diagnostic hierarchies, and dimensional systems.

Comorbidity can occur for a variety of reasons. It may be a chance occurrence or be due to the conjunction of independent risk factors; or it may develop because two disorders have shared or overlapping risk factors, or because one disorder causes the other; or the comorbid condition may be a multiform expression of one of the pure disorders, or a third independent disorder (1). It follows that the best approach for dealing with comorbidity depends on why a given pair of disorders tends to co-occur.

Mixed diagnostic categories are most appropriate when the comorbid condition is a third independent condition that is distinct from each of the two pure disorders. Otherwise, mixed categories may not be helpful. For example, if major depressive disorder and generalized anxiety have the same or highly overlapping etiologies, it would be more appropriate to combine them into a single higher order category than to add a third "mixed depression and anxiety" category to a system that already includes both of the pure categories.

More generally, lumping is the best approach when lower-order categories have shared or highly overlapping etiologies. However, it may be difficult to determine exactly how much overlap is necessary to warrant a lumping strategy. For example, it

appears that most common forms of psychopathology can be placed under the higher order rubrics of internalizing and externalizing disorders (2). Moreover, it is likely that the various internalizing disorders all have some etiological processes in common, as do the various externalizing disorders. However, the internalizing-externalizing distinction may be too coarse to guide treatment. Pincus et al's notion of clinical utility is relevant here. The question of whether to lump or to split must be based on whether the information lost by lumping is useful for prognosis, treatment, or understanding etiology.

A hierarchical approach is useful if the comorbid condition is a multiform expression of one of the pure disorders. For example, major depressive episodes are almost invariably a complication of dysthymia (3). Thus, there may not be any advantages to diagnosing a comorbid major depressive disorder in patients with dysthymia. Hierarchies may be also useful if one disorder causes the other, unless the second disorder has additional clinical implications after it develops.

Finally, although dimensional approaches have many useful properties (4), they generally complicate, rather than simplify, classification systems, unless the number of dimensions is very small. Rather than assigning one or more diagnoses to the patient, each patient receives a score on every dimension in the classification system. Even if scores of zero are ignored, patients will invariably receive more scores than they would receive diagnoses in a categorical system. While having more information is often useful, it increases, rather than reduces, complexity. As a result, many dimensional systems (e.g., the Minnesota Multiphasic Personality Inventory, MMPI) routinely impose categorical cutoffs on their dimen-

sions, bringing us back full circle to the problem of comorbidity.

The problems raised by psychiatric comorbidity cannot be resolved on an a priori basis. They require data on the nature of comorbidity between specific sets of disorders. As a result, there is no single solution to the problem of comorbidity. Rather, different solutions will be required for different combinations of disorders. As the nature of comorbidity is elucidated, it will undoubtedly lead to significant revisions in the categories and boundaries in the psychiatric nomenclature.

References

1. Klein DN, Riso LP. Psychiatric disorders: problems of boundaries and comorbidity. In: Costello CG (ed). *Basic issues in psychopathology*. New York: Guilford, 1993:19-66.
2. Krueger RF. The structure of common mental disorders. *Arch Gen Psychiatry* 1999;56:921-6.
3. Klein DN, Schwartz JE, Rose S. Five-year course and outcome of dysthymic disorder: a prospective, naturalistic follow-up study. *Am J Psychiatry* 2000;157:931-9.
4. Widiger TA, Clark LA. Toward DSM-V and the classification of psychopathology. *Psychol Bull* 2000;126:946-63.

Psychiatric comorbidity presents special challenges in developing countries

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The developments in the field of psychiatric diagnosis over the last 50 years have been significant, but the current phase has both advantages and disadvantages.

I was trained in the early 1970s, when the ICD-8 and the DSM-II were the operating diagnostic systems. Since then, we have moved to a more criteria based diagnosis. The advantages that I have seen with this change are the greater ease for sharing of information and teaching of postgraduates in psychiatry, and the increased number of diagnostic categories. However, there is a big loss in this shift, as most professionals, especially young psychiatrists, tend to use the diagnostic criteria with greater degree of confidence than is appropriate for the current level of knowledge, in spite of the ICD-10 clearly stating "No classification is ever perfect: further improvements and simplifications should become possible with increases in our knowledge and as experience with the classification accumulates"(1). The whole issue of validity and utility of classification is a subject of intense debate (2).

The question of comorbidity is linked to the current phase of evolution of diagnostic systems. Since the presence of a certain number of symptoms/signs forms the basis for the diagnosis, more than one diagnosis becomes possible in the same patient. The article by Pincus et al explores the complexity of the situation very well. I would like to comment on two issues: the needs of developing countries and the difficulties of classifying psychiatric comorbidity in primary health care.

The availability of mental health services in developing countries is very limited. In general, they are available in the range of 1/50 to 1/1000 of what is available in well developed countries. Consequently, the type of patients seen, the duration of contact and the service needs are different. There is greater use of services by people suffering from severe mental disorders, as illustrated by the very low frequency of the diagnosis of personality disorders in psychiatric centers of developing countries. Further, the contact is often cross-sectional for a particular episode of illness or during the acute phase of the illness. The

intervention provided is largely pharmacological, except in some centers where a wide variety of psychosocial treatments are used. These factors lead to the use of the most simple and obvious diagnostic categories. As a result, the identification of comorbidity gets low priority. Clinicians usually diagnose those conditions that can be effectively addressed at the level of available facilities. This situation will change as more and more facilities are created, especially as mental health care reaches the community.

Stimulated and supported by the World Health Organization (WHO), the integration of mental health care with primary health care is now occurring in several developing countries (3,4). In developed countries, the move to primary health care is a way of extending the reach of mental health services, while in developing countries it is the primary and often the only method of providing mental health care. The importance of this approach was reflected in the World Health Report 2001, whose first recommendation was "provide treatment in primary care" (5). This approach to organizing mental health care as part of primary health care has relevance to the discussion of comorbidity. It is well documented that comorbidity of mental disorders (especially of depression and anxiety disorders) is frequent in primary health care (6).

The ICD-10 primary health care version is a very simplified system that is considered suitable for primary care personnel. The twenty categories look simple compared to the full classification. However, experience in developing countries has shown that even this system of classification is difficult to use. The addition of comorbidity, however desirable, is not feasible with the current level of primary health care personnel (7). Moreover, psychiatrists in developing countries are currently making attempts to include psychiatric conditions into the general information system of primary health care. Due to the competing demands on the general information system, only a small

number, usually four to six, conditions can be accommodated. Here again the classification of comorbid conditions would not be feasible. If we insist for such a coding, the general health information system at the level of primary health care may exclude psychiatric conditions. Such an exclusion would be a big loss.

A different problem of greater relevance is the comorbidity of physical conditions with mental disorders. This is a frequent occurrence in developing countries (6,8). The usual practice of the primary care is to give greater importance to physical disorders. A major educational effort has to be directed to sensitize and provide skills to general physicians to identify the comorbid mental conditions.

In conclusion, the proliferation of diagnostic categories to meet the growing numbers seeking help for a wider variety of mental health problems is a valuable addition to the development of psychiatric classification. However, this desire to describe and categorise everything should not come in the way of integrating mental health care in general health services. The classificatory needs of the professionals working in developing countries should be considered before universalizing an exhaustive system of coding all comorbid conditions (9). Professionals who will be caring for the mentally ill need simpler diagnostic systems, which are easy to use and relevant to care rather than academic needs. In that context, the emphasis on coding all comorbid conditions may be premature. The desire for the best should not come in the way of common good.

References

1. World Health Organization. ICD-10 classification of mental and behavioural disorders. Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization, 1992.
2. Kendell R, Jablensky A. Distinguishing between the validity and utility of psychiatric diagnosis. *Am J Psychiatry* 2003; 160:4-12.
3. Srinivasa Murthy R. Reaching the unreached. *Lancet* 2000;356:39.
4. Cohen A. The effectiveness of mental

- health services in primary care: view from the developing world. Geneva: World Health Organization, 2001.
5. World Health Organization. World Health Report 2001: mental health - new understanding, new hope. Geneva: World Health Organization, 2001.
 6. Ustun TB, Sartorius N. Mental illness in general health care: an international study. Chichester: Wiley, 1995.
 7. Srinivasa Murthy R. Experience on psychiatric classification in South Asia primary care facilities. In Mezzich JE, Hon-da Y, Kastrup M (eds). Psychiatric diagnosis - a world perspective. New York: Springer, 1995:311-6.
 8. Okasha A. Epidemiological data: focus on comorbidity of depression and organic chronic illness. WPA Bulletin on Depression, Special issue, 2003: 24-5.
 9. Srinivasa Murthy R, Wig NN. Psychiatric diagnosis and classification in developing countries. In Maj M, Gaebel W, Lopez Ibor JJ et al (eds). Psychiatric diagnosis and classification. Chichester: Wiley, 2002:249-79.

Comorbidity: the African perspective

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Comorbidity in the developing world is a poorly researched harsh daily reality that compounds diagnostic problems at many levels. Allan German (1) described a group of people in Uganda as being in a state of subclinical malnutrition. In this group any insult, either physical or psychological, led to very severe forms of affliction. It is upon subjects of this level of weakness that a variety of mental disorders are superimposed. In rural Africa, over and above the AIDS pandemic, the people have to struggle with traditional infections, including typhoid fever and tuberculosis. Indeed, in many parts of Africa, psychosis is called 'the big malaria', a term that recognises the link between fever and psychosis. Epilepsy due to trauma at birth and head injury in later life is common in Africa. Childhood infections, including measles, are other common causes of epilepsy, a condition that is comorbid with mental illness in some cases.

Pincus captures the dilemma of the developing world most graphically when he states "Many information systems (especially in developing countries) do not have the capacity for incorporating all comorbidities.

Individuals use clinical judgment to prioritize diagnoses, and may fail to account for diagnostic complexity where it exists".

The pressure becomes intense where the ratio of doctors to the population is 1:20,000, whereas that of the influential traditional healer is 1:25. In Tanzania, with a population of 30 million, there are only ten psychiatrists, four of whom work in the capital city (2). In these circumstances, the purpose and utility of remaining faithful to classification systems that have no bearing on the available interventions becomes the subject of legitimate debate. After all, goes the argument, classification systems exist, at least in part, to inform treatment. With the currently available numbers of mental health workers (psychiatrists and nurses), it seems futile to engage in detailed systems of classification.

Recent correspondence in the British Journal of Psychiatry (3) acts to fuel the sense of hopelessness experienced by lonely African scholars, who live without the luxury of intellectual communion with their peers. When Tyrer (3) proposes a trial marriage between depression and anxiety rather than a life of indefinite nosological sin, the African is left wondering how interesting the debate would have been in real life! It however remains clear, at the end of the debate,

that nothing is clear beyond the fact that a system of classification, however imperfect, must remain in place.

Many questions arise from this conclusion, including that of the complicating role played by social cultural factors in the causation and perpetuation of common psychiatric disorders – poverty, internal and external displacement of people, refugee status, malnutrition and anaemia are but a few common conditions that could influence the way disease/health might be defined and classified. These considerations compound any attempt at clarifying the question of comorbidity.

As though to complicate things further, the majority of sub-Saharan countries do not have a mental health policy. A chicken and egg situation. In the absence of mental health policies, African governments do not see mental health as a priority issue worthy of funding. Kenya and Tanzania are good examples of countries in sub-Saharan Africa currently engaged in the development of mental health policy after the successful completion of baseline surveys of psychiatric morbidity in rural areas (4). During this process, it has become clear that comorbidity is a matter of concern at two levels. Firstly, the primary health care workers do not have the skills of identifying different psychiatric disorders. For this reason, the guidelines developed for the health information systems at primary care can only consist of a few disorders (eleven in Kenya), if a meaningful information system is to be planted in the country, in the hope that in time it could develop to the full ICD-10/DSM-IV level of complexity. In this way, Kenya recognises the importance of making the first step in this long journey towards harmony in classification systems.

Legitimate questions then arise from this decision. If there are only a few conditions recognised in this (most modern) African system, what is the scientific value of communication arising from Africa? Is research data from Africa to be respected/

trusted? Are Africans in fact talking the same language with the rest of the world when they discuss the challenges of comorbidity? Within the body of this questions lies the answer. Even as the Africans struggle to develop mental health policies, and even as they seem to be developing a parallel system, this is the reason for greater collaboration with the rest of the world.

The mental health policy support project in Kenya and Tanzania is funded by the British government through the Institute of Psychiatry in London, and with it have come many lessons and challenges. The project has exposed a critical mass of African

scientists to the discipline of research, and the need to remain faithful to international systems of classification, while at the same time remaining grounded on the harsh reality of poor resources at their disposal, a fact that must drive their creativity.

Another challenge to the Africans considering comorbidity is the question of the prevalence of some psychiatric disorders common in the West, rare in Africa. These include anorexia nervosa and obsessive-compulsive disorder. Do they exist in a different form? Are they comorbid with other disorders? The issues raised by Pin-cus demand greater collaboration between the developing world and

those responsible for harmonising classification systems.

References

1. German GA. Aspects of clinical psychiatry in sub-Sahara Africa. *Br J Psychiatry* 1972;121:461-79.
2. Ngoma MC, Prince M, Mann A. Common mental disorders among those attending primary health clinics and traditional healers in Tanzania. *Br J Psychiatry* 2003;183:349-55.
3. Tyrer P. The case for cothymia: an open verdict? Author reply. *Br J Psychiatry* 2001;180:380-81.
4. Njenga FG, Kigamwa P, Okonji M. Africa. The traumatised continent. The continent with hope. *Int Psychiatry* 2003;1:4-7.

The antisaccade task and neuropsychological tests of prefrontal cortical integrity in schizophrenia: empirical findings and interpretative considerations

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To date, every published study of the antisaccade task has replicated the finding that schizophrenia patients make an increased number of errors. This finding has been interpreted as support for frontal and/or basal ganglia dysfunction in schizophrenia, primarily because neurological patients with pathology in these brain regions also make large numbers of errors on the antisaccade task. Here, we compared the performance of schizophrenia patients and nonpsychiatric controls on an antisaccade task and on two neuropsychological tests, the Wisconsin Card Sorting Test, which is assumed to tap frontal lobe functioning, and the interference condition of the Stroop Test, which is thought to tap dorsolateral prefrontal cortex/anterior cingulate functioning. We examined the pattern of intercorrelations among these tasks. Schizophrenia patients made significantly more errors on the antisaccade task, made more perseverative errors and achieved fewer categories on the Wisconsin Card Sorting Test, and were significantly slower during the interference condition of the Stroop Test than were nonpsychiatric controls. Antisaccade errors were significantly correlated with interference performance on the Stroop in schizophrenia patients and in controls, but were not significantly correlated with the measures of Wisconsin Card Sorting Test performance in either group. The pattern of intercorrelation suggests that these tasks should not be thought of as representing a unitary variable of “frontal cortical integrity”. Although aspects of these tasks tap the ability to inhibit prepotent responses, each task is also behaviorally complex. The multifaceted nature of these tasks makes it difficult to isolate which brain regions are part of the network underlying the specific act of inhibiting a prepotent response (for example, the reflexive saccade toward the novel peripheral target) and which regions participate in aspects of task performance that are related to non-inhibitory components (for example, executing an antisaccade). A broadly distributed network is undoubtedly involved in both processes. Parsing the various components of cognitively complex tasks may help to clarify both the specific behaviors that are anomalous and their underlying neural substrates. We also address the complexity of inferring localized brain dysfunction in schizophrenia patients based on seemingly analogous behavioral deficits in neurological populations.

Key words: Antisaccades, schizophrenia, frontal cortex, neuropsychological tests, response inhibition

Every study of antisaccade (AS) performance in schizophrenia (SZ) patients has replicated the finding that they make an increased number of errors on the AS task (1-35). The consistency of this finding has led investigators to pursue its significance by studying clinically unaffected relatives of SZ patients (36) and to explore the neural correlates of poor performance.

In the AS task (37), the subject fixates a central target. A novel visual stimulus appears unpredictably in the left or right periphery, and the task is to inhibit a reflexive saccade toward the target and then to make a voluntary saccade to the opposite periphery. Failure to inhibit a reflexive saccade toward the peripheral target is considered an error.

The poor performance of SZ patients on the AS task has been interpreted as support for frontal or basal ganglia dysfunction, or both, because patients with lesions involving the dorsolateral-mesial frontal lobes (38) as well as patients with lesions of the dorsolateral prefrontal cortex (DLPFC) (4,39), Huntington's disease (40-42), progressive supranuclear palsy (43,44), and Parkinson's disease (45-48; but see 4, 49, 50) also make an increased number of errors on the AS task. In addition, broadly disseminated brain disease, including Alzheimer's disease (51,52), amyotrophic lateral sclerosis (53), and AIDS dementia (54,55), is associated with impaired AS performance.

The possibility that the poor performance of SZ patients on the AS task reflects prefrontal cortical dysfunction has led investigators to examine the relation between performance on this task and other tasks that are thought to tap the functional integrity of the prefrontal cortex. Significant correlations between measures of performance on the various tasks have been interpreted as converging support for varying degrees of “frontal lobe dysfunction” in SZ patients. Rosse et al (6) found that AS errors were significantly correlated with perseverative errors and number of categories achieved on the Wisconsin Card Sorting Test (WCST) in chronic SZ patients. Among nonpsychiatric controls, performance on the two tasks was not significantly correlated. Crawford et al (9) reported that the number of perseverative errors on the WCST was a significant predictor of AS errors in SZ and bipolar patients. Tien et al (14) found a trend for perseverative errors on the WCST to be significantly related to AS errors in SZ patients. In another study (17), AS errors were significantly correlated with perseverative errors on the WCST in controls and with categories achieved in SZ patients. Performance on both parts of the Trail Making Test was significantly correlated with AS errors in SZ patients, but not in controls. Two other tests of frontal lobe functioning, finger tapping and the Rey Auditory Ver-

bal Learning Test, were not significantly associated with AS performance in either group. Karoumi et al (20) found that WCST errors were significantly correlated with AS errors in SZ patients but not in nonpsychiatric controls. In contrast, performance on the Stroop was not correlated with AS errors in either group.

In a study by Snitz et al (27), categories achieved on the WCST and errors on a spatial working memory task were significantly correlated in SZ patients and controls, but perseverative errors on the WCST and spatial working memory were correlated with each other only in the controls. AS errors were not correlated with spatial working memory performance in either group (no results were presented for the relation between AS errors and performance on the WCST). The authors noted that the subgroup of SZ patients who performed poorly on the AS and spatial working memory tasks also had worse smooth pursuit and tended to achieve fewer categories on the WCST than other SZ patients in the sample. Nieman et al (29) reported that AS errors were significantly correlated with poor working memory, poor spatial working memory, and attentional disturbance in SZ patients. AS errors were significantly correlated with performance on the Stroop and the Trail Making Test in SZ patients treated with olanzapine, but not in patients treated with risperidone. Gooding and Tallent (33) found a significant correlation between AS errors and performance on a test of spatial working memory in SZ outpatients (and in the total sample of SZ and bipolar patients and controls). Manoach et al (34) found that AS errors were not significantly correlated with perseverative errors on the WCST.

As the summary above indicates, there is no consistent pattern to the relationship between performance on the AS task and various neuropsychological tests of frontal lobe functioning. The purposes of the present study were: a) to study independent samples of patients and controls, both of whom were tested on the AS task and on neuropsychological measures of frontal cortical integrity, and b) to address issues raised by inferring localized brain dysfunction in SZ patients on the basis of deficits seen in patients with neurological disorders.

METHODS

Subjects

The two subject groups included 23 individuals who met DSM-IV criteria for a diagnosis of schizophrenia or schizoaffective disorder (SZ) and 21 nonpsychiatric control (NC) subjects. These groups are subsets of larger samples of SZ patients and NC whose AS data were previously published (35). SZ participated an average of 9.6 (SD=2.7) months after their most recent psychiatric hospitalization. Their average duration of illness was 14.4 years (SD=7.4) and their average score on the Brief Psychiatric Rating Scale was 43.2 (SD=13.2), indicating symptoms of moderate severity at the time of testing. Two SZ patients were receiving no psychotropic medications.

One patient was taking antianxiety medication only, two were receiving mood stabilizers only, and 18 were taking antipsychotic drugs. All but two of these eighteen patients were also receiving antianxiety, antidepressant and/or mood stabilizing medication. The average daily dose of antipsychotic medication in chlorpromazine equivalents was 423 mg (SD=312.4). Nonpsychiatric control subjects were recruited through the Department of Internal Medicine at a local medical center. Written informed consent was obtained after a complete description of the study.

General inclusion criteria that applied to all subjects included: a) age between 18 and 55; b) no diagnosed central nervous system disease; c) no current or past substance abuse or dependence within one year; d) estimated verbal IQ ≥ 80 based on the vocabulary subtest of the Wechsler Adult Intelligence Scale - Revised; e) fluency in English; f) no tardive dyskinesia.

All subjects received the Structured Clinical Interview for DSM-IV, Patient Edition (56), which was administered by one of four trained interviewers. Nonpsychiatric controls also received the Structured Interview for Schizotypal Symptoms (57). Axis I syndromes and Axis II schizophrenia spectrum personality disorders meeting DSM-IV criteria were assigned based on a consensus of four clinicians who reviewed the interview material as well as a narrative summarizing the interview and records of previous psychiatric hospitalizations. Interviews and the diagnostic assessments were performed without knowledge of group membership or the experimental findings.

NC were excluded if they met DSM-IV criteria for a diagnosis of any psychotic disorder (lifetime), bipolar disorder without psychotic features, or schizoid, schizotypal, or paranoid personality disorder, or if they had a family history of psychosis as determined from a Family Informant Schedule and Criteria interview (58). Six NC were diagnosed as having had a past major depression and one received a diagnosis of panic disorder without agoraphobia. Although the NC group was not free of Axis I disorders, none had a psychotic or schizophrenia-related condition or was a relative of a psychotic individual. We therefore consider the controls to be "clinically unaffected" (i.e., not to have a schizophrenia-related condition), but not to be a "purified" control group (36, 59-62).

The SZ group had a significantly smaller proportion of females than did the NC group (43% vs. 81%, chi square = 5.02, df = 1, $p = 0.025$). The two groups did not differ in age (36.5 ± 7.3 vs. 37.5 ± 11.5 years) or estimated verbal IQ (104.3 ± 11.9 vs. 106.0 ± 12.7).

Procedures

Antisaccade task

Horizontal eye movements were recorded in a darkened room using infrared reflectometry. Signals from photo-detectors were digitized at 1 KHz. The sensors and miniature infrared light emitting diodes, which were mount-

ed on spectacle frames, were aligned for each subject. Head movement was minimized by a custom bite-bar of each subject's dental impression. Eye position was calibrated in 3° intervals to +/- 12°. A vertical signal detected blinks. Calibrations were performed at the beginning of a testing session and again after any interruption in which the subject removed his/her mouth from the bite-bar. Stimuli were presented on a color monitor positioned 22 inches in front of the subject. Data were collected and analyzed using customized software.

The target was a pink filled circle subtending a visual angle of 1.5°. One cycle consisted of the target positioned at the center of the screen for 2000 msec, followed by its abrupt disappearance and the simultaneous appearance of the target at 8° left or right of center for 2000 msec, followed by a return to center, signaling the beginning of the next cycle. The target moved to the left or right quasi-randomly and each direction was presented an equal number of times. Subjects received two practice trials of four cycles each, and two test trials of eight cycles each. Each set of eight trials lasted 32 seconds, separated by a short break. The first trial of each set was discarded, yielding 14 trials that were included in the analyses. Subjects were instructed to look at the target when it appeared at the center of the screen, and then to look as quickly as possible, approximately the same distance from center, in the direction opposite to that of the target movement. Thus, when the target moved to the right, subjects were to look the same distance to the left of center. When the target returned to center, subjects were instructed to follow it back to the center. The following three dependent measures were used: a) the percent (%) error score, calculated as the number of errors/number of valid cycles (an error was counted if the subject looked toward the target instead of to the opposite periphery); b) the symmetry of % error scores (the difference between AS errors to targets appearing on the left and right); c) the % corrected error score, calculated as the number of corrected errors/total number of errors. An error was considered corrected if a saccade toward the peripheral target was followed by an AS to the correct location before the target returned to center. A cycle was considered invalid and eliminated from analysis if a saccade was made just before the target moved (an anticipatory eye movement) or if a blink occurred just before or just after the target moved.

Neuropsychological tests

Two tasks that are commonly thought to be sensitive to the integrity of the frontal cortex (63-65) were administered: the WCST and the Stroop Color-Word Interference Test.

The WCST (66) was administered according to the method proposed by Milner (63). Subjects were presented with a deck of 64 cards and asked to match each test card to one of four stimulus cards. A second deck of 64 cards was administered after the sorting principles were clarified to ensure that sorting errors were not a function of a fail-

ure to comprehend the task (65). The two dependent variables were number of perseverative errors and number of sorting categories achieved on the second deck.

The Stroop Test (67,68) is designed to establish competing response tendencies and to assess ability to suppress interfering stimuli. The task has three parts: a) Color Naming (reporting the color of randomly sequenced color rectangles, each rectangle printed in one of four different colors: red, blue, green, or yellow), b) Word Reading (reading randomly ordered color words printed in black ink), and c) Interference, INTF (reporting the ink color of color words that are printed in an incongruous color, e.g., the word *blue* printed in red ink). The Color Naming conditions the tendency to respond to color and the Word Reading conditions the tendency to read words. The INTF condition requires that the subject inhibit the overlearned and competing response set to read the color word, and instead, to name the color in which the word is printed. The effect of the INTF condition is the time difference between the color naming and INTF conditions.

Statistical methods

Since none of the dependent measures was normally distributed, group comparisons were based on the Wilcoxon Rank Sum Test (WZ). Symmetry of % error scores within groups was tested using the Wilcoxon Signed Rank Test (WT).

RESULTS

Antisaccade task

SZ made significantly more errors than did NC (WZ = -2.0, $p = 0.04$). Neither group showed significant asymmetry in % error score (NC: WT = 25, $p > 0.33$; SZ: WT = -9, $p > 0.71$) and there was no group difference in symmetry of errors (WZ = -0.80, $p > 0.40$). All subjects corrected virtually all errors and there was no group difference in this respect (WZ = 1.36, $p = 0.2$). Error correction averaged 94.6% in SZ and 100% in NC. Descriptive statistics pertaining to the dependent measures can be found in Table 1.

Neuropsychological tests

SZ achieved significantly fewer categories (WZ = 2.82, $p = 0.005$) and had a significantly higher number of perseverations (WZ = -3.1, $p = 0.002$) on the WCST than did NC. The Stroop INTF condition had a significantly greater effect in the SZ patients than in the NC (WZ = -2.4, $p = 0.02$).

Relationship between performance on the antisaccade task and neuropsychological tests

In both SZ patients (Spearman $\rho = 0.45$, $p = 0.04$, $n = 21$) and NC (Spearman $\rho = 0.49$, $p = 0.03$, $n = 20$), AS errors were significantly correlated with the Stroop INTF score. In contrast, AS errors were not significantly corre-

Table 1 Antisaccade task and neuropsychological test results in patients with schizophrenia and nonpsychiatric controls

| | Antisaccade % error score* | Perseverative errors (WCST)** | Categories achieved (WCST)*** | Stroop Interference score (sec)**** |
|-----------------|--|----------------------------------|----------------------------------|---|
| Patients (N=23) | 45.1 ± 30.2 [50.0] R: 46.6 ± 34.6 [50] L: 42.8 ± 30.3 [33.3] | 6.6 ± 6.8 [4] (N=22) | 3.7 ± 1.4 [4] (N=22) | 75.7 ± 43.6 [61] (N=21) |
| Controls (N=21) | 25.7 ± 21.3 [21.4] R: 23.7 ± 21.6 [12.5] L: 28.6 ± 27.0 [16.7] | 2.5 ± 3.9 [1.5] (N=20) | 4.8 ± 0.56 [5] (N=20) | 50.6 ± 13.8 [49.5] (N=20) |

Results are expressed as mean ± SD [median]

WCST – Wisconsin Card Sorting Test; R – Targets appearing to the right of center; L – Targets appearing to the left of center

* Significantly more errors in patients than controls (WZ = -2.0, p = 0.04)

** Significantly higher number of perseverations in patients than controls (WZ = -3.1, p = 0.002)

*** Significantly fewer categories achieved by patients compared to controls (WZ = 2.82, p = 0.005)

**** Significantly greater effect of the Interference condition in patients than controls (WZ = -2.4, p = 0.02)

lated with categories achieved (SZ: Spearman rho = -0.14, p = 0.54, n = 22; NC: Spearman rho = 0.17, p = 0.47, n = 20) or perseverative errors on the WCST (SZ: Spearman rho = 0.10, p = 0.65, n = 22; NC: Spearman rho = 0.19, p = 0.43, n = 20) in either group. The same pattern of results was found when all subjects were considered together: AS errors were significantly correlated with the INTF condition of the Stroop (Spearman rho = 0.53, p = 0.0003, n = 41), but not with categories achieved (Spearman rho = -0.11, p = 0.48, n = 42) or perseverative errors (Spearman rho = 0.27, p = 0.08, n = 42). In each group and in the combined groups perseverative errors were significantly correlated with categories achieved on the WCST (SZ: Spearman rho = -0.70, p = 0.0003, n = 22; NC: Spearman rho = -0.57, p = 0.009, n = 20; combined: Spearman rho = -0.71, p = 0.001, n = 42). The only other significant correlations were between the Stroop INTF condition and both categories achieved (Spearman rho = -0.61, p = 0.003, n = 21) and perseverative errors (Spearman rho = 0.52, p = 0.01, n = 21) in the SZ group and in the combined sample (Spearman rho = -0.52, p = 0.0006, n = 40; Spearman rho = 0.54, p = 0.003, n = 40, respectively).

DISCUSSION

There are two noteworthy findings in the present study. The first pertains to the replications of the AS, WCST, and Stroop findings in SZ. The second concerns the relation of AS errors to performance on the two neuropsychological tests. In the discussion that follows we address each of these findings and the nature of the deficit represented by poor AS performance in SZ patients, and we compare the characteristics of the poor performance of SZ patients with those of patients with frontal pathology of neurological origin.

Schizophrenia, antisaccade errors, and neuropsychological test performance

We found that SZ patients made significantly more errors (45.1%) on an AS task than did NC (25.7%), repli-

cating all other reports in the literature (see introduction). SZ patients and NC corrected 94% and 100%, respectively, of errors. All of these results on the AS task are consistent with those reported for the larger sample from which these subgroups were drawn (35). No asymmetry in the error score was noted within either group and the groups did not differ in magnitude of asymmetry. The larger samples of SZ patients (n = 50) and NC (n = 49) from which these subgroups were drawn also showed no significant asymmetry (SZ: 1.7 ± 29.0; NC: 5.3 ± 22.5; WZ = 0.54, p > 0.59). Nor was significant asymmetry found in 98 first-degree relatives of SZ patients (-1.7 ± 24). Of the 11 studies of SZ patients that examined asymmetry in error rate (1,2,4,7-9,13,15,16,20; the present study), asymmetry in error rate in SZ patients was reported in only two (8,13).

The neuropsychological test results in the present study are also consistent with an extensive literature documenting poorer performance in SZ patients than in controls. Specifically, on the WCST, SZ patients made more perseverative errors and/or achieved fewer categories than controls (34,69-73). On the Stroop, the INTF condition had a significantly larger effect in SZ patients than in controls (74-78). Of interest here, however, is the interrelationship among these measures. The two measures of the WCST were highly correlated with each other (rho = 0.71) as well as with the Stroop INTF (rho = -0.52 for categories achieved; rho = 0.54 for perseverative errors) in the combined groups. Neither of the WCST measures, however, was significantly correlated with the AS error score in either group, even though each measure individually showed dysfunctional performance in the SZ group. The INTF score of the Stroop, on the other hand, was significantly correlated with the AS score (rho = 0.53) accounting for 28% of the variance.

Relation between antisaccade task and neuropsychological test performance

If the INTF condition of the Stroop, perseverative errors and categories achieved on the WCST, and inhibi-

tion of reflexive saccades on the AS task all reflect a common process related to frontal cortical dysfunction, one would expect to find a pattern of significant intercorrelations among all these variables. This outcome was not obtained in this sample, which is consistent with some of the other findings in the literature but not with others (see introduction).

Phenomenologically, one might infer that the common process reflected in the significant association between the % error score and the INTF effect is inhibition of prepotent responses. However, the inconsistent pattern of associations between performance on the AS task and the various neuropsychological test measures of prefrontal cortical integrity suggests that neither the phenomenology of these tasks nor the concept of 'frontal cortical integrity' reflects a unitary process. For example, the perseverative error score would also seem to reflect the ability to inhibit prepotent responses and, although it was correlated with the INTF effect, it was not correlated with % errors in SZ or in the entire sample. The number of categories achieved on the WCST is a secondary consequence of inhibiting competing response tendencies (i.e., not perseverating) and it, too, was correlated with the INTF effect, but not with % errors in patients or in the combined sample.

What is reflected by poor antisaccade task performance?

One factor that may contribute to the unsystematic pattern of intercorrelations among these tasks (both in this study and in others in the literature) is that they are all cognitively complex. Each task requires that the participant follow a complex rule, which mandates that a sequence of behaviors be performed correctly. The ability to inhibit prepotent responses is only one of several behaviors required to perform each task correctly. Consider the AS task as an example. At a minimum, it requires the ability to fixate a target, to inhibit a reflexive saccade toward the novel target, to generate a voluntary eye movement (the antisaccade) to the opposite periphery, to perform all three behaviors in a sequence, to sustain attention, to remember the task requirements, to maintain task set, and to do all of these behaviors repeatedly. This complexity is mirrored in the results from magnetic resonance imaging and electrophysiological studies in human and non-human primates, where networks that involve many brain regions are implicated in the *global* performance of the AS task; these include significant activation of frontal eye fields, supplementary eye fields, supplementary motor area, posterior and superior parietal cortex, anterior cingulate, DLPFC, insula, basal ganglia, thalamus, and superior colliculus (79-92). Which brain regions are critically involved in any specific feature of performing the AS task, and of performing these actions sequentially, and repeatedly, however, is not currently well understood. For example, although the DLPFC has been implicated in saccade inhibition in studies of clinical populations and in studies

of lesioned non-human primates (38, 39, 93), imaging studies have not consistently confirmed changes in activation in this region while performing the AS task (see above). Similar inconsistencies have been reported for the frontal eye fields and supplementary eye fields, both in studies of clinical populations and in functional imaging studies (38,39,81,83,94-97). Moreover, the anterior cingulate cortex has been implicated in inhibiting prepotent responses in several behavioral domains (96,98). It is instructive here to consider that in two tasks as simple as lifting the finger that is touched compared with lifting a specified finger other than the one that is touched, the regions of brain activation differ: the latter activates the anterior cingulate and the DLPFC but the former does not (99). It is therefore not unexpected that the much more cognitively complex AS, Stroop INTF, and WCST tasks would reflect complex activation patterns in the brain.

The antisaccade task: frontal localization revisited

In the discussion that follows, we revisit the interpretation that the AS task indexes frontal dysfunction. Similar considerations could undoubtedly be raised about the WCST and the Stroop task as well as any behaviorally complex tasks, but here we focus on the AS task. There are two particularly influential reports on AS performance in neurological populations, the studies by Guitton et al (38) and Pierrot-Deseilligny et al (39), both of which provide unusually rich sources of information about the oculomotor and clinical behavior in their respective patient populations. It is useful to examine the extent to which the patterns of abnormal AS performance described in those two reports resemble the poor performance of SZ patients on the AS task, because the similarity in performance deficits between neurological patients with frontal pathology and SZ patients has been the basis for inferring localized brain dysfunction in SZ.

Guitton and colleagues administered the AS task to ten patients two weeks after tissue had been removed unilaterally from parts of the patients' dorsolateral-mesial frontal cortex for relief of intractable epilepsy (seven additional frontal patients were tested 1-23 years post-operatively). These ten acutely post-operative "frontal" patients were compared with seven patients who had had "discrete temporal lobe removals" two weeks previously, also for intractable seizure relief, and with nine neurologically healthy controls. Only the patients with probable lesion sites involving the frontal cortex performed abnormally on the AS task. The authors described "specific and non-specific effects of frontal lesions". They attributed the ability to inhibit reflexive saccades - the AS error - to the integrity of dorsolateral and mesial frontal areas, and they attributed the ability to make a correct AS to the integrity of the frontal eye fields and supplementary motor area.

Guitton et al (38) described three patterns of poor AS performance. The first pattern was found in four patients

with probable lesions involving the frontal eye fields and/or supplementary motor area and involved strong bilateral deficits. Error rates ranged from about 66 to 100% regardless of whether the cue was presented ipsilateral or contralateral to the side of the lesion. The principal aspects of this pattern were: a) profoundly impaired ability to inhibit reflexive saccades to the target, b) rare correction of reflexive saccade errors, c) hypometric AS - on the isolated occasions when patients made an AS, it stopped at the midline instead of at the mirror image location in the opposite periphery, and d) when patients did look at the correct location for the AS, it was always after that location had been illuminated; that is, they made a visually-guided saccade, not a voluntary saccade. It is noteworthy that not all patients with extensive prefrontal involvement (including the DLPFC) showed an increase in reflexive glances toward the target. For example, one patient who was tested 23 years after “almost total removal of a prefrontal lobe” could suppress reflexive glances “better than normal”, but could not make an AS.

The pattern of impaired performance described above differs from that found in SZ (and other psychiatric) patients. When SZ patients perform poorly, they make more reflexive errors and/or are slower to make a correct AS than a comparison group. The error rates are not typically of the magnitude reported by Guitton et al (38). Moreover, SZ patients spontaneously correct errors, which is commonly used to support the inference that patients understood the task demands. Furthermore, neither correct AS nor corrected AS errors are hypometric or visually induced. Thus, the first pattern of compromised AS performance in the frontal patients tested by Guitton et al (38) does not match the findings in SZ patients.

The second pattern involves asymmetries in error rate (more errors when the cue was presented contralateral to the side of the lesion). Strong asymmetries occurred in two patients, involving error rates of about 60-80% for cues presented contralateral to the lesion. Weak asymmetries also occurred in two patients; Guitton et al considered the performance of these patients to be “essentially normal”. Asymmetries in error rate are also not characteristic of the performance of SZ patients on the AS task, as noted above.

The third pattern, which occurred in two of Guitton’s patients, was weak bilateral increases in reflexive saccade errors. This is the single compelling similarity between the findings reported by Guitton et al and reports on SZ patients, their relatives, and other psychiatric populations.

Pierrot-Deseilligny et al (39) compared patients with unilateral infarctions in the posterior parietal cortex (n=10) or in one of three frontal regions, the supplementary motor area (n=9), frontal eye fields (n=10), DLPFC (n=16), with 20 neurologically healthy controls. The patients were tested 17 days after the infarction occurred. Only patients with infarctions of the DLPFC showed a significantly higher error rate than controls. The specificity of

a high error rate to infarctions of the DLPFC is widely believed to support two inferences – first, that this region controls the ability to inhibit reflexive saccades, and second, that a dysfunction involving the DLPFC underlies the high AS error rate in SZ patients.

The data on the patients with DLPFC infarctions reveal that two patterns of poor performance characterized all DLPFC patients: a) very marked bilateral increases in error rate (about 50% to near 100%) or b) very marked asymmetries in error rate. As noted above, neither pattern is characteristic of SZ patients. On the basis of these comparisons, we conclude that the evidence for similar deficits in SZ patients and in these groups of neurological patients with frontal and prefrontal pathology is not strong. What is much more striking is the lack of regional brain specificity for high error rates in a wide range of clinical conditions. After reviewing the clinical, imaging, and basic studies on the AS task, Everling and Fischer (100) concluded that there is a “... lack of certainty as to which brain structures are involved in the performance of antisaccades... Despite a high sensitivity of the antisaccade task, its specificity for a disease or the location of the involved brain structure may be low... While many authors tend to interpret failures in the antisaccade task as indications of frontal or prefrontal deficits, review of the different components that must be intact for a successful performance of the antisaccade task shows that such a conclusion may be premature or even wrong”.

We turn now to a comparison of the results reported by Guitton et al (38) and Pierrot-Deseilligny et al (39). In the Guitton et al study, the most profound impairments on the AS task occurred in the patients with frontal eye fields and supplementary motor area lesions. In contrast, in the Pierrot-Deseilligny et al study, patients with infarctions in these regions did not differ in error rate from controls or show the patterns of strong or weak bilateral deficits described by Guitton et al. Nor did Pierrot-Deseilligny et al describe a failure to correct errors or inability to generate an AS. Moreover, in our reading of their data, the most prominent feature of the AS performance of the patients with supplementary motor area, frontal eye fields and posterior parietal cortex infarctions was mild to moderate asymmetry in error rate. These findings thus indicate that central nervous system pathology involving a variety of different brain regions is associated with AS error rate asymmetry and with bilateral increases in error rate.

It is difficult to account for the differences between the findings of the Guitton et al and Pierrot-Deseilligny et al studies. Methodological differences may be one factor. Clinical differences between the patients in the two studies may also explain why Guitton et al found much more profound deficits in AS performance than Pierrot-Deseilligny et al did.

It is useful to note that small sample sizes limit statistical power to detect significant between-group differences unless the effect size is very large, as was clearly the case

for the DLPFC patients in the Pierrot-Deseilligny et al study. One cannot rule out the possibility that patients with infarctions in the supplementary motor area, frontal eye fields and posterior parietal cortex did not differ from controls in error rate because of the low power of very small samples (4–6 per group).

Having examined the seminal studies of AS in neurological patients, we conclude that the AS performance of SZ patients is, for the most part, not like that of the neurological groups studied by Guitton et al and Pierrot-Deseilligny et al. Indeed, SZ patients show a pattern described in the results section of this paper: a moderate number of AS errors, almost all of which are spontaneously corrected, with no significant lateral asymmetry.

In summary, the AS task and each of the neuropsychological tests tap complex psychological functions and behavior. Phenomenologically, each task requires that the person inhibit a prepotent response, although obviously this is not the only behavior involved in the successful performance of these tasks. The poor performance of SZ patients on each of these tasks points to a compromised ability to inhibit a prepotent response. In the case of AS, it is the failure to inhibit the tendency to make a visually guided saccade. In the case of the Stroop INTF score it is the failure to inhibit the more practiced tendency to read the color word rather than to name the color in which the word is printed. It is noteworthy, moreover, that SZ patients are able to generate a correct AS and to correct their Stroop errors. The imperfect and inconsistent pattern of intercorrelations among these tasks indicates that they do not involve one unitary function. The complexity of these tasks requires that they be parsed into their simpler components in order to penetrate to the functional impairments they tap in SZ (22,101,102). In this report, we have called attention to differences in the pattern of error performance on the AS task in SZ patients and in patients with verified central nervous system pathology.

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References

1. Fukushima J, Fukushima K, Chiba T et al. Disturbances of voluntary control of saccadic eye movements in schizophrenic patients. *Biol Psychiatry* 1988;23:670-7.
2. Fukushima J, Morita N, Fukushima K et al. Voluntary control of saccadic eye movements in patients with schizophrenic and affective disorders. *J Psychiatr Res* 1990;24:9-24.
3. Fukushima J, Fukushima K, Morita N et al. Further analysis of the control of voluntary saccadic eye movements in schizophrenic patients. *Biol Psychiatry* 1990;28:943-58.
4. Fukushima J, Fukushima K, Miyasaka K et al. Voluntary control of saccadic eye movement in patients with frontal cortical lesions and parkinsonian patients in comparison with that in schizophrenics. *Biol Psychiatry* 1994;36:21-30.
5. Thaker GK, Nguyen JA, Tamminga CA. Increased saccadic distractibility in tardive dyskinesia: functional evidence for subcortical GABA dysfunction. *Biol Psychiatry* 1989;25:49-59.
6. Rosse RB, Schwartz BL, Kim SY et al. Correlation between antisaccade and Wisconsin Card Sorting Test performance in schizophrenia. *Am J Psychiatry* 1993;150:333-5.
7. Clementz BA, McDowell JE, Zisook S. Saccadic system functioning among schizophrenia patients and their first-degree relatives. *J Abnorm Psychol* 1994;103:277-87.
8. Matsue Y, Saito H, Osakabe K et al. Smooth pursuit eye movements and voluntary control of saccades in the antisaccades task in schizophrenic patients. *Jpn J Psychiatry Neurol* 1994;48:13-22.
9. Crawford TJ, Haegar B, Kennard C et al. Saccadic abnormalities in psychotic patients. I. Neuroleptic-free psychotic patients. *Psychol Med* 1995;25:461-71.
10. Crawford TJ, Haegar B, Kennard C et al. Saccadic abnormalities in psychotic patients. II. The role of neuroleptic treatment. *Psychol Med* 1995;25:473-83.
11. Crawford TJ, Sharma T, Puri BK et al. Saccadic eye movements in families multiply affected with schizophrenia: the Maudsley family study. *Am J Psychiatry* 1998;155:1703-10.
12. Sereno AB, Holzman PS. Antisaccades and smooth pursuit eye movements in schizophrenia. *Biol Psychiatry* 1995;37:394-401.
13. Allen JS, Lambert AJ, Attah Johnson FY et al. Antisaccadic eye movements and attentional asymmetry in schizophrenia in three Pacific populations. *Acta Psychiatr Scand* 1996;94:258-65.
14. Tien AY, Ross DE, Pearlson G et al. Eye movements and psychopathology in schizophrenia and bipolar disorder. *J Nerv Ment Dis* 1996;184:331-8.
15. Katsanis J, Kortenkamp S, Iacono WG et al. Antisaccade performance in patients with schizophrenia and affective disorder. *J Abnorm Psychol* 1997;106:468-72.
16. McDowell JE, Clementz BA. The effect of fixation condition manipulations on antisaccade performance in schizophrenia: studies of diagnostic specificity. *Exp Brain Res* 1997;115:333-44.
17. Radant AD, Claypoole K, Wingerson DK et al. Relationships between neuropsychological and oculomotor measures in schizophrenia patients and normal controls. *Biol Psychiatry* 1997;42:797-805.
18. Arolt V, Teichert H-M, Steege D et al. Distinguishing schizophrenic patients from healthy controls by quantitative measurement of eye movement parameters. *Biol Psychiatry* 1998;44: 448-58.
19. Hutton SB, Crawford TJ, Puri BK et al. Smooth pursuit and saccadic abnormalities in first-episode schizophrenia. *Psychol Med* 1998;28:685-92.
20. Karoumi B, Ventre-Dominey J, Vighetto A et al. Saccadic eye movements in schizophrenic patients. *Psychiatry Res* 1998;77:9-19.
21. Karoumi B, Saoud M, d'Amato T et al. Poor performance in smooth pursuit and antisaccadic eye-movement tasks in healthy siblings of patients with schizophrenia. *Psychiatry Res* 2001;101:209-19.
22. Levy DL, Mendell NR, LaVanher C et al. Disinhibition in antisaccade performance in schizophrenia. In: Lenzenweger MF, Dworkin R (eds). *Origins and development of schizophrenia: advances in experimental psychopathology*. Washington: American Psychological Association Press, 1998:185-210.
23. Maruff P, Danckert J, Pantelis C et al. Saccadic and attentional abnormalities in patients with schizophrenia. *Psychol Med* 1998;28:1091-100.
24. Ross RG, Harris JG, Olincy A et al. Familial transmission of two independent saccadic abnormalities in schizophrenia. *Schizophr Res* 1998;30:59-70.
25. McDowell JE, Myles-Worsley M, Coon H et al. Measuring liability

- ity for schizophrenia using optimized antisaccade stimulus parameters. *Psychophysiology* 1999;36:138-41.
26. Muller N, Riedel M, Eggert T et al. Internally and externally guided voluntary saccades in unmedicated and medicated schizophrenics. II. Saccadic latency, gain, and fixation suppression errors. *Eur Arch Psychiatry Clin Neurosci* 1999;249:7-14.
 27. Snitz BE, Curtis CE, Zald DH et al. Neuropsychological and oculomotor correlates of spatial working memory performance in schizophrenia patients and controls. *Schizophr Res* 1999;38:37-50.
 28. Straube A, Riedel N, Eggert T et al. Internally and externally guided voluntary saccades in unmedicated and medicated schizophrenics. I. Saccadic velocity. *Eur Arch Psychiatry Clin Neurosci* 1999;249:1-6.
 29. Nieman DH, Bour LJ, Linszen DH et al. Neuropsychological and clinical correlates of antisaccade task performance in schizophrenia. *Neurology* 2000;54:866-71.
 30. Brenner CA, McDowell JE, Cadenhead KS et al. Saccadic inhibition among schizotypal personality disorder subjects. *Psychophysiology* 2001;38:399-403.
 31. Curtis CE, Calkins ME, Grove WM et al. Saccadic disinhibition in patients with acute and remitted schizophrenia and their first-degree biological relatives. *Am J Psychiatry* 2001;158:100-6.
 32. Curtis CE, Calkins ME, Iacono W.G. Saccadic disinhibition in patients and their first-degree biological relatives. *Exp Brain Res* 2001;137:228-36.
 33. Gooding DC, Tallent KA. The association between antisaccade task and working memory task performance in schizophrenia and bipolar disorder. *J Nerv Ment Dis* 2001;189:8-16.
 34. Manoach DS, Lindgren KA, Cherkasova MV et al. Schizophrenic subjects show deficient inhibition but intact task switching on saccadic tasks. *Biol Psychiatry* 2001;51:816-26.
 35. Brownstein J, Krastoshevsky O, McCollum C et al. Antisaccade performance in schizophrenia patients and their biological relatives. *Schizophr Res* 2003;63:13-25.
 36. Levy DL, O'Driscoll G, Matthyse S et al. Antisaccade performance in biological relatives of schizophrenia patients: a meta-analysis. Submitted for publication.
 37. Hallett PE. Primary and secondary saccades to goals defined by instructions. *Vision Res* 1978;18:1279-96.
 38. Guitton D, Bachtel HA, Douglas RM. Frontal lobe lesions in man cause difficulties in suppressing reflexive glances and in generating goal-directed saccades. *Exp Brain Res* 1985;58:455-72.
 39. Pierrot-Deseilligny C, Rivaud S, Gaymard B et al. Cortical control of reflexive visually-guided saccades. *Brain* 1991;114:1473-85.
 40. Leigh RJ, Newman SA, Folstein SE et al. Abnormal ocular motor control in Huntington's disease. *Neurology* 1983;33:1268-75.
 41. Lasker AG, Zee DS, Hain TC et al. Saccades in Huntington's disease: initiation defects and distractibility. *Neurology* 1987;37:364-70.
 42. Tian JR, Zee DS, Lasker AG et al. Saccades in Huntington's disease: predictive tracking and interaction between release of fixation and initiation of saccades. *Neurology* 1991;41:875-81.
 43. Pierrot-Deseilligny C, Rivaud S, Pillon B et al. Lateral visually-guided saccades in progressive nuclear palsy. *Brain* 1989;112:471-87.
 44. Blin J, Mazetti P, Mazoyer B et al. Does the enhancement of cholinergic neurotransmission influence brain glucose kinetics and clinical symptomatology in progressive supranuclear palsy? *Brain* 1995;118:1485-95.
 45. Crawford TJ, Henderson L, Kennard C. Abnormalities of nonvisually-guided eye movements in Parkinson's disease. *Brain* 1989;112:1573-86.
 46. Kitagawa M, Fukushima J, Tashiro K. Relationship between antisaccades and the clinical symptoms in Parkinson's disease. *Neurology* 1994;44:2285-9.
 47. Crevits L, De Ridder K. Disturbed striatoprefrontal mediated visual behaviour in moderate to severe parkinsonian patients. *J Neurol Neurosurg Psychiatry* 1997;63:296-9.
 48. Briand KA, Strallow D, Hening W et al. Control of voluntary and reflexive saccades in Parkinson's disease. *Exp Brain Res* 1999;129:38-48.
 49. Lueck CJ, Tanyeri S, Crawford TJ et al. Antisaccades and remembered saccades in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1990;53:284-8.
 50. Vidailhet M, Rivaud S, Gouder-Khouja N et al. Eye movements in parkinsonian syndromes. *Ann Neurol* 1994;35:420-6.
 51. Fletcher WA, Sharpe JA. Saccadic eye movement dysfunction in Alzheimer's Disease. *Ann Neurol* 1986;20:464-71.
 52. Currie J, Ramsden B, McArthur C et al. Validation of a clinical antisaccadic eye movement test in the assessment of dementia. *Arch Neurol* 1991;48:644-8.
 53. Shaunak S, Orrell RW, O'Sullivan E et al. Oculomotor function in amyotrophic lateral sclerosis: evidence for frontal impairment. *Ann Neurol* 1995;38:38-44.
 54. Merrill PT, Paige GD, Abrams RA et al. Ocular motor abnormalities in human immunodeficiency virus infection. *Ann Neurol* 1991;30:130-8.
 55. Johnston JL, Miller JD, Nath A. Ocular motor dysfunction in HIV-1-infected subjects – a quantitative oculographic analysis. *Neurology* 1996;46:451-7.
 56. Spitzer R, Williams J, Gibbon M et al. Structured Clinical Interview for DSM-IV – Patient edition. Washington: American Psychiatric Association, 1994.
 57. Kendler KS. Structured Interview for Schizotypal Symptoms (version 1.5). Richmond: Virginia Medical College, Department of Psychiatry, 1989.
 58. Mannuzza S, Fryer A, Endicott J et al. Family Informant Schedule and Criteria (FISC). New York: Anxiety Disorders Clinic, New York State Psychiatric Institute, 1985.
 59. Smith GN, Iacono WG. Lateral ventricular size in schizophrenia and choice of control group. *Lancet* 1986, i:1450.
 60. Tsuang MT, Fleming JA, Kendler KS et al. Selection of controls for family studies. Biases and implications. *Arch Gen Psychiatry* 1988;45:1006-8.
 61. Schwartz S, Link BG. The 'well control' artefact in case/control studies of specific psychiatric disorders. *Psychol Med* 1989;19:737-42.
 62. Kendler KS. The super-normal control group in psychiatric genetics: possible artifactual evidence for coaggregation. *Psychiatr Genet* 1990;1:45-53.
 63. Milner B. Effects of different brain lesions on card sorting: the role of the frontal lobes. *Arch Gen Neurol* 1963;9:90-100.
 64. Robinson AL, Heaton RK, Lehman RW et al. The utility of the Wisconsin Card Sorting Test in detecting and localizing frontal lobe lesions. *J Consult Clin Psychol* 1980;48:605-14.
 65. Stuss DT, Benson DF. Frontal lobe lesions and behavior. In: Kertesz A (ed). Localization in neuropsychology. New York: Academic Press, 1983:429-54.
 66. Heaton RK. The Wisconsin Card Sorting Test (Manual). Odessa: Psychological Assessment Resources, 1981.
 67. Stroop JR. Studies of interference in serial verbal reactions. *J Exp Psychol* 1935;18:643-62.
 68. MacLeod C. Half a century of research on the Stroop effect: an integrative review. *Psychol Bull* 1991;109:163-203.
 69. Braff DL, Heaton R, Kuck J et al. The generalized pattern of neuropsychological deficits in outpatients with chronic schizophrenia with heterogeneous Wisconsin Card Sorting Test results. *Arch Gen Psychiatry* 1991;48:891-8.
 70. Blanchard JJ, Neale JM. The neuropsychological signature of schizophrenia: generalized or differential deficit. *Am J Psychiatry* 1994;151:40-8.
 71. Sullivan EV, Mathalon DH, Zipursky RB et al. Factors of the Wisconsin Card Sorting Test as measures of frontal-lobe function in

- schizophrenia and chronic alcoholism. *Psychiatry Res* 1993;46:175-99.
72. Morice R, Delahunty A. Frontal/executive impairments in schizophrenia. *Schizophr Bull* 1996;22:125-37.
 73. Perry W, Braff DL. A multimethod approach to assessing perseverations in schizophrenia patients. *Schizophr Res* 1998;33:69-77.
 74. Otteson J, Holzman PS. Cognitive controls and psychopathology. *J Abnorm Psychol* 1976;85:125-35.
 75. Wysocki JJ, Sweet JJ. Identification of brain-damaged, schizophrenic, and normal medical patients using a brief neuropsychological screening battery. *Int J Clin Neuropsychol* 1985;7:40-4.
 76. Schooler C, Nuemann E, Caplan LJ et al. A time course analysis of Stroop interference and facilitation: comparing normal and schizophrenic individuals. *J Exp Psychol* 1997;126:19-36.
 77. Barch DM, Carter CS, Hachten PC et al. The "benefits" of distractibility: mechanism underlying increased Stroop effects in schizophrenia. *Schizophr Bull* 1999;25:749-62.
 78. Cohen JD, Barch DM, Carter CS et al. Schizophrenic deficits in the processing of context: converging evidence from three theoretically motivated cognitive tasks. *J Abnorm Psychol* 1999;108: 20-133.
 79. Anderson TJ, Jenkins IH, Brooks DJ et al. Cortical control of saccades and fixation in man. A PET study. *Brain* 1994;117: 1073-84.
 80. Nakashima Y, Momose T, Sano I et al. Cortical control of saccade in normal and schizophrenic subjects: a PET study using a task-evoked rCBF paradigm. *Schizophr Res* 1994;12:259-64.
 81. O'Driscoll GA, Alpert NM, Matthyse SW et al. Functional neuroanatomy of antisaccade eye movements investigated with positron emission tomography. *Proc Natl Acad Sci* 1995;92: 925-9.
 82. Crawford TJ, Puri BK, Nijran KS et al. Abnormal saccadic distractibility in patients with schizophrenia: a 99mTc-HMPAO SPET study. *Psychol Med* 1996;26:265-77.
 83. Sweeney JA, Mintun MA, Kwee S et al. Positron emission tomography study of voluntary saccadic eye movements and spatial working memory. *J Neurophysiol* 1996;75:454-68.
 84. Doricchi F, Perani D, Incoccia C et al. Neural control of fast-regular saccades and antisaccades: an investigation using positron emission tomography. *Exp Brain Res* 1997;116:50-62.
 85. Muri RM, Heid O, Nirkko AC et al. Functional organization of saccades and antisaccades in the frontal lobe in humans: a study with echo planar functional magnetic resonance imaging. *J Neurol Neurosurg Psychiatry* 1998;65:374-7.
 86. Everling S, Spantekow A, Krappmann P et al. Event-related potentials associated with correct and incorrect responses in a cued antisaccade task. *Exp Brain Res* 1998;118:27-34.
 87. Everling S, Dorris MC, Klein RM et al. Role of primate superior colliculus in preparation and execution of anti-saccades and pro-saccades. *J Neurosci* 1999;19:2740-54.
 88. Everling S, Munoz DP. Neuronal correlates for preparatory set associated with pro-saccades and anti-saccades in the primate frontal eye field. *J Neurosci* 2000;20:387-400.
 89. Connolly JD, Goodale MA, Desouza JF et al. A comparison of frontoparietal fMRI activation during anti-saccades and anti-pointing. *J Neurophysiol* 2000;84:1645-55.
 90. Luna B, Thulborn KR, Munoz DP et al. Maturation of widely distributed brain function subserves cognitive development. *NeuroImage* 2001;13:786-93.
 91. McDowell JE, Brown GG, Paulus M et al. Neural correlates of refixation saccades and antisaccades in normal and schizophrenia subjects. *Biol Psychiatry* 2002;51:216-23.
 92. Raemakers M, Jansma JM, Cahn W et al. Neuronal substrate of the saccadic inhibition deficit in schizophrenia investigated with 3-dimensional event-related functional magnetic resonance imaging. *Arch Gen Psychiatry* 2002;59:313-20.
 93. Funahashi S, Chafee MV, Goldman-Rakic PS. Prefrontal neuronal activity in rhesus monkeys performing a delayed anti-saccade task. *Nature* 1993;365:753-6.
 94. Gaymard B, Pierrot-Deseilligny C, Rivaud S. Impairment of sequences of memory-guided saccades after supplementary motor area lesions. *Ann Neurol* 1990;28:622-6.
 95. Gaymard B, Ploner CJ, Rivaud S et al. The frontal eye field is involved in spatial short-term memory but not in reflexive saccade inhibition. *Exp Brain Res* 1999;129:288-301.
 96. Paus T, Petrides M, Evans AC et al. Role of the anterior cingulate cortex in the control of oculomotor, manual, and speech responses: a positron emission tomography study. *J Neurophysiol* 1993;70:453-69.
 97. Rivaud S, Muri RM, Gaymard B et al. Eye movement disorders after frontal eye field lesions in humans. *Exp Brain Res* 1994;102:110-20.
 98. Pardo JV, Pardo PJ, Janer KW et al. The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proc Natl Acad Sci* 1990;87:256-9.
 99. Frith CD, Friston KJ, Liddle PF et al. Willed action and the prefrontal cortex in man: a study with PET. *Proc Roy Soc Lond Ser B* 1991;244:241-6.
 100. Everling S, Fischer B. The antisaccade: a review of basic research and clinical studies. *Neuropsychologia* 1998;36:885-99.
 101. Holzman PS. Parsing cognition. The power of psychology paradigms. *Arch Gen Psychiatry* 1994;51:952-4.
 102. Levy D. Location, location, location: the pathway from behavior to brain locus in schizophrenia. In: Matthyse S, Levy DL, Kagan J et al (eds). *Psychopathology: the evolving science of mental disorder*. New York: Cambridge University Press, 1996;100-26.

A cross-cultural study of eating attitudes in adolescent South African females

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Eating disorders were first described in black females in South Africa in 1995. A subsequent community based study of eating attitudes amongst adolescent females in an urban setting suggested that there would be increasing numbers of sufferers from within the black community. The current study sought to extend these findings using a larger, more representative urban sample. The results support those of the preliminary study. The underlying basis for the emerging phenomenon is discussed.

Key words: Eating disorders, eating attitudes, South Africa

The published literature on eating disorders in white South Africans dates back to the 1970s (1,2). In a South African study on treatment outcome for hospitalised anorexics and bulimics (3), it was mentioned that no blacks had been referred for treatment of either condition. In January 1993, one of the authors (CPS) first diagnosed an eating disorder in an adolescent black female, leading ultimately to the first published series of such cases in South Africa (4). Subsequently there has been an increase in the number of black eating disordered patients hospitalised for the treatment of their illness (5). The question arose as to whether the initial cases described were forerunners of further cases, in greater numbers, signalling a shift in the demographic profile of eating disorders (4).

The 1990s were a time of increasing racial integration at various levels of South African society, despite the existence of apartheid. One specific area of racial integration was at privately funded schools. Such schools seemed appropriate for the exploration of eating attitudes. Underlying this approach was the theory of socio-cultural factors impacting on the occurrence of eating disorders, whereby the ideals of Western society are specifically implicated (6). In this regard, private schools were viewed as institutions dominated by Western values. Given the existing literature on the link between acculturation and eating disorders (7,8), it was hypothesized that black females (constituting a minority group in an alien cultural milieu) in such a setting may demonstrate an inclination towards the development of eating disorders.

This was explored through the measurement of eating attitudes at a private, all-girl, secondary school in the greater Johannesburg area (i.e. an urban setting). Using the Eating Attitudes Test (EAT-26) (9), a surprising number of black respondents (37.5%) demonstrated potentially pathological eating attitudes (10). This finding suggested that the initial cases described were indeed part of an emerging phenomenon (5). The current study was undertaken to provide further support for this through a larger sample, attending state funded schools, in an urban location.

In essence, the hypothesis of the current study is that black adolescents in an urban setting will demonstrate eating attitudes similar to their white counterparts. The ultimate aim of the study is to demonstrate that setting, and not race or ethnic group, has an important influence on eating attitudes. Within the South African context such data is important in terms of dispelling notions of racial exclusivity regarding the risk for the development of eating disorders, i.e. that only whites and not blacks are at risk, which may have implications for resource allocation.

METHODS

The study was conducted on separate days during 1996 at three secondary schools, which were in an urban area (Johannesburg: Gauteng province). The schools were single sex (i.e. all girls) and were racially diverse in composition. The decision to use three schools in the urban sample was influenced by the required sample size, which was calculated from a pilot study (10).

Prior to commencing the study, extensive liaison took place between the various school principals and the researcher in terms of establishing the acceptability and viability of conducting research at their respective schools. The study was approved by the Committee for Research on Human Subjects at the University of the Witwatersrand.

The completion of questionnaires at each school took place on a designated day at a specific time such that an entire school was simultaneously involved in the process. The process was monitored and supervised by teachers and the researcher. In general the procedure was completed within one hour. During the time of questionnaire completion, no discussion was permitted between respondents. Participation in the study was voluntary and each girl who participated provided written informed consent. Parental permission to participate was also required. Anonymity was guaranteed.

A demographic questionnaire and the EAT-26 were employed. The demographic questionnaire sought to col-

lect information on age, school standard, race, home language, current height, current weight. The last two parameters were directly measured and the value was given to the respondent to fill in on the questionnaire. In addition, the respondent was required to specify paternal/maternal occupation, which was for the purpose of establishing socio-economic status. The EAT-26 (9) is a self report questionnaire comprising questions dealing with attitudes, concerns and behaviours related to food, weight and body shape. A total score of 20 or more represents the cut-off for the existence of disturbed eating attitudes and behaviours.

Prior to conducting the study, the EAT-26 was discussed with each school principal with regard to the terminology used. The wording of the questionnaire was altered in certain instances, based on recommendations from the various schools, e.g. the word "impulse" was replaced by the word "urge". Within the South African context, the EAT-26 has previously been employed in several studies. The study by Szabo and Hollands (10) established, but did not report, Cronbach's alpha values of 0.75 for black respondents and 0.79 for white respondents, whereas the study by Senekal et al (11), involving black university students, established a value of 0.62.

RESULTS

The total sample consisted of 1353 female respondents. The total enrolment of pupils at the three schools was 1579, giving a response rate of 86%. Of the 226 non-responders, 46 had been absent on the day of the study. The racial composition was as follows: black 43% (n=578), white 37% (n=506), other 20% (n=269). Only the data derived from the black and white respondents are reported.

White respondents were significantly taller ($t = -9.82$, $df = 961$, $p < 0.0001$) but had a significantly lower body mass index ($t = 4.73$, $df = 936$, $p < 0.0001$) than black respondents. No significant difference was found for either age ($t = 0.95$, $df = 1079$, $p = 0.34$) or weight ($t = 1.32$, $df = 1004$, $p = 0.18$). The socio-economic status of the white sample was significantly higher ($p = 0.001$).

Within the black sample, 18.7% (108/578) scored 20 or above. The mean score for the entire sample (n=578) was 12.48 (SD = 8.94). The scores for the various subscales were as follows: Dieting = 7.68 (6.75), Bulimic = 1.61 (2.38), Oral control = 3.17 (3.04). For those scoring 20 or above (n=108) on the EAT-26, the mean score was 27.19 (SD = 7.19). Mean scores for the subscales were as follows: Dieting = 18.04 (5.98), Bulimic = 3.84 (3.3), Oral control = 5.3 (3.73). For those scoring below 20 (n=470) on the EAT-26, the mean score was 9.1 (5.02). Mean scores for the various subscales were as follows: Dieting = 5.3 (4.19), Bulimic = 1.1(1.76), Oral control = 2.68 (2.63). Cronbach's alpha was 0.74.

A principal component factor analysis with varimax

rotation revealed that 31% of the variance could be explained by three factors, with factor 1 accounting for 16.6% of the total variance. The first three factors (corresponding to the subscales as follows: Factor 1 = Dieting, Factor 2 = Bulimia, Factor 3 = Oral control) had eigen values of 4.32, 1.95 and 1.78 respectively. Using an orthogonal transformation matrix demonstrated 8/13, 3/6 and 3/7 items for factors 1,2 and 3 respectively with a loading of >0.4 .

Within the white sample, 18.6% (94/506) scored 20 or above. The mean score on the EAT-26 for the entire sample (n=506) was 12.27 (SD = 10.21). The scores on the subscales for the entire sample (n=506) were as follows: Dieting = 8.47 (7.88), Bulimic = 1.29 (2.55), Oral control = 2.5 (2.71). For those scoring 20 or above (n=94), the mean total score was 29.65 (8.53), with scores on the subscales as follows: Dieting = 21.44 (5.84), Bulimic = 4.43 (4.08), Oral control = 3.77(3.58). For those scoring below 20 (n= 412), the mean total score was 8.30 (5.18), with scores on the subscales as follows: Dieting = 5.5(4.62), Bulimic = 0.5(1.21), Oral control = 2.21(2.38). Cronbach's alpha was 0.85.

A principal component factor analysis with varimax rotation revealed that 44% of the variance could be explained by three factors, with factor 1 accounting for 28% of the total variance. The eigen values of the first three factors were 7.38, 2.27 and 1.8 respectively. Using an orthogonal transformation matrix demonstrated 11/13, 5/6 and 6/7 items for factors 1,2 and 3 respectively with a factor loading >0.4 .

The samples did not differ significantly (using a two sample t-test) for either total EAT-26 score ($t = 0.36$, $df = 1082$, $p = 0.71$) or the Dieting subscale ($t = 1.75$, $df = 1082$, $p = 0.07$). However, black respondents scored significantly higher on both the Bulimia subscale ($t = 2.15$, $df = 1082$, $p = 0.03$) and the Oral control subscale ($t = 3.81$, $df = 1082$, $p = 0.001$). Amongst those respondents who scored 20 or above, white respondents had a significantly higher total score than black respondents, utilising two sample t-tests ($t = -2.22$, $df = 200$, $p = 0.02$). This was also observed for the Dieting subscale ($t = -4.07$, $df = 200$, $p = 0.0001$). Black respondents scored significantly higher on the Oral control subscale ($t = 2.95$, $df = 200$, $p = 0.0035$). There was no significant difference for the Bulimia subscale ($t = -1.14$, $df = 200$, $p = 0.255$).

DISCUSSION

The EAT-26 provides information on the possible risk for developing an eating disorder by virtue of the total score obtained, as well as a profile of eating attitudes in terms of the subscale scores obtained. The total EAT-26 scores for the black (12.48) and white (12.27) samples were not significantly different, which concurs with an earlier South African study using the EAT-26 in a racially mixed urban, adolescent sample (10).

Recent studies carried out in South Africa using the EAT-26 found a mean total score of 12.1 in a black, female, university sample with a mean age of 20 (11), and total scores of 11.7 and 12.5 in black and white, respectively, adolescent schoolgirls with mean ages of 16.3 and 16.5 (12). These findings demonstrate a remarkable similarity with the findings of the current study, despite different mean ages between the samples as well different locations for the studies.

It has been proposed that EAT-26 scores be categorised in terms of potential risk for the development of an eating disorder: a score of less than 10 would denote no risk, a score of 10-19 would denote a low risk and a score of 20 or above a high risk (13). From this, 43.4% of our black sample and 50.6% of our white sample would have been categorised as having no risk, compared to 77.5% of the Swiss adolescent sample studied by Buddeburg-Fisher et al (13). It should be borne in mind that scores on the EAT-26 do vary over time in adolescents (14) and thus the significance of a given score at a particular point in time should not be overestimated.

In our study, the percentage of those scoring above the EAT-26 cut-off score of 20 was almost identical for black and white (18.6% versus 18.7%). These findings are comparable to recent South African data which established prevalence rates of 17.9% for black female adolescents and 21.2% for white female adolescents scoring 20 or above (12). Beyond comparisons with South African data, there are studies from the African continent that provide a broader context. The closest study to the current one was conducted in Nigeria, where, utilising the EAT-26, a prevalence figure for abnormal eating attitudes of 18.6% was established (15). Their sample was black and urban in location, comprising secondary school pupils and university students. An Egyptian study (16), conducted at a secondary school in Cairo, established a prevalence rate for abnormal eating attitudes of 11.4% using the EAT-40 (17). It has been noted within the South African setting that utilisation of the EAT-40 was associated with lower prevalence figures for abnormal eating attitudes. A study conducted amongst white adolescents in Cape Town, utilising the EAT-40, revealed the prevalence of abnormal eating attitudes to be 15% amongst the female respondents (18).

From an international perspective, the findings of the current study are generally closer to those in North America than Europe. The application of the EAT-26 and EAT-40 to North American samples of adolescents has generally yielded higher prevalence figures, e.g. 17.5% (19) and 22% (20), whereas in the United Kingdom prevalence figures have been somewhat lower, e.g. 6.9% (21) and 9.3% (14). However, a recent British study conducted on 11-16 year old females established that 18.6% of this population have attitudes and concerns regarding weight and shape which place them at risk for the development of eating disorders (22). In general it would seem that the findings

of the current study, regarding the prevalence of abnormal eating attitudes using the EAT-26, are within the spectrum of findings from around the world.

South Africa is a country in transition (23). Transformation of all aspects of societal functioning is evident and reflective of this transition. Cultures that previously coexisted as almost mutually exclusive entities are now engaging. As a consequence, beliefs and value systems are potentially changing and evolving in the direction of homogeneity within specific settings. The school system provides a possible model for the exploration of this phenomenon given the fluidity of identity within the age group of school attending children and adolescents. Adolescence is a time of significant self awareness and critical self evaluation (24). It is generally during this developmental period that, amongst females predominantly, body dissatisfaction and subsequent efforts to address this through dieting occur (25). Against this background, which involves more than aesthetic concerns (26), conditions such as eating disorders emerge. Western culture appears to have a powerful impact on the development of such conditions (27). Whilst clinical experience has contributed to the understanding that these conditions affect predominantly white females (28), the emergence of black sufferers in urban settings in both the Western (29) and non-Western world (4) suggests culture relates to milieu and value system rather than race, and by implication, is a more powerful mediator of illness expression than race. With the advent of and the means for mass communication, Western culture is becoming increasingly pervasive. With a seductive emphasis on consumerism (30), it is indeed a powerful culture. Assimilation of the associated Western value system, where physical appearance and self worth are seemingly synonymous (31), seems inevitable.

Contemporary South African society provides an opportunity for exploring the impact of this phenomenon. Rural dwellers with so-called traditional beliefs (32) are migrating into the cities, interfacing with urban dwellers of all races who, with the advent of desegregation, are in turn being more vigorously exposed to one another's cultures. In this instance, adolescent eating attitudes and their putative relationship with eating disorders provide a perspective for exploration of this phenomenon. On one level this study may be viewed as a measure of the prevalence of abnormal eating attitudes, which may have implications for the future epidemiology of eating disorders, within various groups of South African adolescents. However, beyond the concrete measures and data, there would appear to be implications for an urbanizing, female African population within the broader context of identity. Within the South African setting it has been proposed that black, female emancipation at both a political and socio-economic level has occurred rapidly and without either historical precedent or meaningful mentorship, thus creating circumstances that may increase vulnerability to iden-

tity seeking through weight and shape preoccupations leading potentially to abnormal eating attitudes and the possible development of eating disorders (33).

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References

1. Beumont PJ, George GCW, Smart DE. "Dieters" and "vomitters" and "purgers" in anorexia nervosa. *Psychol Med* 1976;6:617-32.
2. Norris DL. Clinical diagnostic criteria for primary anorexia nervosa. *South African Med J* 1979;56:987-93.
3. Nash ES, Colborn AL. Outcome of hospitalised anorexics and bulimics in Cape Town, 1979-1989. *South African Med J* 1994;84:74-9.
4. Szabo CP, Berk M, Tlou E et al. Eating disorders in black female South Africans. A series of cases. *South African Med J* 1995;85:588-90.
5. Szabo CP. Eating attitudes among black South Africans. *Am J Psychiatry* 1999;156:981.
6. Dolan B. Cross-cultural aspects of anorexia nervosa and bulimia: a review. *Int J Eat Disord* 1991;10:67-78.
7. Pumariega A. Acculturation and eating attitudes in adolescent girls: a comparative and correlational study. *J Am Acad Child Psychiatry* 1986;25:276-9.
8. Furkawa T. Weight changes and eating attitudes of Japanese adolescents under acculturative stresses: a prospective study. *Int J Eat Disord* 1994;15:71-9.
9. Garner DM, Olmsted MP, Bohr Y et al. The Eating Attitudes Test: psychometric features and clinical correlates. *Psychol Med* 1982;12:871-8.
10. Szabo CP, Hollands C. Abnormal eating attitudes in secondary-school girls in South Africa, a preliminary study. *South African Med J* 1997;87:524-30.
11. Senekal M, Steyn NP, Mashego TB et al. Evaluation of body shape, eating disorders and weight management parameters in black female students of rural and urban origins. *South African J Psychol* 2001;31:45-53.
12. Caradas AA, Lambert EV, Charlton KE. An ethnic comparison of eating attitudes and associated body image concerns in adolescent South African schoolgirls. *J Hum Nutr Diet* 2001;14:11-120.
13. Buddeburg-Fischer B, Bernet R, Sieber M et al. Epidemiology of eating behaviour and weight distribution in 14- to 19- year- old Swiss students. *Acta Psychiatr Scand* 1996;93:296-304.
14. Patton GC, Johnson-Sabine E, Wood K et al. Abnormal eating attitudes in London schoolgirls - A prospective epidemiological study: outcome at twelve months. *Psychol Med* 1990;20:382-94.
15. Oyewumi LK, Kazarian SS. Abnormal eating attitudes among a group of Nigerian youths: II. Anorexic behaviour. *East African Med J* 1992;69:667-9.
16. Nasser M. Screening for abnormal eating attitudes in a population of Egyptian secondary school girls. *Soc Psychiatry Psychiatr Epidemiol* 1994;29:25-30.
17. Garner DM, Garfinkel PE. The Eating Attitudes Test: an index of the symptoms of anorexia nervosa. *Psychol Med* 1979;9:273-9.
18. le Grange D, Tibbs J, Selibowitz J. Eating attitudes, body shape, and self-disclosure in a community sample of adolescent girls and boys. *Eat Disord* 1995;3:253-64.
19. Fisher M, Pastore D, Schneider M et al. Eating attitudes in urban and suburban adolescents. *Int J Eat Disord* 1994;16:67-74.
20. Leichner P, Arnett J, Rallo JS et al. An epidemiological study of maladaptive eating attitudes in a Canadian school age population. *Int J Eat Disord* 1986;5:969-82.
21. Mann AH, Wakeling A, Wood K et al.. Screening for abnormal eating attitudes and psychiatric morbidity in an unselected population of 15-year-old schoolgirls. *Psychol Med* 1983;13:573-80.
22. Cooper PJ, Goodyer I. Prevalence and significance of weight and shape concerns in girls aged 11-16 years. *Br J Psychiatry* 1997;171:542-4.
23. Bornman E. Self-image and ethnic identification in South Africa. *J Soc Psychol* 1999;139:411-25.
24. Morgan CT, King RA, Weisz JR et al. Introduction to psychology, 7th ed. New York: McGraw-Hill, 1986.
25. Moore DC. Body image and eating behavior in adolescents. *J Am Coll Nutr* 1993;12:505-10.
26. Steiger H. Anorexia nervosa: is it the syndrome or the theorist that is culture- and gender- bound? *Transcult Psychiatr Res Rev* 1993;30:347-58.
27. Nasser M. Eating disorders: the cultural dimension. *Soc Psychiatry Psychiatr Epidemiol* 1988;23:184-7.
28. American Psychiatric Association. Practice guideline for eating disorders. *Am J Psychiatry* 1993;150:212-28.
29. Hsu LKG. Are the eating disorders becoming more common in blacks. *Int J Eat Disord* 1987; 6:113-24.
30. Swartz L. Anorexia nervosa as a culture-bound syndrome. *Soc Sci Med* 1985;20:725-30.
31. Freedman RJ. Reflections on beauty as it relates to health in adolescent females. *Women and Health* 1984;9:29-45.
32. Swartz L. Issues for cross-cultural psychiatric research in South Africa. *Cult Med Psychiatry* 1985;9:59-74.
33. Szabo CP, le Grange D. Eating disorders and the politics of identity: the South African experience. In: Nasser M, Katzman M, Gordon R (eds). *Eating disorders and cultures in transition*. London: Routledge, 2001:24-33.

Depression among older people in Europe: the EURODEP studies

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The data from nine centres in Europe which had used the Geriatric Mental Scale (GMS) AGE CAT were analysed to compare prevalence of diagnoses in subjects aged 65 years and over living in the community. Levels of depressive illness were: Iceland 8.8%, Liverpool 10.0%; Zaragoza 10.7%; Dublin 11.9%; Amsterdam 12.0%; Berlin 16.5%; London 17.3%; Verona 18.3% and Munich 23.6%. Taking all levels of depression, five high (Amsterdam, Berlin, Munich, London and Verona) and four low (Dublin, Iceland, Liverpool, Zaragoza) scoring centres were identified. Meta-analysis of all 13,808 subjects yielded a mean level of depression of 12.3% (95% CI 11.8-12.9), 14.1% for women (95% CI 13.5-14.8) and 8.6% for men (95% CI 7.9-9.3). Symptom levels varied between centres: 40% of the total study population in Amsterdam reported depressive mood against only 26% in Zaragoza. To incorporate studies from other centres using other methods for depression identification, the EURO-D scale was developed from 12 items of the GMS and validated against other scales and expert diagnosis. A two factor solution emerged, an 'affective suffering factor' and a 'motivation factor'. The EURO-D scale was applied to 14 population based surveys. Depression score tended to increase with age unlike levels of prevalence of depression. Large between centre differences were evident in levels of depression unexplained by age, gender or marital status. These data show that depressive illness defined as suitable for intervention is common among older people in Europe. Opportunities for effective treatment are almost certainly being lost. Levels of depressive symptoms vary significantly between high and low scoring centres, prompting the next phase of this study, an examination of risk factors in Europe.

Key words: EURODEP, depression, old age, GMS-AGECAT, EURO-D

The EURODEP consortium consists of a number of independent community based studies of depression among older people, conducted in centres in Europe which have been brought together to form a Concerted Action Programme under the European Community BIOMED I initiative. In the first studies, EURODEP is trying to answer the questions: how much depression exists among older people in Europe? Does its level vary from place to place? Does the clinical picture differ between populations? Is the level of depression consistent with suicide levels? Is it treated and what are its risk factors?

The increasing proportions of older people in the populations of Europe lent urgency to the need to know their levels of mental illness, of which one of the most prevalent is depression. First, a systematic review of the world literature on community-based studies of the prevalence of depression in later life (aged 55+) was undertaken (1). Thirty-four studies were eligible for inclusion, with a range of prevalence rates for depression of 0.4-35%. They revealed a weighted average for major depression of 1.8% and for minor depression of 9.8%, while all depressive syndromes considered clinically relevant reached 13.5%. A higher rate of prevalence of depression was a consistent finding for women and among older people in poor socio-

economic circumstances. Because of the diversity of measures used, it was not possible in such a survey to make comparisons between individual studies in order to identify areas of high and low depression prevalence. The need for a uniform standardised method was clear.

The aims of the first studies were: a) to study the variation in the prevalence of diagnosable depression among people aged 65 and over living in the community in different centres in Europe using a standardised method; b) to examine core symptoms and clinical profiles across centres: how do they differ? c) to interpret them in relation to existing socio-economic and risk factor variables; and d) the harmonisation of scales of depression to allow other centres to join the consortium for comparing levels of depressed mood by scale score.

METHOD

Formation of the consortium

The original members of the consortium (Study 1) had used the Geriatric Mental State (GMS) AGE CAT as the principal case finding and diagnostic instrument for their studies: Amsterdam (2); Berlin (3); Dublin (4); Iceland (5); Liverpool (6); London (7); Munich (8); Verona, Italy

(9); Zaragoza, Spain (10). The centres decided to come together and form a Concerted Action, pool their data and thus give added strength to their analyses of risk factors and generate new hypotheses for further studies.

To the original nine GMS AGE-CAT centres, another centre was added with expertise in the technique of 'Experience Sampling' (Maastricht). Five further centres (Study 2) applied to join - Gothenburg, Sweden (11); Antwerp, Belgium (12); Bordeaux, France (13); Oulu, Finland (14); and Amsterdam (15) - which had used other measures. It was decided to try to harmonise their measures of depression with those of the other centres. Under the European Community PECO initiative, an East European centre was added: Tirana, Albania (not reported here).

Characteristics of the centres

Details on the individual centres for Study 1 are reported elsewhere (16). All the centres took random community samples collected between 1990 and 1996, except for Iceland which had a total population birth cohort born 1895-1897 and interviewed in 1983, and Dublin which used a general practitioner complete register. The age range was from 65 upwards for most samples, except Amsterdam (65-84), Berlin (70+), Munich (85+) and Iceland (88-89). Only one centre excluded nursing homes entirely (Verona). Most samples were urban, except Iceland which was mixed rural/urban. Their size varied between 202 in Verona and 5222 in Liverpool. The two samples in Italy and Spain were predominantly catholic. The samples in the UK (London and Liverpool), in Germany (Berlin and Munich), in the Netherlands and in Iceland were predominantly protestant, while the sample in Dublin was catholic.

Measures

The studies were undertaken using the GMS (17,18) community version in approved translation. AGE-CAT (19,20) is a computerised diagnostic algorithm which uses scores on GMS items in stage one for each subject to produce a level of confidence of diagnosis on a scale of 0-4 or 0-5 for each of eight diagnostic syndrome clusters: organic brain syndrome, schizophrenia, mania, depression (psychotic and neurotic), and obsessional, hypochondriacal, phobic and anxiety neuroses. Stage 2 reaches a final differential diagnosis by comparing level for level, recorded as either a diagnostic subcase (confidence levels 1 and 2) or a diagnostic case (confidence levels 3,4 and 5). Thus it allows the identification of comorbid states. Level 3 and above on any diagnostic cluster accords with what psychiatrists would usually recognise as a case for treatment or intervention, if available. Good agreement has been shown between AGE-CAT cases of depression and DSM-III major depressive episode and dysthymia taken together (21,22). In addition to the GMS, most centres collected

risk factor information and seven undertook follow-up of their samples.

Data analysis for the pooled data took place in Liverpool. The Liverpool and Berlin samples were gender and age stratified. The overall prevalence figures for these centres are therefore adjusted using the appropriate weights to take this into account.

STUDY 1A: PREVALENCE OF DEPRESSION IN EUROPEAN CENTRES

Results

Substantial differences in the prevalence of depression were found, with Iceland having the lowest level at 8.8%, followed by Liverpool 10.0%; Zaragoza 10.7%; Dublin 11.9%; Amsterdam 12.0%; Berlin 16.5%; London 17.3%; Verona 18.3% and Munich 23.6%. When all five AGE-CAT depression levels, including both subcases of depression and cases, were added together, five high scoring centres emerged (Amsterdam, Berlin, Munich, London and Verona) with a prevalence of all levels of depression of 30.4 to 37.9%, and four low scoring centres (Dublin, Iceland, Liverpool, Zaragoza) with prevalence levels between 17.7 to 21.4%. Women almost invariably dominated over men. The examination of the proportions of subcases to cases, and psychotic to neurotic depression, although revealing some striking differences between centres, provided no obvious explanation for the difference in prevalence (see also 16).

Although age-specific prevalence rates varied between centres, there was no constant association between prevalence and age.

The meta-analysis of the pooled data on the nine European centres yielded 13,808 subjects, with an overall prevalence of depression of 12.3% (95% CI 11.8-12.9); 14.1% for women (95% CI 13.5-14.8) and 8.6% for men (95% CI 7.9-9.3).

Discussion

It was concluded that considerable variation existed in the levels of depression across Europe, although the cause was not immediately obvious. Cases and subcases taken together showed even greater variability, particularly for women, suggesting that it was not simply a matter of variation in case/subcase criteria, which were in any event standardised by computer. It is possible that risk factors for well/subcase and subcase/case transitions in both directions vary, although this was not true for Liverpool (23), where subcases shared similar risk factors to cases. Although there were substantial levels of depression in all centres, it can also be said that between 62 and 82 percent of older persons had no depressive level on the AGE-CAT system. It was finally concluded that substantial opportunities for treatment existed. Not all studies assessed treatment. Those that did, e.g. Liverpool,

found around 10 % of case level depression received anti-depressant medication.

STUDY 1B: PRESENTATION OF DEPRESSION AND DEPRESSIVE SYMPTOMS IN EUROPE

Results

The proportions of depressive symptoms were found to vary between centres. In Amsterdam, for example, 40% of the general population of older people reported depressive mood compared to 26% in Zaragoza. Symptoms such as 'future bleak', 'hopelessness', 'wish to be dead' were generally rare, but the last reached higher levels in Berlin, Munich and Verona. Sleep disturbance was admitted by only 15% of the population in Dublin, but 54% and 60% in Munich and Berlin. Large differences for some symptoms were found within the very old populations in Iceland, Berlin and Munich: in men aged 85 and over, the prevalence of 'depressed mood', 'crying', 'cannot cry', 'life not worth living' and 'wish to be dead' was 9%, 3%, 4%, 2% and 3%, respectively, in Iceland, whereas it was 50%, 33%, 26%, 30% and 29% in Munich, and 41%, 21%, 15%, 16% and 25% in Berlin, whereas there was no difference for 'guilt' and 'energy loss'. These differences were also evident for women (see also 24).

As expected from the prevalence levels of depression, many symptoms were more common among women. Centres where the prevalence of depression was low tended to have fewer symptoms among the well (i.e., those with no depressive level), but there were inconsistencies, so that a low level of symptoms in the 'well' did not necessarily predict a lower level in the depressed.

Discussion

We conclude that surprising variations in prevalence of depressive symptoms occurred between centres, and were not always consistent with levels of depressive illness. The high level of serious symptoms of depression in populations aged 85 and over in the German centres compared to others, and particularly to Iceland, may have been due to the lingering aftermath of the Second World War in this generation.

Less variation with age occurred than expected and was inconsistent between centres. There was no consistent relationship between proportions of symptoms in well persons and cases for all centres. In all, it can be said that the levels of depressive symptoms among over 60% of the older general population of Europe were low, so that pejorative stereotypes of old age in Europe as naturally depressed were not upheld.

STUDY 2A: HARMONISATION OF MEASURES OF DEPRESSION IN OLDER PEOPLE

Method

Because new centres had entered the consortium, which had not used the GMS AGE-CAT, attempts were

made to harmonise the depression measures which they had used with the GMS items, so that a common scale could be derived (the EURO-D, 25). Most of the non-GMS AGE-CAT centres had used the Short Care, the Center for Epidemiological Studies - Depressive Scale (CES-D), the Comprehensive Psychopathological Rating Scale (CPRS) and the Zung Self Rating Depression Scale (ZSDS). Common items were identified by scrutiny of these instruments, and algorithms for fitting items from other instruments to the GMS were derived. This was undertaken by direct observation of item correspondence or by expert opinion. The resulting twelve item scale was checked in each centre for internal consistency, criterion validity and uniformity of factor and analytic profiles.

Results

It was concluded that the EURO-D Scale, from whichever scale it had been derived, was entirely consistent and seemed to capture the essence of its parent instrument. It was also judged to have a comparable factor structure whatever its origin, but a two factor solution was optimal. 'Depression', 'tearfulness', and 'wishing to die' loaded on the first factor, which we called 'affective suffering', while 'loss of interest', 'poor concentration' and 'lack of enjoyment' loaded on the second, called the 'motivation factor'. It was concluded that the diverse depression measures covered common conceptual domains, and often had similarly worded items. Even differences in modes of administration (for example, self report versus semi-structured clinical interview) did not seem to prevent the extraction of broadly comparable data with common scaling properties.

STUDY 2B: APPLICATION OF THE EURO-D SCALE

Method

Subjects from the 14 population based surveys were used to test the EURO-D with respect to the main interactive effects of centre, age, gender and marital status (see also 26). Between centre variance was partitioned according to centre sub-characteristics, geographical region, prominent religion and survey instrument used.

Results

It was noted that EURO-D scores tended to increase with increasing age, unlike the levels of prevalence of depression. Women had generally higher scores than men, and widowed and separated subjects higher scores than those who were currently or never married.

Discussion

The EURO-D Scale, it appeared, could be reduced to two well characterised factors, 'motivation' and 'affective

suffering'. The motivation factor seemed to account for the positive association with age, while the affective suffering factor was responsible for the gender difference. The gender difference was modified by marital status, being negligible among those who had never married, and equally evident among the currently married, the widowed and the separated. There was no evidence for a continuation of the gender difference with increasing age. The effects of age, gender and marital status accounted for less than 1% of the variance in the EURO-D Scale.

It was concluded that there were large between centre differences, which could be explained neither by the age, gender or marital status characteristics of the population, nor by the instruments used to survey them.

Reasonably consistent, but small effects of age, gender and marital status on depression symptoms have been observed across the 14 European centres. It is concluded that while symptoms of depression increase with age, depression may be over-diagnosed in older persons because of an increase in complaints of lack of interest and motivation which may be affectively neutral and possibly related to cognitive decline.

STUDY 3: RELIGIOUS BEHAVIOUR AND DEPRESSIVE SYMPTOMS IN EUROPE

Method

We examined the influence of religion on the level of both depressive symptoms and illness across the European centres. The EURO-D Scale was used in this analysis to allow all the centres, original and additional, to participate. The influence of religion is of interest to the collaboration because of the split between North and South Europe and between protestant and roman catholic groups, with some catholic centres (Ireland, Belgium, and in part Liverpool) being in the more northerly parts of the continent, and Spain, Italy and France in the more southerly. The addition of Albania had the advantage of introducing a largely Muslim religious group (see also 27).

The protective effects of being a religious person were examined using data from 13 community based studies in 11 centres.

Results

Fewer depressive symptoms were found in those catholic countries with high rates of regular church attendance, while in protestant countries high levels of depressive symptoms were associated with lower levels of attendance. It was concluded that religious practice, especially when it is embedded within a traditional value-orientation, may facilitate coping with adversity in later life.

Interactive effects showed the results to be more pro-

nounced among older women. It was concluded that older Europeans appear to benefit from religious practice.

CONCLUSIONS

However interpreted, these results make it plain that depression as an illness is common among older people in Europe. Although it was not possible to assess the size of the population receiving treatment, it is known that in the London and Liverpool centres it falls often well below 15% of depressed persons judged as likely to benefit. There is no reason to suppose these figures are better in other European countries. Opportunities for effective treatment are almost certainly being lost.

THE FUTURE

The consortium is now addressing the risk factors for depression in this age group, and prognosis. We shall also be examining comorbidity with organic states, especially dementia. Issues such as daily life, handicap and depression, the validity of social measures of depression and the concept of handicap and the daily life of older people in Europe are being explored by the use of the Experience Sampling Method developed at the Maastricht Centre.

The consortium looks to extend its work into randomised controlled treatment trials of depression between centres and to study better methods for service delivery.

The EURODEP consortium gave rise to the ASIADep consortium, consisting of nine similar centres distributed in Asian countries (Japan, China, South Korea, Taiwan, Singapore, Malaysia and India) which will shortly be reporting their results.

References

1. Beekman ATE, Copeland JRM, Prince MJ. Review of community prevalence of depression in later life. *Br J Psychiatry* 1999;174: 307-11.
2. Van Ojen R, Hooijer C, Jonker C et al. Late-life depressive disorder in the community, early onset and the increase of vulnerability with increasing age. *J Affect Disord* 1995;33:159-66.
3. Helmchen H, Linden M, Wernicke T. Psychiatrische Morbidität bei Hochbetagten: Ergebnisse aus der Berliner Altersstudie. *Nervenarzt* 1996;67:739-50.
4. Lawlor BA, Bruce I, Swanwick GRJ et al. Prevalence of mental illness in an elderly community dwelling population using AGE-CAT. *Irish J Psychol Med* 1994;11:157-9.
5. Magnusson H. Mental health of octogenarians in Iceland. An epidemiological study. *Acta Psychiatr Scand* 1989;79 (Suppl. 349).
6. Saunders PA, Copeland JRM, Dewey ME et al. The prevalence of dementia, depression and neurosis in later life: the Liverpool MRC-ALPHA study. *Int J Epidemiol* 1993;22:838-47.
7. Livingston G, Hawkins A, Graham N et al. The Gospel Oak Study: prevalence rates of dementia, depression and activity limitation among elderly residents in inner London. *Psychol Med* 1990;20:137-46.
8. Meller I, Fichter M, Schroppel H et al. Mental and somatic health and needs for care in octo- and nonogenerians: an epidemiologi-

- cal study. *Eur Arch Psychiatry Clin Neurosci* 1993;242:286-92.
9. Turrina C, Perdon G, Bianchi L et al. Disturbi psichici (DSM-III-R) nella popolazione anziana del quartiere di Verona-Sud. Dati preliminari. *Riv Sper Fren* 1991;64:1006-13.
 10. Lobo A, Dewey M, Copeland JRM et al. The prevalence of dementia among elderly people living in Zaragoza and Liverpool. *Psychol Med* 1992;22:239-43.
 11. Skoog I, Nilsson L, Landahl S et al. Mental disorders and the use of psychotropic drugs in an 85 year old urban population. *Int Psychogeriatrics* 1993;5:33-48.
 12. Roelands M, Wostyn P, Dom H et al. The prevalence of dementia in Belgium: the population-based door-to-door survey in a rural community. *Neuroepidemiology* 1994;13:155-61.
 13. Barberger Gateau P, Chaslerie A, Dartigues JF et al. Health measures correlates in the French elderly community population: the PAQUID study. *J Gerontol* 1992;47:S88-S95.
 14. Kivela S-L, Pahkala K, Laipala P. Prevalence of depression in an elderly Finnish population. *Acta Psychiatr Scand* 1988;78: 401-13.
 15. Beekman ATF, Deeg DJH, van Tilberg T et al. Major and minor depression in later life: a study of prevalence and associated factors. *J Affect Disord* 1995;36:65-75.
 16. Copeland JRM, Beekman ATF, Dewey ME et al.. Depression in Europe. Geographical distribution among older people. *Br J Psychiatry* 1999;174:312-21.
 17. Copeland JRM, Kelleher MJ, Kellett JM et al. A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly. The Geriatric Mental State Schedule. I. Development and reliability. *Psychol Med* 1976;6:439-49.
 18. Gurland BJ, Fleiss JL, Goldberg K et al. A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly. The Geriatric Mental State Schedule 2. A factor analysis. *Psychol Med* 1976;6:451-9.
 19. Copeland JRM, Dewey ME, Griffiths-Jones HM. Computerised psychiatric diagnostic system and case nomenclature for elderly subjects: GMS and AGE-CAT. *Psychol Med* 1986;16:89-99.
 20. Dewey ME, Copeland JRM. Computerised psychiatric diagnosis in the elderly: AGE-CAT. *J Microcomputer Appl* 1986;9:135-40.
 21. Copeland JRM, Dewey ME, Griffiths-Jones HM. Dementia and depression in elderly persons: AGE-CAT compared with DSM III and pervasive illness. *Int J Geriatr Psychiatry* 1990;5:47-51.
 22. Ames D, Flynn E, Tuckwell V et al. Diagnosis of psychiatric disorder in elderly, general and geriatric hospital patients: AGE-CAT and DSM-III-R compared. *Int J Geriatr Psychiatry* 1994;9:627-33.
 23. Copeland JRM, Chen R, Dewey ME et al. Community-based case-control study of depression in older people. Cases and subcases from the MRC-ALPHA study. *Br J Psychiatry* 1999;175:340-7.
 24. Copeland JRM, Beekman ATF, Dewey ME et al. Cross-cultural comparison of depressive symptoms in Europe does not support stereotypes of ageing. *Br J Psychiatry* 1999;174:322-9.
 25. Prince MJ, Reischies F, Beekman ATF et al. Development of the EURO-D Scale - A European union initiative to compare symptoms of depression in 14 European centres. *Br J Psychiatry* 1999;174:330-8.
 26. Prince MJ, Beekman ATF, Deeg DJH et al. Depression symptoms in late life assessed using the EURO-D Scale. The effect of age, gender and marital status in 14 European centres. *Br J Psychiatry* 1999;174:339-45.
 27. Braam A, van den Eeden P, Prince MJ et al. Religion as a cross cultural determinant of depression in elderly Europeans: results from the EURODEP collaboration. *Psychol Med* 2001;31:803-14.

Traumatic grief in Kenyan bereaved parents following the Kyanguli School fire tragedy

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Following the death of 67 boys in a fire tragedy at Kyanguli School in rural Kenya, the level of traumatic grief was assessed in a sample of 164 parents and guardians whose sons died in the fire. The study was cross-sectional. Counseling services were offered to all the bereaved parents soon after the tragedy. The subjects were interviewed using the Traumatic Grief Scale. A group of 92 parents/guardians was interviewed 2 months after the event, while the other group of 72 was assessed 7 days later. The second group of bereaved parents also completed the Self Rating Questionnaire (SRQ) and the Ndetei-Othieno-Kathuku scale (NOK). Over 90% of parents from both groups had a yearning for the departed and found themselves searching for him quite often. There was no much difference in terms of symptoms profile or intensity between the two groups. It appears that the counseling offered had minimal impact on the levels of distress.

Key words: Kyanguli School Fire, traumatic grief, post-traumatic stress disorder, counseling

On the night between March 25 and 26, 2001, alleged arsonists started a fire at Kyanguli High School. The fire consumed one of the dormitories in which students were sleeping. Sixty-seven of the students were burnt to death and others sustained various physical injuries. It was not possible to identify 58 of the students who died in the fire, as the bodies were burnt beyond recognition. The parents therefore opted to have a mass burial within the school compound. Soon after the event several interventions were put in place. Counselling was offered starting from the first week after the fire.

In other similar tragedies, a strong similarity between grief in the relatives of the victims and post-traumatic stress disorder (PTSD) has been noted (1). However, a number of differences have been also reported. Symptoms of re-experiencing and preoccupation are not always upsetting and can be consoling (2). Traumatic grief people report avoidance of situations and activities that evoke loneliness and reminders of the loss and not of those that precipitate fear and anxiety (3). Hyperactivity and hyper-vigilance have been found only in a small proportion of cases (4) and relate to scanning of the environment for cues of the deceased (5) rather than to fear of the unwanted experience.

Amichai (6) and Archer (7) reported grief as being heavier with increasing age and consistent with the value of the offspring lost. Previous exposure to traumatic events and the presence of other psychiatric morbidity have been noted to worsen the grief reaction (8). Most previous studies of traumatic grief have largely depended on convenient sampling of individuals presenting for psychiatric and medical help, and have been conducted in Western settings with some samples having less than 40 subjects. The present study reports on traumatic grief in parents and

guardians of the victims of the Kyanguli School fire tragedy.

METHODS

This was a cross-sectional descriptive study. The study subjects included parents and guardians of the deceased students of Kyanguli High School. The subjects were interviewed in two groups of 92 and 72 parents/guardians (referred to as group I and II), the former two months after the event and the latter one week later. Informed consent was obtained from the subjects prior to the interviews. Clearance from the local administration had earlier been obtained. Sociodemographic data were recorded using a questionnaire that had earlier been specifically developed for the purpose and was appropriate for the community that was being studied. Traumatic grief was assessed using the Traumatic Grief Scale developed by Prigerson et al (9). Appropriate translation and back translation from English to the local language had earlier been done. The symptoms of traumatic distress assessed by the Traumatic Grief Scale are grouped in four subcategories that measure both frequency and intensity of occurrence: preoccupation, avoidance, re-experiencing and hyperactivity.

In addition to the Traumatic Grief Scale, the second group of parents/guardians was assessed using the Self Rating Questionnaire (SRQ) (10) and the Ndetei-Othieno-Kathuku scale (NOK) (11). SRQ and NOK are symptoms checklists. The NOK is a culture sensitive instrument developed to pick up anxiety and depression symptoms most commonly complained of by Kenyans but also suitable for other populations with similar social-cultural backgrounds. After the assessment using the structured instruments, the parents/guardians were asked to offer any

additional information that they felt had not been adequately covered in the questionnaires. Data analysis was done using the computer programme Statistical Package for Social Sciences (SPSS) – version 10.0.

RESULTS

Sociodemographic variables

In group I, 47.8% were male and 52.2% female, while in group II there were 60.6% males and 39.4% females respectively. The mean age for both groups was 45 years (range 27-67 years). The living arrangements at home had a similar pattern, the majority (81% and 60% respectively) staying together (when schools were in vacation) with both children and spouse. Only 25% in both groups were in formal employment, with an average monthly income of KSh 5,000.00 (US\$ 60). All the parents, except two, had other remaining children, with only 9.8% and 10.7% respectively having no other remaining child. All the students who died in the fire were male. Of the deceased children, 60% and 66% fell between the 1st and 3rd position in birth order.

Traumatic grief

Table 1 reports the mean scores on the preoccupation, avoidance, re-experiencing and hyperactivity subscales of the Traumatic Grief Scale. The percentage of positive ratings on the individual items of the scale are summarized in Table 2. Although the two groups were interviewed one week apart, their symptom profiles and intensity were similar. On the preoccupation dimension, the scores for both groups were similar.

The intensity of avoidance was negatively related to the number of surviving children (Pearson coefficient = - 0.319; $p = 0.008$). Hyperactivity was also negatively related to the number of surviving children, but not significantly (Pearson coefficient = - 0.231; $p = 0.058$). A similar relationship was demonstrated with the symptom of re-

Table 1 Mean scores on the subscales of the Traumatic Grief Scale among the bereaved parents (the maximum score for each subscale is shown in brackets)

| Subscale | Group I (N=92) | Group II (N=72) |
|-----------------|-------------------|--------------------|
| Preoccupation | | |
| frequency (12) | 10.22 | 10.56 |
| intensity (12) | 9.71 | 10.43 |
| Avoidance | | |
| frequency (32) | 25.91 | 28.12 |
| intensity (32) | 24.93 | 27.61 |
| Re-experiencing | | |
| frequency (4) | 2.36 | 3.71 |
| intensity (4) | 2.27 | 3.60 |
| Hyperactivity | | |
| frequency (20) | 16.25 | 14.32 |
| intensity (20) | 15.46 | 17.17 |

experiencing, but again this was not significant. There was no relation between the intensity of avoidance and the gender of the bereaved. The symptoms of re-experiencing were more common among females compared to males, but the difference was not significant.

Other symptoms of stress

The summary for SRQ scores for group II are summarized in Table 3. On the NOK scale, out of a possible maximum score of 132, a mean score of 74.68 (median 77, mode 132, range 1-32) was obtained. The average score on each of the NOK items was 2.26.

Qualitative data

Typical case vignettes for the various symptoms and dysfunctions are summarized in Appendix I. The subjects freely offered these after completion of the formal questionnaires.

DISCUSSION

The symptoms of traumatic distress as assessed by the Traumatic Grief Scale are similar to those reported among the bereaved following the Oklahoma disaster (12). Over 90% of parents/guardians from both groups had a yearning for the departed and found themselves searching for him quite often. Despite having other children and family members, they felt lonely following the bereavement. There were feelings of detachment and a strong sense of disbelief – 94% for both groups. 73.8% and 90.7% in group I and II felt a strong sense of futility about the future, having difficulties imagining a fulfilling life without the deceased and feeling insecure as a result of the loss.

Although the intensity of re-experiencing was more common among the females than males, the differences were not statistically significant. The explanation for this observation is not clear, though it could indicate the stronger bonding that exists between the mothers and their children.

The symptom profiles in this study and other similar ones suggest a relationship between traumatic grief and PTSD. Other authors (1) have reported low diagnostic agreement between traumatic grief and PTSD. In that study, up to 63% of the respondents with traumatic grief did not meet the diagnostic criteria of PTSD. The nature of the relationship needs to be clarified in further studies.

Although the two groups were interviewed one week apart, there was not much difference in terms of the symptom profile or intensity. Prigerson et al (9) noted that traumatic grief symptoms resolve quadratically with time. Initially there is a steep decline followed by flattening. Since the present study was done two months after the tragedy, this could explain the minimal differences noted between the two groups of parents.

The high levels of trauma found in this study could be

Table 2 Percentage of positive ratings on the individual items of the Traumatic Grief Scale among the bereaved parents

| Item | Positive ratings (%) | |
|---|----------------------|--------------------|
| | Group I (N=92) | Group II (N=72) |
| 1 Do you yearn or long for the deceased? | 98.6 | 97.3 |
| 2 Do you find yourself searching for the deceased? | 88.4 | 94.7 |
| 3 Do you feel lonely as a result of the death? | 97.1 | 100 |
| 4 Was the loss traumatic for you? | 97.1 | 100 |
| 5 Do you feel numb or detached from people? | 83.1 | 89.3 |
| 6 Do you feel stunned, dazed or shocked about the death? | 97.1 | 98.8 |
| 7 Do you feel disbelief over the death? | 94.0 | 94.7 |
| 8 Do you actively avoid reminders of the deceased? | 87.0 | 93.3 |
| 9 Do you feel a sense of futility about the future? | 73.8 | 90.7 |
| 10 Do you feel that life is empty or meaningless? | 71.8 | 86.7 |
| 11 Do you have difficulty imagining a fulfilling life without the deceased? | 95.7 | 96.0 |
| 12 Do you feel that a part of yourself has died? | 94.2 | 98.8 |
| 13 Have you experienced symptoms or recognized harmful behaviour in yourself that are similar to those of the deceased? | 95.6 | 96.0 |
| 14 Do you feel a lost sense of security as a result of the loss? | 95.6 | 96.0 |
| 15 Do you feel a lost sense of control as a result of the loss? | 82.6 | 94.7 |
| 16 Do you feel mistrustful of others as a result of the loss? | 90.1 | 92.0 |
| 17 Do you feel angry or bitter as a result of the loss? | 94.3 | 98.7 |
| 18 Do you feel on edge or anxious? | 91.4 | 97.3 |

Table 3 Mean scores on the Self-Rating Questionnaire (SRQ) scores among the bereaved parents (the maximum score for each dimension is shown in brackets)

| SRQ Dimension | Mean |
|-------------------------|-------|
| Anxiety/depression (20) | 14.76 |
| Psychotic (4) | 1.24 |
| Organic (1) | 0.05 |
| Alcohol (5) | 0.28 |

understood in various ways. Firstly, these children embodied the hopes and future not only of their parents but also of their entire families. They were regarded as important factors in the future economic and emotional well being of their families. Although there is no data on the education status of the other siblings, it is possible that the other children may not have been offered the same privileges. Secondly, the parents felt that they contributed to the death of their own children by having placed them in that particular school. Thirdly, the horrific nature of the event, including failure to identify the bodies of the deceased, may have contributed to the high level of psychotrauma. Bereaved families often like to bury their relatives according to their customs and beliefs. Lastly, the deliberate action by some of the students leading to the deaths was quite painful.

Other forms of psychopathology were assessed by the NOK and the SRQ. The NOK scores of the group II parents showed high levels of stress. The SRQ scores were also high. The instruments recorded high levels of anxiety and depression. Psychotic symptoms were generally low among the parents. Alcohol use to relieve the stress among those affected was not reported by the majority of the parents interviewed.

All the parents interviewed had received some form of counseling and debriefing sessions prior to the study.

Their reactions to these sessions were varied, but the majority reported that they were not helped by the experience. This is reflected in the results, which show high scores on the Traumatic Grief symptoms.

CONCLUSIONS

From the findings of this study it seems that the symptoms of traumatic grief overlap with those of PTSD. The nature of the relationship is currently unclear. Further investigations, such as a follow-up of the parents in this study, would provide an insight into this. It appears that the counseling offered had minimal impact since the levels of distress were still high 2 months after the event. It is therefore debatable whether in dealing with bereaved persons the grief process should be left to take its natural course. Further studies are needed to confirm this.

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APPENDIX I

Examples of responses given by the bereaved parents

Hyperarousal and avoidance

It (the thought) makes me tremble at times, when it comes. At times shaking can develop, though I always try to avoid it.

It is difficult for me to talk about this child. He was the only hope for the family. The father died a long time ago.

Irritability, loss of appetite and numbness

The death of my son has affected me emotionally and in my relations with other people. I have become short-tempered, I have lost appetite and most of the times I feel numb.

It has brought sensitivity to my legs, from my toes upwards and walking has become a problem due to the sensitivity.

Intrusive thoughts

These disturbances have made all my daily routines difficult. When I attend classes I always remember my son.

The feelings of grief keep increasing day by day, more especially when I am alone, because I keep thinking about the deceased and the kind of painful death he underwent.

Sleep disturbances and loss of memory

I spend sleepless nights.

I have constant nightmares.

There have been times when it has not been possible to sleep or concentrate on my activities. A typical example is when I am in class teaching. The thought of the death of my son makes me lose trail of the topic I was teaching and I end up writing the wrong spelling.

Denial and fear

In our family, I started grieving earlier than any other person since I was the first one in my family to hear the news from the radio. My wife and other children could not believe it and kept thinking that he would come back even weeks after the burial. As a result of the death, I feel that I should reschedule my future plans for the family including relocating my home because I feel insecure where we are now living.

Impaired function and social isolation

Since the tragedy occurred people have been coming to my home to console my family, but most of the times I feel that if it were not for the Akamba custom during grieving, I would chase them away. I have difficulties accepting the reality of the death. I keep wishing that it were just a bad dream.

I could not attain my work targets and I was warned twice in one month. I could not concentrate on my work. In many social and church functions, I feel that I should be left alone to “reason” quietly. I do not even want to see people enjoying.

Substance abuse

Since this thing happened, I began to drink alcohol heavily for quite some time.

I tend to like alcohol. It helps me to forget.

Reduced libido

I have no more feelings for my wife.

Amenorrhoea and menorrhagia

My monthly periods have stopped.

I have an increased monthly flow.

Depression and anhedonia

I do not have any interest in anything, and nothing is important to me. I have developed an “I don’t care attitude”. Death is nothing to me.

Startle response

Ever since the child died, the doctor says I am suffering from “too much acid”. I am easily startled and my head feels cold.

Paranoia and suspiciousness

My life has changed since I do not go to work. Every time I see neighbors, I think they are laughing at me because I am always sorrowful.

I have a feeling that people try to avoid me and sometimes I also try to avoid people. I want to be left alone.

References

1. Prigerson HG, Shear MK, Jacobs SC et al. Consensus criteria for traumatic grief. *Br J Psychiatry* 1999;174:67-73.
2. Rees WB. The hallucinations of widowhood. *Br Med J* 1971;4:37-41.
3. Prigerson HG, Bierhals AJ, Wolfson L et al. Case histories of complicated grief. *Omega* 1997;35:9-24.
4. Horowitz MJ, Siegel B, Holen A et al. Criteria for complicated grief. *Am J Psychiatry* 1997;154:905-10.
5. Raphael B, Martinek N. Assessing traumatic bereavement and posttraumatic stress disorder. In: Wilson JP, Keane TM (eds). *Assessing psychological trauma and PTSD*. New York: Guilford, 1997;373-95.
6. Amichai Y. The aging process and grief. *Am J Psychiatry* 1985;2:210-6.
7. Archer J. Grief and pregnancy loss. *Am J Psychiatry* 1999;1:148-51.
8. Smith EM, North CS, McCool RE et al. Acute post disaster psychiatric disorder, identification of persons at risk. *Am J Psychiatry* 1990;147:202-6.
9. Prigerson HG, Katherine M, Jacobs C et al. Grief and its relation to posttraumatic stress disorder. In: David N, Jonathan RT, Joseph Z. (eds). *Posttraumatic stress disorder. Diagnosis, management and treatment*. London: Dunitz, 2000:163-86.
10. Harding TW, Dearango MV, Baltazar J et al. Mental disorders in primary health care: a study of their frequency and diagnosis in four developing countries. *Psychol Med* 1980;10:231-4.
11. Dech H, Richter P, Sandermann S et al. Transcultural research on depression – study concept and preliminary results from a Kenyan population. *Eur Psychiatry* 1996;11(Suppl. 4):287s.
12. Pfefferbaun B, Call JA, Lensgraf SJ et al. Traumatic grief in a convenience sample of victims seeking support services after a terrorist incident. *Ann Clin Psychiatry* 2001;13:1-6.

The World Federation for Mental Health: its origins and contemporary relevance to WHO and WPA policies

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The World Federation for Mental Health (WFMH) is an international, multi-professional non-governmental organization (NGO), including citizen volunteers and former patients. It was founded in 1948 in the same era as the United Nations (UN) and the World Health Organization (WHO). For many years, led mainly by psychiatrists focused on social, peace-related and human rights issues, it was the only international mental health NGO consulting with UN agencies. Since the late 1990s, as a global alliance of national mental health associations focused mainly on traditional mental health issues and on prevention and promotion, it has continued its long-time collaboration with WHO. Its policy concerns and those of international professional associations such as the WPA could be mutually advanced through partnerships aimed at achieving common goals.

Key words: Mental health, policy, WFMH, WHO, WPA

In a recent article in *World Psychiatry* (1), Wolfgang Rutz of the World Health Organization (WHO) Regional Office for Europe made a plea for policy based on the revival of “social psychiatry and social mental health approaches”. He observed that “mental health and peace in a society are strongly linked to each other”, that “mental health impact assessments and consequent analysis of political decisions should become a routine”, and that mental health policy requires a major focus on destigmatization and counteracting discrimination, understood in part as “a reconciliation process” with the mentally ill. Finally, he noted that modern psychiatry cannot abdicate responsibility for “promotional and prevention aspects of mental health”.

Translating this perspective into action has already been enhanced through partnerships between WHO and international non-governmental organizations (NGOs), including both professional associations, such as the WPA, and the World Federation for Mental Health (WFMH), which is a voluntary citizen’s organization including a broad spectrum of professions. To the degree that NGOs are free from obligations to governmental, inter-governmental or corporate entities, their formulation and defense of advocacy positions in response to political decisions may be facilitated. This was given concrete form by Morris Carstairs, WFMH President from 1967 to 1972, in his belief that the only justification for an international NGO was for it to take positions in defense of what it considered just and right.

The purpose of this note is to call the attention of WPA members to the evolving status, current positions, and possible partnerships with WFMH, which shares some of the WPA policy concerns, as well as those of WHO. A detailed account of WFMH history from its founding in 1948 until 1997 may be found elsewhere (2).

THE ORIGINS OF THE WFMH

WFMH has twin roots. One was expressed in its predecessor organization, the International Committee for Men-

tal Hygiene (ICMH), which was first organized in 1919 by Clifford Beers - a former mental hospital patient (3,4) who planned an international network of national mental health associations devoted to “the protection of the insane”- and then reorganized in 1930 with the First International Congress of Mental Hygiene, which brought an estimated 4000 people (psychiatrists, psychologists, health planners and others) to Washington.

The second and more immediate root of the WFMH lay in the post-World War II hope for peace through international collaboration. It culminated in 1948 in the founding of the United Nations (UN) and its associated WHO. The WHO and the United Nations Educational, Cultural and Scientific Organization (UNESCO), founded earlier, were key sponsors of the Third International Congress of Mental Hygiene in London (1948) at which ICMH was transformed into WFMH. This Congress was an opportunity for national mental health associations to resume their international contact interrupted by the war. But the actual impetus for a new international mental health entity came from psychiatrists, who conceived it both as an advocacy agency for world peace and a bridging organization between the UN and the world’s voluntary mental health associations. The concept of a new NGO, and its name, were first suggested by G. Brock Chisholm, the Canadian psychiatrist and former Major General who, in 1948, became the first Director General of WHO. His suggestion came in November 1946 at a small gathering of psychiatrists in New York City in the office of George Stevenson, Medical Director of the US national mental health association, convened by Britain’s John R. Rees, a pioneer in social psychiatry, who had founded the Tavistock Clinic and fostered the use of group methods in the British army.

The WFMH founding document, “Mental Health and World Citizenship”, understood “world citizenship” in terms of a “common humanity” respecting individual and cultural differences, and declared that “the ultimate goal

of mental health is to help [people] live with their fellows in one world". The document was produced at a special meeting in August 1948, and a key contributor was the psychiatrist Harry Stack Sullivan, an ardent proponent of "world mindedness", who arrived just after participating in UNESCO's first conference on reducing the "tensions which cause war". Along with Chisholm, he hoped that a kind of "world loyalty" might replace primary allegiance to a nation or ethnic group. In 1945 he had invited Chisholm to lecture at the William Alanson White Foundation on "The Psychiatry of Enduring Peace and Social Progress". Chisholm's challenge to his fellow psychiatrists was unequivocal: "With the other human sciences psychiatry must now decide what is to be the immediate future of the human race. No one else can" (5). Sullivan published it in the pages of the journal *Psychiatry*, with an editorial calling for a "cultural revolution to end war" to be led by psychotherapists and social scientists (6).

WFMH POLICY

At its very outset, therefore, the WFMH was concerned with educating both the public and influential professionals, and with human relations, with a view both to the health of individuals and that of groups and nations. This fitted the interests of Rees, who agreed to become the first President and then, a year later, Director of the WFMH, remaining in that position through 1961. In 1949 he guided WFMH's first recommendation to WHO (then directed by Chisholm) for the establishment of a mental health section. In the next several years, he wrote, the joint work of the two organizations "did a great deal to ... help change the climate of opinion about mental illness and mental health in many countries" (7). This WFMH emphasis has expanded with education and advocacy carried on since 1992 through an annual World Mental Health Day, celebrated internationally. It has become a vehicle for transmitting information to local mental health associations and agencies about a variety of relevant issues.

As the family of UN affiliated agencies grew, WFMH consulted closely with many of them in a broad and diverse range of projects. However, without UN collaboration, it was able, with the freedom of an NGO, to take independent stands on some issues. Thus, in 1971, it became the world's first mental health organization to take a public stand against the totalitarian exploitation of psychiatry. In the early 1990s it voiced its formal opposition to a UN endorsement of state mechanisms of social control.

Among concerns shared with UN agencies were the mental health casualties of migrants and refugees, children in armed conflict, human rights in relation to biomedical technologies, women's rights, and the rights of oppressed minorities. In 1981-83 WFMH was among the most active NGOs promoting the formation of a UN working group, culminating in the 1991 General Assembly's recognition of

the human rights of psychiatric patients. It worked with the Pan-American Health Organization (PAHO) on the 1990 Declaration of Caracas, dealing with patients' rights and standards of care. In the mid-1990s, the WFMH had a major role in developing the NGO Committee on Mental Health at UN, today a key platform for work with units of the Economic and Social Council (ECOSOC). The Federation's emphasis on promotion and prevention has led to two recent international conferences, with WHO representation, focusing on these issues.

Its past and current concerns with destigmatization and the welfare of persons diagnosed as mentally ill are reflected in the representation of psychiatric hospitalization survivors in its programs, membership, and Board of Directors, and its adoption in 1989 of a Declaration of Mental Health and Human Rights. However, it has not become primarily a patient advocate organization and psychiatrists and other professionals continue to be influential in determining its policy positions. Twenty-three of its 32 presidents between 1948 and 1997 were psychiatrists. In its first fifty years the leaders who held the post of Secretary General or an equivalent designation were all psychiatrists.

PARTNERSHIPS

For many years after its founding, the WFMH was the only NGO of its kind with a close working relationship with UN agencies, particularly the WHO. In recent decades, though, a number of international mental health organizations, often limited to members of particular professions, have developed. In varying degree they have filled needs formerly addressed mainly by WFMH. The WPA, in particular, has become a powerful global force. In 1983, for example, it began to deal with issues surrounding the totalitarian abuse of psychiatry. Now, destigmatization is part of its agenda. Rutz' article in this journal suggests areas in which fruitful partnerships between the WFMH, the WPA and their component members, including national mental health associations, might be considered.

References

1. Rutz W. Rethinking mental health: a European WHO perspective. *World Psychiatry* 2003;2:125-7.
2. Brody EB. The search for mental health. A history and memoir of WFMH 1948-1997. Baltimore: Williams and Wilkins, 1998.
3. Beers C. A mind that found itself. New York: Longmans, 1908.
4. Dain N. Clifford W. Beers, advocate for the insane. Pittsburgh: University of Pittsburgh Press, 1980.
5. Chisholm GB. The psychiatry of enduring peace and social progress. *Psychiatry* 1946;9:1-44.
6. Sullivan HS. The cultural revolution to end war. *Psychiatry* 1946;9:81.
7. Rees JR. Reflections: a personal history and an account of the growth of the World Federation for Mental Health. New York: United States Committee of the World Federation for Mental Health, 1966.

The practice research network: benefits and limitations

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The 5-year experience of the Canadian Psychiatric Association's practice research network (PRN) in providing a window on physician practices in typical clinical settings is reviewed. The strengths of the PRN reside in the active participation of clinicians in self-monitoring as well as in the instrument adaptability and flexibility in addressing current issues of national relevance, including identification of educational needs. The PRN limitations are in the fact that the responders in general have limited research experience and the instrument can provide broad-based answers only. The creation of a national PRN is however an effective strategy in narrowing the gap between practice and research.

Key words: Practice, research, survey, network

A practice research network (PRN) provides a window on patient care and physician practices that are typical of a clinical setting (1). PRNs were developed from the 1960s to the 1980s in the fields of pediatrics and family practice (2). In psychiatry, the American Psychiatric Association was prompted to develop its PRN to address the problem of translation of evidence-based guidelines to practice settings (1). As the Canadian Psychiatric Association (CPA) completed a 5-year experience with its PRN, a review of the benefits and limitations of the methodology involved is timely.

Developing the network for Canadian psychiatry involved a long gestation as we grappled with intertwined issues of focus and funding. An earlier attempt to establish the network as an instrument to conduct industry sponsored clinical trials ended, due to lack of support from industry and physicians alike. Refocussing the main thrust of the network to become an annual audit of the practice of Canadian psychiatrists was positively perceived as responding to an unmet need to track and assess the impact of a range of real world psychiatric practices on patient care in Canada. In these days, where the social values of accountability and transparency are highly upheld, a self-regulating medical specialty like psychiatry needs to live up to rigorous internal standards. In 1997, under the aegis of the Association, we began recruiting a network of volunteers, who had to be psychiatrists, members of the Association, actively practicing in Canada and spending a minimum of 15 hours per week providing patient care. Initially, upon volunteering, each member completed a questionnaire with demographic, training and practice data. The data were compared with a recent national profile of psychiatrists to assess the network's representativeness. In subsequent years, the PRN group was compared to a randomized comparison sample of Association members stratified by region of practice. Additionally, each psychiatrist joining the PRN committed to adhere to the methodological rigor required and to provide feedback to improve the network's performance.

Topics for the annual survey were selected from among

the top issues addressed by the Association at the time. Each year, experts for the designated issues formed a Steering Committee to draft the questions, which were then collated and edited by the PRN coordinators, authors of this paper. The survey questionnaire was reviewed by the Association's Executive Committee. Staffing was drawn from the Association's office and a limited fund set up to cover mailing and analysis expenses.

The PRN expanded from 129 volunteers in 1998 to 291 in 2001. The validity of the process was reinforced by the consistent finding across three surveys showing that the characteristics and responses of our PRN volunteers were very similar to those of the comparison groups of practicing psychiatrists stratified by region of practice. The responders from the latter group contributed to the PRN core membership in the next survey. Confidentiality was assured by the investigators being blind to the responder's names.

SURVEYS CONDUCTED BY THE PRACTICE RESEARCH NETWORK

To date, the three surveys conducted have covered a broad range of topics of interest to the profession. As mentioned, every year a group of topics was selected from among the main tasks promoted by the Association. With subsequent surveys, a maximum of four topics added to a standard practitioner profile was considered optimal. Resisting the tendency to develop lengthy questionnaires, a maximum of ten pages to be filled in approximately 30-45 minutes was adopted to avoid responder fatigue.

The three surveys conducted so far have linked with the following initiatives of importance to the profession.

Monitoring of practice profiles

The training and maintenance of human resources available to meet our patients' needs is critical. The PRN provides a cost-effective method to monitor our profession's demographic characteristics, location and hours of

practice, the number of patients seen as well as the range of activities (3,5,9). While more intensive surveys of our activities are still required from time to time, the PRN provides a yearly snapshot sensitive to abrupt changes.

Appraising the access to services

The issue of access to our services has become a matter of public concern. The PRN records the perceptions of practitioners about the timeliness of this access. Our survey confirmed however that access did not seem to be solely related to the regional ratio of psychiatrists to population density but depended more on practice styles and service organization. This matter therefore remains a challenge for our profession (7).

Evaluation of Clinical Practice Guidelines (CPGs)

The development of CPGs arose out of an attempt to bridge research findings with everyday practice and has been another major endeavor of our profession. While their proliferation and diverse recommendations resulted in some concern on the part of practitioners, CPGs must be viewed as a further step in the journey towards identification of best practices. Evaluating the concordance of current practices with the guidelines and the impact of guideline dissemination on practice is a major contribution of the PRN.

The CPA has so far produced three major sets of CPGs related to mood disorders, schizophrenia and psychotherapy and the PRN was involved in the evaluation of each set (3,6,13).

A wide range of suggestions arose from each of our CPG survey activities. Recommendations from practitioners included the need for simplified and more user friendly algorithms, the need to account for prevailing comorbidities (4) as well as areas of under-use either in medication or psychosocial approaches (6). Gender differences in practice were highlighted as one of the findings in our psychotherapy survey (13).

A PRN not only evaluates established CPGs, but informs us about the practice areas in need of such guidelines. For example, in our surveys, the need for CPGs was identified for anxiety disorders, where the relative merits of various strategies continue to be an area of controversy (3). The same need was identified for guidelines regarding the assessment and management of the suicidal patient as well as the violent patient (4,12). A gripping finding was the toll exacted by a suicide or an act of violence not only understandably on the patient and his or her family but on the therapist as well.

An important recent development has also been the promotion of a shared care program of delivery for mental health/illness services with our colleagues in family practice settings. The PRN helped identify perceptions by psychiatrists as to the opportunities and challenges offered by the partnership with family practice. It is hoped that a

comparison of these perceptions with those of family practitioners will be the topic of a future survey.

Opinion polls guiding our relationship with industry

The CPA guidelines on the ethical conduct of clinical trials were tested on our membership. Of concern at the time, one half of the PRN group and an even lower proportion of the control group acknowledged awareness of these guidelines (4).

The process of developing practice guidelines depended on the availability of external funding mainly from industry. This funding was largely at arm's length from industry and the evaluation of such guidelines by an independent body like the PRN enhanced the credibility of the process.

Matters related to physician health and well-being

The motto of 'physician heal thyself' appears regularly in the press and the PRN proved to be a good barometer for issues troubling the well-being of our colleagues. Their subjective responses were also compared to the data arising from a national survey of professionals (11).

A cornerstone of Maintenance of Competence (MOCOMP)

The major MOCOMP effort guiding the medical profession into the 21st century is based on regular evaluation of professional education needs and the PRN can become a national cornerstone for these evaluations. Analysis of the responses to the issues addressed as well as direct questions about continuing education needs and preferred approaches can provide the needs assessment critical to successful educational endeavors (4). MOCOMP credits for psychiatrists' participation in the process are an appropriate incentive.

LIMITATIONS

'Broad brush' answers

Inherent in the PRN methodology is the fact that the cross-sectional survey is limited to be a 'listening post' on our practice. While practitioners' perceptions about the relative value of an assessment or treatment strategy versus another can be gathered, the method does not lend itself to a rigorous testing of these perceptions. The gold standard for that expectation remains the clinical trial, but the PRN provides the gold standard for an overview of a 'real world' practice outside of the controlled academic research settings. This dimension has been largely ignored up till now.

The quality of responses from untrained responders

The PRN methodology targeting clinicians, some with limited research experience and very busy practices, raises

the questions of validity and reliability of the responses. Confidence is raised by the general concordance of responses between the PRN and comparison groups over three surveys.

While the PRN respondents reported being involved in more research hours weekly, their perceptions of the amount of time spent in clinical practice did not differ from the comparison group. This concordance in practice seems to be a remarkable characteristic in Canadian psychiatry. On the other hand, the responses of 'real world' clinicians to 'real world' clinical issues have traditionally been under-reported and the PRN provides some remedy to this deficiency.

Tailoring the questions to the target populations

Our experience over three surveys led us to become weary of certain types of questions. In our surveys, Likert-type questions often appeared to elicit non-discriminating responses clustered around the mid-point. An impression of social desirability response could not be avoided. As with public opinion polls, the better questions were those eliciting as clear a choice as possible.

The gathering of qualitative information

Our experience with commentary responses to the PRN suggested that the use of qualitative or evaluative responses be stringently limited to only the essential questions. A disproportionate amount of coding time (over 85%) went into the preparation of this information, which typically far outweighed its usefulness. A major culprit was the 'other' category, which was often tacked onto too many multiple response questions. Participants' responses to open-ended items were often more informative, but required a higher level of experience with qualitative coding and linguistics, eventually requiring the engagement of the services of a Ph.D. in literature!

Again this type of information is costly to collect and should be limited to only the very essential. It should however also be recognized that this approach was crucial in defining the various facets of response to a patient's suicide (6).

Survey fatigue

Busy practitioners are weary of irrelevant questionnaires distracting them from their clinical tasks. The issue of responder fatigue from frequent or lengthy questionnaires was a major consideration. Timing the mailing of the questionnaire away from other Association demands was important. As already noted, the length of the questionnaire and time required to fill it influenced response rate and we learned to simplify the questions and limit the forms to 10 pages. A once yearly frequency for our Association has been striven for and our enrolment of the

responders as the PRN group for the next survey also helped reduce fatigue by ensuring a rotation among the membership surveyed.

CONCLUSION

As we pass the PRN torch to another team, it is our view that pursuing this effort is an essential element in furthering the clinical practice and integrity of our profession. Our experience with the PRN so far provides a foundation to be built upon. Other countries, such as the United States, the United Kingdom and Australia, have established an Office of Psychiatric Research Network/Peer Review with independent funding from government and other sources. Our PRN project relies on limited funding from our Association and as such may provide an affordable strategy for countries with more limited resources.

With newly created partnership between professional associations and consumer groups, this activity should be advocated as a funding priority not only to monitor the practices of psychiatrists but of allied psychiatric health professionals as well (14). The PRN offers practicing clinicians an opportunity not only to participate in research activities but also set a research agenda for psychiatry. Psychiatric practices vary markedly around the world and 'real world' practices differ from textbook recommendations. The PRN is a timely strategy to assess the relevance of current knowledge and narrow the gap between practice and research.

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References

1. Zarin DA, Pincus HA, West JC et al. Practice-based research in psychiatry. *Am J Psychiatry* 1997;154:1199-208.
2. Green LA, Wood M, Becker L et al. The Ambulatory Sentinel Practice Network: purpose, methods, and policies. *J Fam Pract* 1984;18:275-80.
3. Swinson RP, Kennedy SH, Kusumakar V et al. The Canadian Psychiatric Association's Practice Research Network: findings from the First Project 1998 - Part I. *Bull Can Psychiatr Assoc* 1999;31:49-52.
4. Links P, Langley J, Teehan M et al. The Canadian Psychiatric Association's Practice Research Network: findings from the First Project 1998 - Part II. *Bull Can Psychiatr Assoc* 1999;31:52-5.
5. el-Guebaly N, Atkinson M. The Canadian Psychiatric Association Research Network: findings from the Second Project, 1999. Part I: The practitioner's profile. *Bull Can Psychiatr Assoc* 2000;32:162-3.
6. Addington D, el-Guebaly N, Chandarana P et al. The Canadian Psychiatric Association Research Network: findings from the Second Project, 1999. Part II: Canadian Clinical Practice Guide-

- lines for the Treatment of Schizophrenia: adherence and awareness. *Bull Can Psychiatr Assoc* 2000;32:164-7.
7. el-Guebaly N, Atkinson M. The Canadian Psychiatric Association Research Network: findings from the Second Project, 1999. Part III: Access to psychiatrists' care. *Bull Can Psychiatr Assoc* 2001;33:9-12.
 8. Kates N, Craven M, Atkinson M et al. The Canadian Psychiatric Association Research Network: findings from the Second Project, 1999. Part IV: How psychiatrists view their relationships with family physicians. *Bull Can Psychiatr Assoc* 2001;33:13-5.
 9. el-Guebaly N, Atkinson M. The CPA's Practice Research Network - Part I: Findings from the third project, 2001. *Bull Can Psychiatr Assoc* 2002;34:39-40.
 10. el-Guebaly N, Atkinson M. The CPA's Practice Research Network - Part II: Psychiatrist perceptions of medical assessments related to life and disability insurances. *Bull Can Psychiatr Assoc* 2002;34:41-2.
 11. el-Guebaly N, Atkinson M, Patten S. The CPA's Practice Research Network - Part III: How stressful is the practice of psychiatry? *Bull Can Psychiatr Assoc* 2002;34:43-5.
 12. Bourget D, el-Guebaly N, Atkinson M. The CPA's Practice Research Network - Part IV: Assessing and managing violent patients. *Bull Can Psychiatr Assoc* 2002;34:25-27.
 13. Leszcz M, MacKenzie R, el-Guebaly N et al. The CPA's Practice Research Network - Part V: Canadian psychiatrists' use of psychotherapy. *Bull Can Psychiatr Assoc* 2002;34:28-31.
 14. Atkinson MJ, el-Guebaly N. Research productivity among Ph.D. faculty members and affiliates responding to the Canadian Association of Professors of Psychiatry and Canadian Psychiatric Association Survey. *Can J Psychiatry*, 1996;41,509-12.

Women's mental health in Pakistan

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In Pakistan, societal attitudes and norms, as well as cultural practices (Karo Kari, exchange marriages, dowry, etc.), play a vital role in women's mental health. The religious and ethnic conflicts, along with the dehumanizing attitudes towards women, the extended family system, role of in-laws in daily lives of women, represent major issues and stressors. Such practices in Pakistan have created the extreme marginalisation of women in numerous spheres of life, which has had an adverse psychological impact. Violence against women has become one of the acceptable means whereby men exercise their culturally constructed right to control women. Still, compared to other South Asian countries, Pakistani women are relatively better off than their counterparts.

Key words: Pakistan, women's mental health, cultural practices, honor-killing, stove-burns, violence

The women's movement in Pakistan in the last 50 years has been largely class bound. Its front line marchers voiced their concerns about issues mainly related to the urban-middle class woman. It is only in the last few years that rural women's issues like 'Karo Kari' (honour killing) and rape have been brought to light. Feudal/tribal laws of disinheritance, forced marriages and violence against women (acid-throwing, stove-burning homicide and nose-cutting) in the name of honour are being condemned by non-governmental organizations and human rights activists in the cities. Still a vast majority of the women in the rural areas and urban slums are unaware of the development debates.

The urban Pakistani women in many aspects are almost at par with the women of developed countries. In the rural scenario, the picture is entirely different. It is archaic, brutal and clearly oppressive. These trends often seep into the urban lives of women through migratory movements of rural population, which has yet to adjust to urban ways.

At the societal level, restricted mobility for women affects their education and work/job opportunities. This adds to the already fewer educational facilities for women. Sexual harassment at home, at work and in the society has reached its peak. Lack of awareness or denial of its existence adds to further confine women to the sanctity of their homes. Violence against women further adds to restriction of mobility and pursuance of education and job, thereby lowering prospects of women's empowerment in society.

At the family level, birth of a baby boy is rejoiced and celebrated, while a baby girl is mourned and is a source of guilt and despair in many families. Boys are given priority over girls for better food, care and education. Subservient behaviour is promoted in females. Early marriage (child-brides), *Watta Satta* (exchange marriages), dowry and *Walwar* (bride price) are common. Divorcees and widows are isolated and considered 'bad omens', being victims of both male and female rejection especially in villages. Marriage quite often leads to wife-battering, conflict with

spouse, conflict with in-laws, dowry deaths, stove burns, suicide/homicide and acid burns to disfigure a woman in revenge.

VIOLENCE AGAINST WOMEN

In Pakistan, there are cultural institutions, beliefs and practices that undermine women's autonomy and contribute to gender-based violence. Marriage practices can disadvantage women, especially when customs such as dowry and bride's price, *Watta Satta* and marriage to the Quran (a custom in Sindh where girls remain unmarried like nuns to retain family property in the family) exist. In recent years dowry has become the expected part of marriage. This increasing demand for dowry, both before and after marriage, can escalate into harassment, physical violence and emotional abuse. In extreme cases homicide or "stove-burns" and suicides can provide husbands an opportunity to pursue another marriage and consequently more dowry.

Women are confined to abusive relationships and lack the ability to escape their captors due to social and cultural pressures. Parents do not encourage their daughters to return home for fear of being stigmatized as a divorcee, which tantamount to being a social pariah. Moreover, if a woman leaves her husband, her parents have to repay him to compensate his loss. Cultural attitudes towards female chastity and male honour serve to justify violence against women.

Violence against women is very common in Pakistan. The violation of women's rights, the discrimination and injustice are obvious in many cases. A United Nations research study (1) found that 50% of the women in Pakistan are physically battered and 90% are mentally and verbally abused by their men. A study by Women's Division on "Battered Housewives in Pakistan" (2) reveals that domestic violence takes place in approximately 80% of the households. More recently the Human Rights Commission report (3) states that 400 cases of domestic violence are reported each year and half of the victims die.

In Balochistan and Sindh provinces, *Karo Kari* is practiced openly. A woman suspected of immorality is declared a *Kari* while the *Karo* is a man declared to be her lover. A woman suspected of adultery or infidelity is liable to face the death penalty at the hands of her husband or in-laws. Usually the killer goes scot-free as he is regarded to have committed the crime in order to retrieve the lost family honour, which a woman is expected to uphold at all costs.

Watta Satta is also a tradition in many families in Punjab and Sindh, whereby a girl is married off to her sister-in-law's brother. Such an arrangement often leads to a complicated situation, since a woman ends up becoming a mere object of revenge in the instance that her brother mistreats or physically abuses his wife.

Sadistic urges may be satisfied by a man by totally humiliating as well as disfiguring his wife. Women who are victims of this particular form of violence are usually young and attractive.

Hundreds of women are disfigured or die of stove-burns every year. The victims are usually young married women and the aggressors include husbands and in-laws. The motive behind stove burning is to get rid of the woman and remarry for more dowries or have an heir for the family.

Battering or "domestic violence" or intimate partner abuse is generally part of the patterns of abusive behaviour and control rather than an isolated act of physical aggression. Partner abuse can take a variety of forms, including physical violence, assault such as slaps, kicks, hits and beatings, psychological abuse, constant belittling, intimidation, humiliation and coercive sex. It frequently can include controlling behavior such as isolating women from family and friends, monitoring her movements and restricting her access to resources. Physical violence in intimate relationship is almost always accompanied by psychological abuse and in one-third to one-half of cases by sexual abuse.

A woman's response to abuse is often limited by the options available to her. Women constantly cite reasons to remain in abusive relationship: fear of retribution, lack of other means of economic support, concern for the children, emotional dependence, lack of support from family and friends and the abiding hope that the husband may change one day. In Pakistan divorce continues to be a taboo and the fear of social stigma prevents women from reaching out for help. About 70% of abused women have never told anyone about the abuse.

The psychological consequences of abuse are more severe than its physical effects. The experience of abuse erodes women's self-esteem and puts them at a greater risk for a number of mental disorders like depression, post-traumatic stress disorder, suicide, alcohol and drug abuse.

Children who witness marital violence face increased risk for emotional and behavioural problems, including anxiety, depression, poor school performance, low self-

esteem, nightmares and disobedience. Boys turn to drugs and girls become severely depressed and sometimes totally refuse to get married. Children under 12 years have learning, emotional and behavioural problems almost 6-7 times more compared to children of non-abusive parents.

Health care providers can play a key role. They must recognize victims of violence and help them by referring to legal aid, counsellors and non-governmental organizations. They can prevent serious conditions and fatal repercussions. However, many doctors/nurses do not ask women about the experience with violence and are not prepared to respond to the needs of the victims.

A variety of norms and beliefs are particularly powerful perpetrators of violence against women. These include the notions that men are inherently superior to women, that it is appropriate for men to discipline women, and that women's sexual behaviour is linked to male honour. Nobody is expected to intervene on behalf of the victim as such issues are considered private matters to be resolved by the immediate parties themselves.

Programs designed to change these beliefs must encourage people to discuss rather than antagonize or alienate them by appearing to 'demonize' men. A good tool is to encourage people to develop new norms by using techniques such as plays on TV and theatre.

PSYCHIATRIC ILLNESS IN PAKISTANI WOMEN

A large study at Jinnah Post Graduate Medical Center, Karachi back in early 1990s (4) showed that twice as many women as men sought psychiatric care and that most of these women were between 20s and mid 40s.

Another 5-year survey (1992-1996) at the University Psychiatry Department in Karachi (Agha Khan University/Hospital) (5) showed that out of 212 patients receiving psychotherapy, 65% were women, 72% being married. The consultation stimuli were conflict with spouse and in-laws. Interestingly, 50% of these women had no psychiatric diagnosis and were labeled as 'distressed women'. 28% of women suffered from depression or anxiety, 5-7% had personality or adjustment disorders and 17% had other disorders.

The 'distressed women' were aged between 20 to 45. Most of them had a bachelor's degree and had arranged marriage relationships for 4-25 years with 2-3 kids, and the majority worked outside home (running small business, teaching or unpaid charitable community work or involved in voluntary work). Their symptoms were palpitations, headaches, choking feelings, sinking heart, hearing weakness and numb feet.

A study on stress and psychological disorders in Hindu Kush mountains of North West Frontier Province of Pakistan (6) showed a prevalence of depression and anxiety of 46% in women compared to 15% in men.

A study on suicidal patients (7) showed that the majority of the patients were married women. The major source

of suffer was conflict with husband (80%) and conflict with in-laws (43%).

A study of parasuicide in Pakistan (8) shows that most of the subjects were young adults (mean age 27-29 years). The sample showed predominance of females (185) compared to males (129), and the proportion of married women (33%) was higher than males (18%). Housewives (55%) and students (32%) represented the two largest groups among females. Most female subjects (80%) admitted problems with spouse.

A four-year survey of psychiatric outpatients at a private clinic in Karachi (9) found that two thirds of the patients were females and 60% of these females had a mood disorder. 70% of them were victims of violence (domestic violence, assault, sexual harassment and rape) and 80% had marital or family conflicts.

CONCLUSIONS

Pakistani women are relatively better off than their counterparts in other developing countries of South Asia. However, fundamental changes are required to improve their quality of life. It is imperative that constructive steps be taken to implement women friendly laws and opportu-

nity be provided for cross-cultural learning. Strategies should be devised to enhance the status of women as useful members of the society. This should go a long way to improving the lives and mental health of these, hitherto "children of a lesser God".

References

1. Tinker GA. Improving women's health in Pakistan. World Bank, Karachi, 1999.
2. National Commission on the Status of Women. Report of the status on women in Pakistan. Islamabad, 1997.
3. Rehman IA. The legal rights of women in Pakistan: theory and practice. Human Rights Commission of Pakistan, Karachi, 1998.
4. Naem S. Psychological risk factors for depression in Pakistani women. Thesis, College of Physicians and Surgeons, 1990.
5. Zaman R. Five-year survey (1992-1996), University Psychiatry Department, Karachi. Unpublished manuscript.
6. Mumford D, Nazir M, Jilani FM. Stress and psychiatric disorders in Hindu Kush: a community survey of mountains villages in Chitral, Pakistan. *Br J Psychiatry* 1996;170:473-47.
7. Niaz U. Human rights abuse in family. *Journal of Pakistan Association of Women's Studies* 1994;3:33-41.
8. Khan MM, Islam S, Kundi AK. Parasuicide in Pakistan: an experience at University Hospital. *Acta Psychiatr Scand* 1996;93:264-7.
9. Niaz U. Contemporary issues of Pakistani women: a psychosocial perspective. *Journal of Pakistan Association Women's Studies* 1997;6: 29-50.

September 2005: Cairo

PEDRO RUIZ

WPA Secretary for Meetings

Following in the footsteps of the 12th World Congress of Psychiatry, organized by the WPA and held in Yokohama, Japan, in August 2002, the 13th World Congress of Psychiatry, also organized by the WPA, will be held in Cairo, Egypt, on September 10-15, 2005. This World Congress of Psychiatry is the first ever held in the African continent. It additionally seems to be the most memorable World Congress of Psychiatry ever held by the WPA. Never before had a World Congress of Psychiatry been held at the four crossroads of the world, that is, where East meets West and South meets North. Also, in the most ancient and historic cornerstone of civilization of our planet. The fact that close to 2000 psychiatrists and mental health professionals from all over the world positively responded to our first announcement of this Congress is very significant with respect to the enthusiasm shown by the profession and field vis-à-vis this Congress.

Currently, all pertinent structures of the organization of this Congress are already in place and actively working towards making this Congress the most unforgettable one in the history of the WPA. For instance, the Organizing, Supervisory and Scientific Committees, as well as those for Finance, Partnership, Evaluation, Continuing Medical Education (CME), Publications, Artistic and Cultural Activities, Review of Scientific Submissions and Program Coordination, Fellowships and Young Participants, Coordination with WPA Scientific Sections, Public Relations and Social Events, and the Local Organizing and Advisory Committees, are all already fully operational. Additionally, an International Advisory Committee has been selected and appointed, with representation from all key scientific areas of the pro-

fession and field. The theme of the Congress, selected by the WPA President, Ahmed Okasha, with wide input from all relevant sectors of the WPA, beautifully represents the significance and unique message of this Congress: "5000 Years of Science and Care: Building the Future of Psychiatry".

The second announcement was recently sent out to the field, calling for submissions of scientific proposals, and providing key and relevant information about the venue of the Congress and also about accommodations as well as socially-related activities. Given the reputation of the city and country selected for this Congress, the time of the year, and the popularity of this scientific event, it is not too early to start making final plans to attend and participate in this very unique and significant scientific meeting. Two outstanding organizations have been contracted to assist in all pertinent issues related to this Congress. One is Tilesa, which pro-

vides scientific and technical secretariat services for the Congress, and the other is Emeco Travel, which offers accommodation and tourist services. Both can be accessed via the Congress website (www.wpa-cairo2005.com).

As WPA Secretary for Meetings and as Chairperson of the Organizing Committee for the Cairo Congress, I feel both very privileged and enthusiastic for the opportunity to actively participate and assist in making the 13th World Congress of Psychiatry one of the best ever scientific events in the history of the WPA. The unique opportunity to serve both the WPA and the Egyptian Psychiatric Association in this very relevant and important scientific event for the profession and field at a worldwide level, makes me very proud and happy. Additionally, this Congress is taking place in Africa, in the Middle-East, in an Arab country, and in the Mediterranean Region of the world. What else can we ask for and expect as psychiatrists and mental health professionals. I look forward to seeing all of you in Cairo in September 2005.

WPA finance forecast 2004

SAM TYANO

WPA Secretary for Finance

The financial policy of a world organization changes according to circumstances, to the general world economic situation, to the needs of its members, to the general policy decided by its executive committee and to the hierarchy of priorities decided by its general assembly.

The main role of the WPA Secretary for Finance is to propose new resources to fund the activities of the Association, applying the policy dictated by the Executive Committee, following the advice provided by the Board, and taking into account the needs of the Member Societies, the WPA Committees, the Secretariat

and the Executive Committee members.

The regular sources of income of the WPA budget are the membership dues, the World Congresses, other international and regional congresses, royalties from publications, and the 15% overhead taken from each educational program budget.

The standard expenses of the WPA are those related to the Secretariat (including salaries and office expenses), those related to the Executive Committee (meetings and assignments), the support to Zonal Representatives and Standing and Operational Committees, the maintenance of the Educational Coordination Center, and the expenses for public relation materials.

The first portion of the income is

represented by the membership dues. The WPA policy has been to adjust dues according to the economic situation of each country. Although it is apparent that certain Member Societies can afford to pay more, while others struggle to pay the current fee, the problem of equity is difficult to resolve. In addition, there is a shift in worldwide economic situation every 2-3 years. The total amount of fees, as of today, constitutes 25% of the income and barely covers the salaries of the Secretariat staff.

The most constant and steady income of the WPA is that represented by royalties from publications. Due to the very intensive and significant activity of the Secretary for Publications, the number of books published by the WPA is increasing every year. Many members of the WPA worldwide also receive *World Psychiatry*, the official journal of the Association, directly to their postal addresses, free of charge.

Another extremely significant source of income during the past eight years has been the overhead of each educational program. According to the Finance Committee decision, approved by the General Assembly, this overhead recently increased to 15%. However, the number of programs has decreased during the past few years and the only remaining one is the Global Child Mental Health Program. Therefore, the charges from the educational programs, used to cover various expenses, are sorely lacking.

One of the main resources of any

international association is obviously represented by meetings, conferences and congresses. The last two World Congresses did not meet financial expectations based on technical grounds. Here is one of the primary vital confrontations between substantial needs and ideological and political considerations. In essence, it is very easy to attract numerous people to participate at inviting tourist sites throughout the world. Alternatively, it should be of top priority to bring WPA activities to all corners of the world, allowing each Member Society to meet with WPA scientific, educational and policy makers, while simultaneously exposing its own viewpoints, presenting its country's mental health infrastructure and demonstrating the way the specific culture has integrated modern mental health concepts while conserving its basic beliefs, roots and cultural institutions.

The discussion on where future congresses and conferences should be held is open and continuing. The destination must be attractive and secure enough for people, inviting to the pharmaceutical industry and, simultaneously, maintain equity from a financial perspective. This resource is imperative and the utmost must be done to maintain its flow. One way of doing so, that is already being implemented, is the Corporate Supporters Sponsorship Program. The basic concept, which is the essence of the project, is to obtain enough cash flow between two World Congresses to maintain WPA activities and, in addition,

to assure that the WPA will receive its own portion from the World Congress budget. The host of the World Congress also benefits, as the amount of money paid by the industry is given enough time in advance to ensure the necessary preparations for the event.

One of our major concerns is of course to establish the priorities of our Association, which should be reflected by the policy of distributing the resources. Each topic seems important and each activity has its priority in the eyes of the one who proposes it, but this priority is not necessarily agreed upon by other people. This discrepancy creates sometimes some involuntary misunderstandings.

Finances are not the factor which determines the values which will be defended by our Association, but certainly play a role in the determination of the quality and the amount of investment in each activity. I invite all Member Societies to take part in this discussion. All their comments will be brought and discussed in the Finance Committee and presented to the Executive Committee. My role is just to implement the Executive Committee financial decisions and to try to accommodate them to the financial situation of the Association. I hope that in the future we will be able to find more resources, in order to enlarge the educational, scientific and training programs, side by side with being able to offer the well-known feeling of getting back at least as much as we have invested.

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