World Psychiatry

OFFICIAL JOURNAL OF THE WORLD PSYCHIATRIC ASSOCIATION (WPA)

Mental disorders as "brain diseases" and Jaspers' legacy

Mario Maj

Department of Psychiatry, University of Naples SUN, Naples, Italy

This year we celebrate the 100th anniversary of the publication of the first edition of Karl Jaspers' *General Psychopathology* (1), and some authors (2,3) have already noticed significant analogies between the historical moment in which that classical text appeared and the present one. The most striking analogy is that nowadays, exactly like one century ago, the enthusiasm brought about by a period of exceptional progress of research in neurosciences is being followed by some disillusionment, due to the limited relevance of that progress to the elucidation of the pathophysiology of mental disorders. To this disillusionment, the psychiatric field is now reacting in a way that resonates in several respects with Jaspers' analysis, making a revisitation of his *General Psychopathology* extremely useful.

In line with Jaspers' analysis is first of all the current acknowledgment of the limitations of the scientific paradigm of 19th century medicine (identifying signs and symptoms, constructing syndromes, taking course into account, and then looking for biological processes that explain the syndromes), when applied to psychiatry. That paradigm seemed to work in the case of general paresis of the insane. However, as Jaspers notices, that disease was not an appropriate "model for clinical psychiatric research" (1, p. 566). In fact, the symptomatic psychoses occurring in general paresis were "in no way different from other psychoses associated with brain disease, neither in the psychological symptoms nor in the sequence of psychic phenomena throughout the illness" (1, p. 566). Apart from general paresis, Jaspers argues, "there has been no fulfillment of the hope that clinical observation of psychic phenomena, of the life-history and of the outcome might yield characteristic groupings which would subsequently be confirmed in the cerebral findings" (1, p. 568). "The idea of the disease-entity is in truth an idea in Kant's sense of the world" (1, p. 569). Even in the cases of schizophrenia and manic-depressive illness, "one is always confined to types" (1, p. 611), that is, "fictitious constructs which in reality have fluid boundaries" (1, p. 560).

These arguments are now being reproposed by several leaders in the psychiatric field. According to First (4, p. 13), "besides the identification of the spirochete as the etiological factor underlying the psychotic disorder general paresis of the insane, the reductionistic 19th-century disease model has not been applicable to any other psychiatric syndrome". For Kupfer et al (5, p. xix),

"reification of DSM-IV entities, to the point that they are considered to be equivalent to diseases, is more likely to obscure than to elucidate research findings". According to Hyman (6, p. xix), "scientists attempting to discover genetic or neural underpinnings of disease have all too often reified the disorders listed in DSM-IV-TR as 'natural kinds'". For Charney et al (7, p. 34), "many, and perhaps most, of the current symptom clusters of DSM will ultimately not map onto distinct disease states".

A second element of the current debate resonating with Jaspers' analysis is the critique of Griesinger's maxim (8) that "all mental illnesses are cerebral illnesses" (see 9 in this issue of the journal). This statement, according to Jaspers, "is nothing but dogma" (1, p. 496). "We know that in general no psychic event exists without the precondition of some physical basis; there are no 'ghosts'" (1, p. 459), but "cerebral diseases are just one of the causes of psychic disturbance among many" (1, p. 496). Indeed, according to Bolton (10), a damage to the neural substrate is not necessary for failure of psychological function. Building upon Dennett's model (11), Bolton points out that, just as irrational moves by a chess-playing computer may be explained not only from a "physical stance" (short-circuits, over-heating, blown fuses) but also from a "design stance" (suboptimal programming), mental dysfunction may involve lesions or functional abnormalities of the brain, but also, for instance, maladaptive operating rules acquired by learning. These maladaptive rules will certainly be implemented through the brain, but this does not mean that an actual dysfunction of neural circuits must be present (a suboptimal chess-playing program can be implemented through an intact computer machinery). Of course, as Jaspers recognizes, primarily psychic events may themselves produce a brain dysfunction ("cerebral changes may also be the result of primary psychic phenomena") (1, p. 496), but this is a possibility, not a prerequisite. Analogous to Dennett's example revisited by Bolton is that proposed by Kendler (12, p. 435) of the young man performing a statistical analysis on his computer and getting the wrong result because he has made a mistake in his statistical program. He tries to solve the problem by taking off the back of his computer, pulling out the motherboard and reaching for his soldering iron, hoping to find a loose connection to solder, while in fact there is none.

Even when a primary morbid cerebral process is actually occurring – Jaspers argues – there is not a one-to-one

correspondence between that cerebral process and the psychic event that will result. "We do not know a single physical event in the brain which could be considered the identical counterpart of any morbid psychic event. We only know conditioning factors for the psychic life; we never know *the* cause of the psychic event, only *a* cause" (1, p. 459). Furthermore, "the specific psychic disposition of the individual conditions the specific type of psychic reaction to the cerebral disease process" (1, p. 458), so that a given cerebral process may correspond to a variety of psychic events. These arguments resonate with the current acknowledgment that "any given [mental] disorder can be marked by disruptions among multiple mechanisms, and one particular mechanism may contribute to the psychopathology of a large number of disorders" (13, p. 632). Even the notion of a final common neural pathway leading from multiple determinants to a single clinical syndrome is "an empirical matter, not an a priori one" (14, p. 10).

In this context, the basic heterogeneity of mental disorders should not be overlooked. "Contemporary neo-Kraepelinian American psychiatry… practices as if there were biological commitments to over 300 DSM-defined entities" (15, p. 7), while the biological model may apply only to a few mental disorders, for instance, "schizophrenia, manic-depressive illness, melancholic depression and obsessive-compulsive disorder" (16, p. 56). These recent statements resonate with Jaspers' classification of mental disorders into three groups — cerebral illnesses (such as Alzheimer's disease), major psychoses (such as schizophrenia and manic-depressive illness), and personality disorders (including neurotic syndromes and abnormal personalities) — which are "essentially different from each other" (1, p. 610) and not equally amenable to biological research (those of the third group may just represent "variations of human nature").

Finally, Jaspers' emphasis on patients' "working through the illness" (1, p. 416), of which the "laborious development of a delusional system out of delusional experiences" is the best example, resonates with the contemporary notion that "the role of the person in mental disorder is not peripheral, merely as a passive victim of a disease to be fixed by medicine" (17, p. 182), and that person–disorder interactions are crucial in the shaping of psychopathological symptoms. So, not only patients' primary subjective experiences should be a major focus of psychopathological and neuroscientific enquiry, but patients' "attitude to their illness" (1, p. 414) can represent an important target for both research and intervention.

These are just a few examples of basic philosophical issues in psychiatry that are as relevant today as they were one century ago. They suggest that, although our diagnostic systems may be devised as "atheoretical", contemporary psychiatry does need a guiding philosophy. "If anyone thinks he can exclude philosophy and leave it aside as useless, he will eventually be defeated by it in some obscure form or other" (1, p. 770). This is one of the reasons (see also 18–22) why a revisitation of Jaspers' *General Psycho-*

pathology, on the occasion of the 100th anniversary of the publication of its first edition, may represent a useful exercise for everyone involved in psychiatric research and practice.

References

1. Jaspers K. Allgemeine Psychopathologie. Berlin: Springer, 1913. English translation of the 7th edition: General psychopathology. Baltimore: Johns Hopkins University Press, 1997.
2. Fulford KWM, Morris KJ, Sadler JZ et al. Past improbable, future possible: the renaissance in philosophy and psychiatry. In: Fulford KWM, Morris KJ, Sadler JZ et al (eds). Nature and narrative: an introduction to the new philosophy of psychiatry. Oxford: Oxford University Press, 2003:1-41.
3. Broome MR. Jaspers and neuroscience. In: Stanghellini G, Fuchs T (eds). One century of Karl Jaspers' General Psychopathology. Oxford: Oxford University Press (in press).
4. First MB. The National Institute of Mental Health Research Domain Criteria (RDoC) project: moving towards a neuroscience-based diagnostic classification in psychiatry. In: Kendler KS, Parnas J (eds). Philosophical issues in psychiatry II: nosology. Oxford: Oxford University Press, 2012:12-8.
5. Kupfer DJ, First MB, Regier DE. Introduction. In: Kupfer DJ, First MB, Regier DE (eds). A research agenda for DSM-V. Washington: American Psychiatric Association, 2002:xv-xxiii.
6. Hyman SE. Foreword. In: Phillips K, First M, Pincus H (eds). Advancing DSM: dilemmas in psychiatric diagnosis. Washington: American Psychiatric Association, 2003:xi-xix.
7. Charney DS, Barlow DH, Botteron K et al. Neuroscience research agenda to guide development of a pathophysiologically based classification system. In: Kupfer DJ, First MB, Regier DE (eds). A research agenda for DSM-V. Washington: American Psychiatric Association, 2002:31-83.
8. Griesinger W. Die Pathologie und Therapie der psychischen Krankheiten. Stuttgart: Krabbe, 1845.
9. Bolton D. Should mental disorders be regarded as brain disorders? 21st century mental health sciences and implications for research and training. World Psychiatry 2013;12:24-5.
10. Bolton D. What is mental disorder? An essay in philosophy, science, and values. Oxford: Oxford University Press, 2008.
11. Dennett D. Brainstorms: philosophical essays on mind and psychology. Sussex: Harvester, 1979.
12. Kendler KS. Toward a philosophical structure for psychiatry. Am J Psychiatry 2005;162:433-40.
13. Sanislow CA, Pine DS, Quinn KJ et al. Developing constructs for psychopathology research: Research Domain Criteria. J Abnorm Psychol 2010;119:631-9.
14. Bolton D. Classification and causal mechanisms: a deflationary approach to the classification problem. In: Kendler KS, Parnas J (eds). Philosophical issues in psychiatry II: nosology. Oxford: Oxford University Press, 2012:6-11.
15. Ghaemi SN. Nosologomania: DSM & Karl Jaspers' critique of Kraepelin. Philosophy, Ethics and Humanities in Medicine 2009;4:10.
16. Bolton D. What is psychiatric disease? A commentary on Dr Ghaemi's paper. In: Kendler KS, Parnas J (eds). Philosophical issues in psychiatry II: nosology. Oxford: Oxford University Press, 2012:54-7.
17. Strauss JS. Subjective experiences of schizophrenia: toward a new dynamic psychiatry – II. Schizophr Bull 1989;15:179-87.
18. Maj M. The relevance of Jaspers' General Psychopathology to current psychiatric debate. In: Stanghellini G, Fuchs T (eds). One

century of Karl Jaspers' General Psychopathology. Oxford: Oxford University Press (in press).

19. Mishara A, Fusar-Poli P. The phenomenology and neurobiology of delusion-formation during psychosis onset: Jaspers, Truman signs and aberrant salience. Schizophr Bull (in press).

20. Parnas J. Rediscovering psychopathology: the epistemology and phenomenology of the psychiatric object. Schizophr Bull (in press).

21. Fulford B, Stanghellini G, Bolton D. Person-centered psychopathology of schizophrenia. Building on Karl Jaspers' understanding of the patient's attitude towards his illness. Schizophr Bull (in press).

22. Maj M. Karl Jaspers and the genesis of delusions in schizophrenia. Schizophr Bull (in press).

Secondary psychoses: an update

Matcheri S. Keshavan[1], Yoshio Kaneko[2]

[1]Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA; [2]Longwood Psychiatry Residency Training Program and Harvard Medical School, Boston, MA, USA

Psychotic disorders due to a known medical illness or substance use are collectively termed secondary psychoses. In this paper, we first review the historic evolution of the concept of secondary versus primary psychosis and how this distinction supplanted the earlier misleading classification of psychoses into organic and functional. We then outline the clinical features and approach to the diagnosis of secondary psychotic disorders. Features such as atypical presentation, temporal relation to detectable medical cause, evidence of direct physiological causal relationship to the etiological agent, and the absence of evidence of a primary psychotic illness that may better explain the presentation suggest consideration of a secondary psychosis. Finally, we discuss how careful studies of secondary psychotic disorders can help elucidate the pathophysiology of primary, or idiopathic, psychotic disorders such as schizophrenia. We illustrate this issue through a discussion of three secondary psychotic disorders – psychoses associated with temporal lobe epilepsy, velocardiofacial syndrome, and N-methyl D-aspartate (NMDA) receptor encephalitis – that can, respectively, provide neuroanatomical, genetic, and neurochemical models of schizophrenia pathogenesis.

Classification of psychotic disorders has continued to generate debate. The idea that psychoses may be categorized into those with detectable anatomic pathology (organic) and those without such pathology (functional) has been prevalent for more than a century (1). Unfortunately, this distinction sometimes misled the field, particularly during the early decades of the 20th century, when functional was equated with psychogenic, and the etiology of schizophrenia was ascribed to psychologic factors such as parental upbringing.

Recent decades have seen a growing realization that it is more useful to categorize psychiatric disorders as secondary, when the symptoms are due to a known medical illness or substance use, and as primary (or idiopathic), if the symptoms cannot be explained by another cause. In this context, the DSM-IV dropped the terms organic and functional used in the earlier editions (2). The emphasis was shifted from the presence or absence of discernible brain pathology (which is often difficult to identify, even in many neurological disorders) to etiology (known, presumed, or unknown). The DSM-IV also made the distinction between psychotic disorders secondary to medical illness versus those secondary to substance use.

In this paper, the primary versus secondary approach has been taken to understand psychotic disorders, since the term primary has the advantage of not ruling out a neurobiological basis. While a variety of secondary psychotic disorders are described throughout this text, it is important to have an appreciation of how such understanding can elucidate the neurobiological substrates that might underlie primary (or idiopathic) psychotic illness. We seek to briefly provide an overview of what is known in this regard and offer an approach to the differential diagnosis between primary and secondary psychotic disorders.

DISORDERS PRESENTING WITH SECONDARY PSYCHOTIC SYMPTOMS

Virtually any substance, prescribed drug, or medical condition affecting nervous system function can present with psychiatric symptoms, including psychosis (Table 1). The mnemonic TACTICS MDS USE may offer the clinician an easy way to remember the main groups of disorders to consider in the differential diagnosis.

Traumatic brain injury

Traumatic brain injury (TBI) has been proposed as a risk factor for schizophrenia-like psychosis, though there have been few systematic studies of the relationship between these two conditions.

Fujii and Ahmed (3) conducted a review of case reports in which they retroactively applied DSM-IV criteria to a total of 69 cases and concluded that TBI can be either a primary cause of psychosis or contribute to the development of psychosis through inducing seizures, though this study was limited by the heterogeneity of the case reports. A cohort study of 3552 Finnish World War II veterans (4) reported a rate of psychosis of 8.9% following TBI, but the proportion of open injuries, potential comorbidities, and lack of standard diagnostic tools call into question the generalizability of these findings.

Evidence from additional studies, including epidemiologic and case control studies, indicates that TBI may marginally increase the risk of psychosis, though the increased risk may be significantly higher for individuals with a genetic predisposition to psychosis (5-7). The risk of psychosis appears to be elevated when TBI is severe, diffuse, involves the frontal and temporal lobes, and is associated with abnormal findings on electroencephalography and

Table 1 Possible causes of secondary psychoses

	Examples	Investigations
Trauma	Traumatic head injury	CT, MRI
Autoimmune disorders	Systemic lupus erythematosus, NMDA receptor encephalitis	Autoantibody titers
Cytogenetic/congenital disorders	Velocardiofacial syndrome, agenesis of corpus callosum	Karyotyping, MRI
Toxic/substance-induced disorders	PCP, MDMA, LSD, cannabis, alcohol Lead, mercury or arsenic poisoning	Careful medication history; urine screen for drugs, heavy metal screen; trial off the offending agent
Iatrogenic disorders	Antimalarials, steroids, isoniazid	Careful medication history; trial off the offending agent
Cerebrovascular disorders	Stroke, subdural hematomas	CT, MRI
Space-occupying disorders	Cerebral tumors	CT, MRI
Metabolic disorders	Phaeochromocytoma, metachromatic leukodystrophy, Wilson's disease	Urinary catecholamines; arylsulphatase-A levels, copper and ceruloplasmin levels
Dietary disorders	Pellagra, B12 deficiency; vitamin D deficiency	B12, Folate, D3 levels
Sepsis/infectious disorders	Neurosyphilis, toxoplasmosis, HIV disease	RPR to rule out syphilis; HIV antibody titers; glucose, protein in CSF
Unknown cause/degenerative/ demyelinating disorders	Lewy body dementia, Parkinson's disease, Huntington's disease, multiple sclerosis, Fredreich's ataxia	MRI, CT, EEG, evoked potentials
Seizure disorders	Partial complex seizures, temporal lobe epilepsy	EEG, including sleep deprivation; telemetric EEG as indicated
Endocrine disorders	Hyperthyroidism, hypothyroidism, hyperparathyroidism	Serum calcium, thyroid/parathyroid hormone levels

CT – computed tomography; MRI – magnetic resonance imaging; NMDA – *N*-methyl D-aspartate; PCP – phencyclidine; MDMA – 3,4-methylenedioxy-*N*-methyl-amphetamine; LSD – lysergic acid diethylamide; RPR – rapid plasma reagin; HIV – human immunodeficiency virus; CSF – cerebrospinal fluid; EEG – electroencephalography

neuroimaging investigations (8-10). When psychosis does occur in the setting of TBI, it is characterized by prominence of persecutory delusions and auditory hallucinations, with a relative paucity of negative symptoms (3,8). Given this evidence, further research examining the interaction between TBI and genetic risk factors for schizophrenia is warranted.

Autoimmune disorders

Psychotic symptoms are a rare, but known, manifestation of systemic lupus erythematosus (SLE), with a reported prevalence ranging from 1 to 11% (11-13). Retrospective analyses reveal that, when psychosis does occur in SLE, it tends to appear early in the disease course. Approximately 30–60% of the patients found to have psychosis were psychotic when diagnosed with SLE and up to 80% developed these symptoms within 1 year of SLE diagnosis (13-15). While psychosis can be the presenting symptom in SLE, it occurs more frequently in the context of other lupus symptoms, most commonly cutaneous involvement and arthritis (13).

Symptomatically, lupus psychosis is frequently characterized by paranoia, auditory and visual hallucinations, and delusions of grandeur (14). Immunosuppressive treatment, including steroids and antimalarials, typically results in resolution of the psychotic symptoms, although these medications themselves can induce psychotic symptoms and contribute to diagnostic difficulty (14).

Autoimmune disease as a cause of psychotic symptoms is not limited to SLE. Psychosis may be the presenting symptom in multiple sclerosis (16,17) and has been documented to occur in Hashimoto's disease (18,19).

Congenital/cytogenetic disorders

Velocardiofacial syndrome (VCFS), discussed in further detail below, is caused by a heterozygous chromosome 22q11.2 deletion and is associated with the strongest link yet identified between a genetic condition and psychosis, with 25–30% of VCFS patients developing symptoms analogous to schizophrenia (20-23).

VCFS is of particular interest because the neurocognitive findings, brain structural abnormalities, psychotic symptoms, and patterns of treatment response seen in this disorder are all highly similar to those seen in schizophrenia (24).

Other genetic disorders in which psychosis can occur include Prader-Willi syndrome (in which the rate of schizophrenia-like psychosis is approximately 16%) (25,26), Huntington's disease (rate of 5–16%) (27,28), and Fahr's disease/syndrome (29,30).

Toxic/drug-induced disorders

Individuals using psychoactive substances can experience psychotic symptoms in various contexts, including acute intoxication, withdrawal, delirium induced by either intoxication or withdrawal, substance-induced mood disorder with psychotic features, and substance-induced psychosis (SIP). SIPs are best conceptualized as those conditions where the psychosis begins in the context of substance use but persists for days to weeks in the absence of continued substance use.

The substances with the clearest psychotogenic properties include stimulants (amphetamine, cocaine) (31-33), cannabis (34,35), and the psychotomimetics (phencyclidine, ketamine) (36,37). Opiates and nicotine have not been clearly shown to produce psychosis, while alcohol and benzodiazepines may induce psychosis just in acute withdrawal states (33,38,39). Lysergic acid diethylamide and 3,4-methylenedioxy-N-methylamphetamine (MDMA) can produce hallucinations in acute intoxication, but there is no clear evidence that they induce an ongoing psychotic disorder (40,41).

The interaction between susceptibility to psychotic disorders and to substance abuse disorders is complex, with evidence suggesting both that genetic vulnerability to psychosis in combination with drug abuse can bring about psychosis and that individuals with such vulnerability may be more likely to abuse substances.

Toxic psychosis can also occur in the context of exposure to heavy metals, including lead (42), mercury, and arsenic (43).

Iatrogenic psychoses

Toxic psychosis can be caused by numerous centrally active medications and is often characterized by acutely impaired cognitive function in addition to psychotic symptoms.

The reported rates of psychotic symptoms in patients receiving glucocorticoids have varied widely, although there is evidence that mood disorders secondary to glucocorticoids are associated with psychotic symptoms more frequently than are primary mood disorders. While onset of psychotic symptoms usually occurs within several days of initiating glucocorticoid treatment, the symptoms can occur anytime from hours to weeks after the first dose is administered (44).

Elderly patients are at particular risk for developing toxic psychosis in reaction to medications with anticholinergic properties (45,46) and to benzodiazepines (47,48). The development of psychotic symptoms following initiation of isoniazid treatment has been documented in a growing number of case reports (49), and ascribed to alteration in catecholamine and serotonin levels via monoamine oxidase inhibition. Antimalarial drugs including chloroquine and mefloquine have been linked to psychotic symptoms, with evidence that the risk is higher in patients with a history of

psychiatric illness (50,51). Additional classes of medications that have been linked to toxic psychosis include antidepressants (52), anticonvulsants (53), antiemetics (54), antiparkinsonian agents (55), antipsychotics (47), opioids (47), histamine antagonists (56), and antibiotics (57), particularly when toxic blood levels occur.

Cerebrovascular disorders

Post-stroke psychosis prevalence rates are estimated at 3–4% (58,59). While psychotic symptoms have occurred in association with strokes in numerous brain regions, they are most common in temporo-parietal-occipital lesions (59). The nature of the psychotic symptoms differs from schizophrenia, with post-stroke psychosis more likely to include visual, tactile, and olfactory hallucinations (60-62). Hallucinatory behavior in post-stroke patients has been further classified into frank hallucinations and hallucinosis, the latter distinguished by ego-dystonia and retained insight into the fact that the perceptions are not real (63).

There has been little systematic study of the link between vascular dementia and the development of psychotic symptoms, though the fact that psychosis occurs at similar rates in both vascular and Alzheimer's dementia argues against a psychotogenic mechanism specific to the cerebrovascular disturbance (64).

Studies that have searched for evidence of cerebrovascular injury in patients who develop late-onset psychotic symptoms have produced conflicting results (65). Based on the limited data currently available, it is difficult to conclude to what extent cerebrovascular disease predisposes a patient to the development of psychotic symptoms, and further research is needed to confirm or disprove this link.

Space-occupying intracranial disorders

Brain tumors are an uncommon, but important, cause of secondary psychosis, and there is evidence that the prevalence of intracranial tumors is increased in patients with psychiatric illness (66-68). Symptomatically, psychosis secondary to intracranial tumors can be indistinguishable from schizophrenia, although it is more commonly associated with visual hallucinations, simple unelaborated delusions, and absence of formal thought disorder (69-72).

Tumors located in the temporal lobes or limbic structures are the most likely to produce psychosis, with one study demonstrating that 20% of tumors in the temporal lobe resulted in psychotic symptoms (69,73). There has been no demonstrated link between the histological type of tumor and the frequency of psychosis, though low-grade, slow-growing tumors seem most likely to produce psychotic symptoms in the absence of neurological signs (74).

Neuroimaging with either computed tomography (CT) or magnetic resonance imaging (MRI) is recommended in older patients presenting with new-onset psychosis

and in patients who present with focal neurological findings on exam (75).

Metabolic disorders

Disordered neural connectivity, the putative mechanism for the symptoms of schizophrenia, can also occur in metabolic disorders that result in disrupted neuronal function or neuronal death. For example, although lysosomal storage disorders typically produce early severe neurological deficits and often death, there are adolescent or adult forms of these disorders that are associated with secondary psychosis. These disorders, such as Niemann-Pick disease type C (NPC), Tay Sachs disease, and alpha mannosidosis, likely produce psychotic symptoms through the interaction of the neuropathological processes with neurodevelopmental changes including synaptic pruning, myelination, and changes in connectivity (76-78). A similarly disordered interplay of functional connectivity and neurodevelopment is seen in the leukodystrophies, with the late-onset form of metachromatic leukodystrophy (MLD) as the prototypical example of the link between this group of disorders and secondary psychosis (76,79).

Psychotic symptoms have been reported to occur in up to 50% of patients with late-onset NPC and MLD (76). In addition, there have been case reports of patients with mitochondrial disorders presenting with psychotic symptoms, and it is thought that the leukodystrophy that can occur in mitochondrial disorders is implicated in the psychosis (80-82).

Wilson's disease, involving abnormal deposition of copper in the liver and brain, has reported prevalence rates of psychosis ranging from 2 to 11% (83,84). The psychosis in Wilson's disease is characterized not only by typical symptoms of hallucinations, delusions, and thought disorders, but also by a myriad of additional symptoms, including euphoria, sexual preoccupation, hebephrenia, and catatonia (85).

Dietary disorders

The link between nutritional deficiencies and psychiatric symptoms has been investigated and debated for decades. There is evidence that psychiatric symptoms of cobalamin deficiency may occur without evidence of hematological or neurological abnormalities (86,87). Case reports of psychosis secondary to cobalamin deficiency describe symptoms of persecutory delusions, auditory and visual hallucinations, disorganized thought processes, and psychomotor agitation that were unresponsive to treatment with antipsychotic medications but resolved completely after parenteral cobalamin therapy (88-90). Additional evidence in support of this connection includes a study in which depressed patients with psychotic depression had significantly lower cobalamin levels than those with nonpsychotic depression (91).

Although the link between folate deficiency and psychosis is tenuous, there is some evidence that folate supplementation in patients with schizophrenia enhances recovery (92). Psychotic symptoms including auditory hallucinations, persecutory delusions, and delusional parasitosis can very rarely develop in the setting of pellagra (niacin deficiency), though this occurs almost exclusively in chronic alcohol abuse (93,94).

Sepsis/infectious diseases

Historically, neurosyphilis has been closely tied to psychiatric hospitalization. In 1900, approximately 5% of institutionalized mental patients were diagnosed with general paresis of the insane (95). Syphilis infection rates have increased since the appearance of human immunodeficiency virus (HIV), and patients presenting with exclusively psychiatric symptoms in the setting of neurosyphilis have been noted (96). Such patients can present with both affective and psychotic symptoms that are indistinguishable from primary psychiatric disorders (97). Treatment of neurosyphilis with antibiotics, and adjunctive antipsychotic medications as necessary, typically halts, but does not reverse, mental status deterioration due to neuronal loss (98), although there are case reports of significant clinical improvement (99,100).

Estimates of new-onset psychosis associated with HIV infection have ranged between 0.23% and 15%, with symptoms generally presenting either in late-stage HIV or when patients have transitioned to acquired immunodeficiency syndrome (AIDS) (101,102). Symptomatically, HIV-related psychosis is characterized by persecutory, grandiose, and somatic delusions, with hallucinations as a second prominent symptom cluster (103). The effect of treatment with highly active antiretroviral therapy on psychotic symptoms remains unclear, and is further complicated by the fact that side effects of antiretrovirals include hallucinations (102). Although antipsychotics are generally effective in HIV psychosis, patients with HIV are at increased risk of developing extrapyramidal symptoms and tardive dyskinesia, particularly with the use of typical antipsychotics (101,104).

Additional infectious causes of secondary psychosis have been postulated, including infection with *Toxoplasma gondii*. This link is based on the fact that studies have demonstrated an increased prevalence of antibodies to *Toxoplasma gondii* in patients with schizophrenia (105,106). Moreover, toxoplasmosis has been linked to psychotic symptoms including delusions and auditory hallucinations, even in the absence of concurrent AIDS (107,108).

Finally, there is increasing evidence to support prenatal infections as potential risk factors for schizophrenia. Although still inconclusive, prenatal infections with influenza, toxoplasmosis, rubella, herpes simplex virus, and

syphilis have been associated with the development of secondary psychosis (109-113).

Unknown cause/degenerative/demyelinating disorders

Although the data on psychosis in multiple sclerosis (MS) have been inconsistent, recent evidence suggest that MS does increase the likelihood of developing psychotic symptoms (17). A large, population-based study from Canada reported that 2–4% of patients with MS became psychotic (114). Delusions are the primary psychotic symptom observed in MS, while hallucinations and negative symptoms are rarely seen (115). Reports of a temporal correlation between MS symptoms and psychosis, however, have been conflicting (74,115). Psychotic symptoms may be related to increased lesion burden in the periventricular white matter and temporal horns, though a precise mechanism remains to be elucidated (116). Chronic psychosis due to MS seems to be rare.

Estimates of the prevalence and incidence of psychotic symptoms in Alzheimer's disease (AD) have varied, with one review of 55 studies reporting an overall prevalence of 41%, consisting of 36% of patients with delusions and 18% with hallucinations (117). The first three years of AD are characterized by an increasing incidence of psychotic symptoms, after which there seems to be a plateau (117,118). Psychotic symptomatology in AD most often consists of delusions, typically of theft or suspicion, visual hallucinations more frequently than auditory hallucinations, and misidentifications (119). These symptoms are typically coincident with other psychiatric symptoms including aggression, agitation, apathy, and depression (120).

While psychotic symptoms appear to be less common in the fronto-temporal dementias, they have been noted in approximately 20% of patients, with higher rates in particular subtypes (121,122).

Seizure disorders

Schizophrenia-like psychosis has been associated with epilepsy for more than a century, and there is strong evidence that chronic psychosis occurs more frequently in patients with epilepsy than in the general population. Psychotic syndromes in epilepsy have traditionally been categorized based on their temporal association with clinical seizures. Ictal psychoses represent psychotic symptoms occurring in the context of an active nonconvulsive seizure, tend to last for minutes to hours, and consist of prominent hallucinations and paranoid delusions (65). Post-ictal psychoses are brief psychotic episodes that typically occur hours to days following a seizure cluster, consist of delusions, hallucinations, and affective symptoms, and generally resolve within several days (123,124). The development of a chronic psychosis

associated with epilepsy is also well documented, with a recent study reporting a relative risk of 2.48 (125,126).

Phenomenologically, psychosis associated with epilepsy is difficult to distinguish from schizophrenia, as suggestions that it is characterized by a more benign course and relative lack of negative symptoms have not been verified in the literature (65,125). Risk factors for developing secondary psychosis include a more severe form of epilepsy with multiple seizure types (127), history of status epilepticus (125), and resistance to medication treatment (65). The preferential, though not exclusive, association of temporal lobe epilepsy and the development of psychotic symptoms is discussed in further detail below.

Endocrine disorders

While psychotic symptoms secondary to abnormal thyroid functioning are rare, there are case reports of psychosis developing secondary to hyperthyroidism, hypothyroidism, and even rapid alteration of thyroid state. Psychosis has been described as the presenting symptom of thyrotoxicosis in Graves disease (128), thyroid storm (129), toxic nodular goiter (130), subacute thyroiditis (131), and painless thyroiditis (132). Similarly, hypothyroidism has been associated with psychosis, with one study reporting that as many as 5–15% of myxedematous patients have some form of psychotic symptoms (133-135).

Thyroid-associated psychosis does not contain a characteristic psychotic symptom cluster, as patients have presented with auditory and visual hallucinations, delusions, and paranoia. The majority of patients present with affective disturbance (130,136). Treatment of the underlying thyroid abnormality tends to result in resolution of the psychosis, such that antipsychotic medications are necessary only in the acute setting (128,132,136). Interestingly, however, rapid correction of abnormal serum thyroid hormone levels can both induce and exacerbate thyroid-associated psychosis (137,138).

The association of hyperparathyroidism and hypercalcemia with significant psychiatric symptoms is well known, although the specific prevalence of psychosis in patients with hyperparathyroidism remains unclear. Case reports describe patients presenting with auditory and visual hallucinations, persecutory delusions, and disorganized thought processes in the setting of hyperparathyroidism-induced hypercalcemia (139,140). The available evidence indicates that correction of the hypercalcemia, generally via parathyroidectomy, results in resolution of the psychotic symptoms with no subsequent recurrence (139-141).

Although less common and investigated, hypoparathyroidism can also present with psychosis. A review of 268 cases of hypoparathyroidism published in 1962 reported

that 11% of the patients had psychotic symptoms, often in the setting of surgically induced hypoparathyroidism (142). Subsequent case reports have supported the occurrence of psychotic symptoms in hypoparathyroidism and emphasized the fact that symptomatic improvement requires normalization of magnesium and calcium levels, in addition to treatment with antipsychotics (141,143).

INVESTIGATING SECONDARY PSYCHOSIS VERSUS SCHIZOPHRENIA

Establishing a cause-effect relationship between substance use/medical illness and psychosis is not easy. Suspecting an underlying medical illness is a logical initial step when encountering psychosis in general medical settings. Comorbid medical illnesses are also quite common in patients presenting with psychotic symptoms, especially among the elderly. Suspecting and identifying an underlying medical illness in younger patients with psychosis in mental health settings is more challenging.

In making the distinction between primary and secondary psychosis, it is important to first establish the presence of the general medical condition. The next step – establishing the cause-effect relationship between the medical condition and psychosis – is often difficult, but can be helped by considering the following three key principles: atypicality, temporality, and explicability.

Is the presentation of the psychosis atypical?

An underlying medical cause for psychosis should be especially suspected if the presentation is atypical. An example is later age of onset: new onset of psychosis in a 70-year-old man should raise suspicion of an underlying medical illness. It is to be kept in mind that no single clinical feature or combination of symptoms reliably distinguishes between primary and secondary psychotic disorders. If, however, a particular feature predominates, that should raise a red flag. Thus, a strong component of severe disorientation and/or confusion must raise suspicion. Catatonic symptoms, altered states of consciousness (i.e., confusional or "dream like" states), and visual hallucinations are more frequent in secondary psychoses. Certain delusions, such as those involving beliefs of mistaken identity of others (i.e., Capgras delusions), are thought to be more common in secondary psychoses than in schizophrenia. The presence of multimodality hallucinations (e.g., visual and tactile) also increases the likelihood of a secondary psychotic disorder.

Accompanying symptoms that are disproportionate to what may be expected from a psychotic disorder should also make one consider a potential underlying medical disease. For example, a large weight loss that may not be easily explained by the mild depression in a psychotic patient may trigger suspicion of a medical illness.

Specific types of psychotic symptoms may also often point to regional alterations in brain function and raise suspicion of neurological disease. Thus, denial of blindness that may appear delusional should trigger suspicion of Anton's syndrome (cortical blindness, due to visual cortex lesions) and denial of paralysis should lead to a consideration of anosognosia (due to lesions in the nondominant parietal cortex). Likewise, both an isolated delusion of misidentification (Capgras syndrome) and olfactory hallucinations are suggestive of temporal lobe disease.

Is the medical condition or substance use temporally related to the psychosis?

Secondary psychosis is likely when the psychosis begins following the onset of the medical condition, varies in severity with the severity of the medical condition, and resolves when the medical condition improves. An example is the appearance of delusions when a patient with hypothyroidism stops taking thyroid treatment and the resolution of the symptoms after resuming medication. This rule, however, has many exceptions; for example, psychosis in temporal lobe epilepsy appears several years after the onset of the seizures. Conversely, a medical illness may simply worsen or trigger a relapse of schizophrenia without being the direct cause of the illness.

Is the psychosis not better explained by a primary psychotic disorder or another mental disorder?

Comorbid medical illness is very common in patients with chronic psychotic disorders such as schizophrenia. In some cases, even if a concomitant medical illness may raise suspicion of a secondary psychosis, the presence of a strong family history of schizophrenia and a premorbid schizoid personality point to a diagnosis of schizophrenia. Similarly, in patients with a known history of an affective illness, the appearance of psychotic symptoms is likely related to the affective illness rather than a medical condition.

Is psychosis a direct physiological consequence of a medical illness or substance use?

The answer to this question depends first on establishing the presence of an underlying medical condition or substance use that might be the etiological agent. This will require a careful history, physical, and neurological examination, along with appropriate laboratory investigations. Second, even if such a causal agent exists, it is often difficult to ascertain whether the symptoms of psychotic illness are a direct physiological consequence of that factor. Sometimes such a direct link may be obvious. For example, the presence of autonomic hyperactivity (e.g., dilated pupils, tachycardia) along with paranoid anxiety might suggest a sympathomimetic agent such as amphetamine, phencyclidine, or an adrenal tumor.

In every patient with a first episode of psychosis, it is critical to obtain a detailed history and complete

physical, including neurological examination and laboratory evaluation to rule out common medical disorders (Table 2). Additional investigations such as brain imaging, cerebrospinal fluid and electrophysiologic studies may be needed, especially for those with atypical presentations and those in whom there is reason to rule in a primary disorder.

The question of whether to conduct a brain scan in a patient with suspected schizophrenia on a routine basis is debatable. When brain scans, commonly structural (such as MRI or CT), have been used clinically as part of the work-up of a psychotic patient, the purpose has been to rule out a space occupying lesion or developmental malformation as potentially causative of the psychosis. Although incidental findings have been reported in MRI studies of patients who present with psychosis (144), and indeed occur even in healthy individuals (145), such findings are rare. Thus, in the absence of quantitative analysis, routine brain imaging cannot aid in the differential diagnosis of psychosis without considering the clinical presentation (146).

CAN SECONDARY PSYCHOSES ILLUMINATE PATHOPHYSIOLOGY OF SCHIZOPHRENIA?

Schizophrenia, a common and highly disabling disease with unclear causation, is a broad heterogeneous entity that may comprise several idiopathic psychotic disorders (147). To unravel its complexity, it is important to identify homogeneous subgroups within this illness to better characterize its pathophysiology. One approach to address this is to examine syndromes of known etiology that present with clinical manifestations similar to schizophrenia, that is, phenocopies of this illness.

To identify suitable phenocopies of schizophrenia to study, one needs to first define what we know about the pathophysiology of schizophrenia. At an anatomical level, schizophrenia is characterized by brain structural abnormalities in frontal, temporal, parietal, basal ganglia, thalamic, and limbic regions (148). Functional imaging studies suggest impaired function of prefrontal regions (hypofrontality) and impaired interhemispheric and intrahemispheric connectivity. At a neurochemical level, increasing evidence points to dopaminergic, glutamatergic, and GABAergic dysfunction in the pathophysiology of psychosis, with the *N*-methyl D-aspartate (NMDA) hypofunction hypothesis as a leading theory to describe the pathogenesis of schizophrenia. Several lines of evidence also point to alterations in immune and oxidative stress mechanisms in schizophrenia (149). From an etiological point of view, schizophrenia is thought to be highly heritable (heritability > 70%), but several environmental factors including viruses, drugs of abuse, head trauma, and obstetric complications have been implicated (150). It is thus likely that the symptoms of the illness arise through the combination of genetic vulnerability and environmental stressors.

Table 2 Approach to investigating patients to rule out secondary psychoses

First line assessments (to be routinely considered in all first psychotic episode patients)

Detailed medical and neurological/psychiatric history

Physical/neurological examination

Neuropsychological tests

Laboratory tests: complete and differential blood count, erythrocyte sedimentation rate, glucose, electrolytes, thyroid function tests, liver function tests, urinary drug screen

Second line assessments (to be considered when above assessments raise specific diagnostic possibilities)

Laboratory tests: rapid plasma reagin to rule out syphilis; HIV testing; serum heavy metals; copper and ceruloplasmin levels; serum calcium levels; autoantibody titres (e.g., antinuclear antibodies for lupus); B12, folate levels; arylsulfatase-A levels; urine: culture and toxicology, drug screen

Neuroimaging: computed tomography, magnetic resonance imaging, positron emission tomography, single proton emission tomography

Electroencephalography, polysomnography, evoked potentials

Cerebrospinal fluid investigations: glucose, protein, cultures, cryptococcal antigen

Karyotyping

Given the substantive heterogeneity of schizophrenia, it would be difficult to envision any individual secondary psychosis being an adequate model to explain all aspects of that disorder. Not surprisingly, several psychotic disorders stemming from the etiological factors listed in Table 2 demonstrate only partial similarity to what we clinically identify as schizophrenia. Some disorders, however, are more likely to show clinical, pathophysiological, or etiological parallels to schizophrenia; we discuss them briefly as they may provide clues to better understand that disease.

Temporal lobe epilepsy: an anatomical model of schizophrenia

Psychosis appears to occur in 7–11% of patients with epilepsy, a rate much higher than in the general population (151). Psychosis, when it occurs in temporal lobe epilepsy, has been thought to closely resemble schizophrenia, as described by Slater in a classic early paper (125). The emergence of psychoses in temporal lobe epilepsy has been associated with onset of epilepsy under the age of 20 years, a history of epilepsy lasting for more than 10 years, a history of complex partial seizures, and lesions on the left side (152).

The occurrence of psychoses in temporal lobe epilepsy is consistent with the medial temporal structural alterations reported in schizophrenia (148). In particular, positive symptoms such as auditory hallucinations and formal thought disorder have been linked to structural alterations in the auditory association areas in the superior temporal gyri (148). The striking prominence of positive symptoms

in temporal lobe epilepsy has led to the question whether there might be a resemblance between temporal lobe epilepsy and the neurochemical models of schizophrenia. Ando et al (153) examined alterations of central dopaminergic systems in the kainate model of temporal lobe epilepsy using methamphetamine-induced locomotor activity as an index of dopaminergic sensitivity in adult rats. They found evidence of dopaminergic hypersensitivity, which can clearly explain the mechanisms underlying epileptic psychosis and can also indicate similar alterations in idiopathic psychoses.

Velocardiofacial syndrome (VCFS): a genetic model of schizophrenia

While the etiology of schizophrenia remains unclear, it is widely agreed that genetic factors have a substantial contribution, with heritability estimates of greater than 70%. Currently, schizophrenia is thought to be polygenic and multifactorial, with a small proportion of cases due to copy number variations, such as microdeletions or microduplications of chromosomal regions. A large portion of the genetic etiology of schizophrenia remains uncharted, necessitating the study of discrete genetic syndromes that present with schizophrenia-like features.

VCFS is characterized by a large deletion on one copy of chromosome 22 (comprising up to 30 genes), which can be detected with genetic testing. It is the most common chromosomal microdeletion in humans, and is characterized by congenital abnormalities of the heart, facial dysmorphism, and cognitive deficits in childhood. A substantial proportion of affected individuals develop major psychiatric illnesses in adolescence or early adulthood, with schizophrenia spectrum disorders occurring in 25–30% of affected individuals (20-23).

VCFS may offer a model of the relationship between genetic liability and risk of symptomatic manifestations of schizophrenia. The cognitive deficits in schizophrenia have been linked to a polymorphism of the gene coding for catechol *O*-methyltransferase (COMT), an enzyme involved in the degradation of dopamine. Individuals who have the Val/Val polymorphism have reduced prefrontal dopamine because of increased activity of COMT, and as a result, may have impaired cognitive function (154). VCFS is associated with similar cognitive impairments to those seen in schizophrenia, including deficits in executive control, memory, and attention. Individuals with VCFS who have the hemizygous COMT Val(158)Met genotype have been shown to have improved cognition associated with decreased enzymatic degradation of dopamine (154). Brain morphologic brain abnormalities, including prefrontal and cingulate gray matter loss, similar to those seen in schizophrenia, are also observed in VCFS, and such abnormalities appear to be correlated with cognitive impairments (155). Longitudinal studies suggest that brain structural alterations in VCFS

appear to predict the emergence of psychotic symptoms (156), though further research is needed to identify the neurobiological and genotypic signatures of VCFS patients that go on to develop secondary psychoses. Studies, such as these, of the neurobiological basis of psychosis in VCFS will likely elucidate the pathophysiology of at least a subgroup of patients with schizophrenia.

NMDA encephalitis: a pathophysiological model of schizophrenia

While there has been considerable progress in our understanding of the pathophysiological substrate of schizophrenia, the core dysfunctions remain a matter of debate. A leading theory is that glutamatergic and GABAergic dysfunction may underlie the developmental pathophysiology of psychosis, perhaps through glutamatergic NMDA receptor hypofunction (157,158). This model is supported by clinical observations of a psychosis closely resembling schizophrenia that is caused by the NMDA receptor antagonist phencyclidine as well as by neuropathological observations of altered NMDA receptor binding and expression in postmortem brains of patients with schizophrenia (159). The NMDA receptor, therefore, provides a natural biological model for further study and may provide insight into the complex pathophysiological heterogeneity of schizophrenia.

Glutamatergic dysfunction may result from a failure of GABA-mediated regulation of predominantly glutamatergic pyramidal neurons. This theory is supported by observations of reduced GABA synthesis, as reflected by decreased neuronal activity of the 67-kDa isoform of glutamic acid decarboxylase (GAD67), in patients with schizophrenia (160). Several lines of evidence also point to alterations in immune mechanisms in schizophrenia (161). There is a need to draw connections between the NMDA/GABA alteration model and theories of immune system modulation in schizophrenia; studies of the appropriate forms of secondary psychoses might be one way to further elucidate this model.

An intriguing form of secondary psychosis that leads to a hypofunctioning NMDA receptor state may provide insight into the pathophysiology of schizophrenia. Since 2007, many case reports described a form of encephalitis in which patients presenting with neurological and psychiatric symptoms were found to have positive autoantibodies to the NR1/NR2 heteromers of the NMDA receptor. Dalmau and colleagues (162,163) examined 100 patients presenting with NMDA receptor encephalitis and showed that 77% presented with a variety of psychiatric symptoms that included anxiety, insomnia, fear, grandiosity, delusions, hyper-religiosity, mania, and paranoia.

A model that integrates NMDA receptor encephalitis with GABA/glutamate dysfunction includes the idea that an antibody-mediated decrease in NMDA receptors may inactivate GABAergic neurons, which normally serve to inhibit extracellular glutamate. Without the regulating presence of GABA,

the resultant excessive glutamate may induce and exacerbate psychosis. A similar mechanism may be present in schizophrenia, though definitive evidence of alterations of brain glutamatergic neurotransmission in this disease has yet to be produced. The NMDA receptor provides a biological mechanism for the development of psychosis that warrants further study, with the further possibility that variations in the nature of NMDA receptor dysfunction could inform the complex pathophysiological heterogeneity of schizophrenia.

CONCLUSIONS

The historical distinction between organic (or structural) and functional (purely psychological) disease has not served the field and stunted systematic investigation in the disorders that were earlier deemed purely psychological. Classification of psychiatric disorders into those with or without identifiable etiology has been more clinically meaningful.

Identification of an underlying medical, toxic, or iatrogenic cause in a patient presenting with psychosis can be diagnostically challenging. Careful history taking, physical examination, and judicious use of modern medical testing, combined with an informed mind, can help the clinician to arrive at a timely diagnosis and optimum intervention, which can be quite gratifying.

In appreciating the causes of secondary psychoses, the clinician-scientist also gains potential insight into the puzzling pathophysiology and etiology of primary psychotic disorders such as schizophrenia.

References

1. Mendel LB. The American Physiological Society. Science 1907; 26:765.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed., text revision. Washington: American Psychiatric Association, 2000.
3. Fujii D, Ahmed I. Characteristics of psychotic disorder due to traumatic brain injury: an analysis of case studies in the literature. J Neuropsychiatry Clin Neurosci 2002;14:130-40.
4. Achté KA, Hillbom E, Aalberg V. Psychoses following war brain injuries. Acta Psychiatr Scand 1969;45:1-18.
5. Silver JM, Kramer R, Greenwald S et al. The association between head injuries and psychiatric disorders: findings from the New Haven NIMH Epidemiologic Catchment Area Study. Brain Inj 2001;15:935-45.
6. Harrison G, Whitley E, Rasmussen F et al. Risk of schizophrenia and other non-affective psychosis among individuals exposed to head injury: case control study. Schizophr Res 2006;88:119-26.
7. David AS, Prince M. Psychosis following head injury: a critical review. J Neurol Neurosurg Psychiatry 2005;76 (Suppl. 1):i53-60.
8. Sachdev P, Smith JS, Cathcart S. Schizophrenia-like psychosis following traumatic brain injury: a chart-based descriptive and case-control study. Psychol Med 2001;31:231-9.
9. Cutting J. The phenomenology of acute organic psychosis. Comparison with acute schizophrenia. Br J Psychiatry 1987;151:324-32.
10. Miller BL, Benson DF, Cummings JL et al. Late-life paraphrenia: an organic delusional syndrome. J Clin Psychiatry 1986;47:204-7.
11. Sibley JT, Olszynski WP, Decoteau WE et al. The incidence and prognosis of central nervous system disease in systemic lupus erythematosus. J Rheumatol 1992;19:47-52.
12. Stojanovich L, Zandman-Goddard G, Pavlovich S et al. Psychiatric manifestations in systemic lupus erythematosus. Autoimmun Rev 2007;6:421-6.
13. Pego-Reigosa JM, Isenberg DA. Psychosis due to systemic lupus erythematosus: characteristics and long-term outcome of this rare manifestation of the disease. Rheumatology 2008;47:1498-502.
14. Appenzeller S, Cendes F, Costallat LTL. Acute psychosis in systemic lupus erythematosus. Rheumatol Int 2008;28:237-43.
15. Ward MM, Studenski S. The time course of acute psychiatric episodes in systemic lupus erythematosus. J Rheumatol 1991;18:535-9.
16. Carrieri PB, Montella S, Petracca M. Psychiatric onset of multiple sclerosis: description of two cases. J Neuropsychiatry Clin Neurosci 2011;23:E6.
17. Kosmidis MH, Giannakou M, Messinis L et al. Psychotic features associated with multiple sclerosis. Int Rev Psychiatry 2010;22: 55-66.
18. Lee M-J, Lee H-S, Hwang J-S et al. A case of Hashimoto's encephalopathy presenting with seizures and psychosis. Korean J Pediatr 2012;55:111-3.
19. de Holanda N, de Lima D. Hashimoto's encephalopathy: systematic review of the literature and an additional case. J Neuropsychiatry Clin Neurosci 2011;23:384-90.
20. Shprintzen RJ, Goldberg R, Golding-Kushner KJ et al. Late-onset psychosis in the velo-cardio-facial syndrome. Am J Med Genet 1992;42:141-2.
21. Pulver AE, Nestadt G, Goldberg R et al. Psychotic illness in patients diagnosed with velo-cardio-facial syndrome and their relatives. J Nerv Ment Dis 1994;182:476-8.
22. Papolos DF, Faedda GL, Veit S et al. Bipolar spectrum disorders in patients diagnosed with velo-cardio-facial syndrome: does a hemizygous deletion of chromosome 22q11 result in bipolar affective disorder? Am J Psychiatry 1996;153:1541-7.
23. Murphy KC, Jones LA, Owen MJ. High rates of schizophrenia in adults with velo-cardio-facial syndrome. Arch Gen Psychiatry 1999;56:940-5.
24. Chow EWC, Watson M, Young DA et al. Neurocognitive profile in 22q11 deletion syndrome and schizophrenia. Schizophr Res 2006;87:270-8.
25. Vogels A, Matthijs G, Legius E et al. Chromosome 15 maternal uniparental disomy and psychosis in Prader-Willi syndrome. J Med Genet 2003;40:72-3.
26. Vogels A, De Hert M, Descheemaeker MJ et al. Psychotic disorders in Prader-Willi syndrome. Am J Med Genet A 2004;15; 127A:238-43.
27. Folstein SE, Chase GA, Wahl WE et al. Huntington disease in Maryland: clinical aspects of racial variation. Am J Hum Genet 1987;41:168-79.
28. Shiwach R. Psychopathology in Huntington's disease patients. Acta Psychiatr Scand 1994;90:241-6.
29. Bourgeois JA. Fahr's disease – a model of neuropsychiatric illness with cognitive and psychotic symptoms. Acta Psychiatr Scand 2010;121:78.
30. Shakibai SV, Johnson JP, Bourgeois JA. Paranoid delusions and cognitive impairment suggesting Fahr's disease. Psychosomatics 2005;46:569-72.
31. Curran C, Byrappa N, McBride A. Stimulant psychosis: systematic review. Br J Psychiatry 2004;185:196-204.
32. Post RM. Cocaine psychoses: a continuum model. Am J Psychiatry 1975;132:225-31.
33. Thirthalli J, Benegal V. Psychosis among substance users. Curr Opin Psychiatry 2006;19:239-45.
34. Fergusson DM, Horwood LJ, Swain-Campbell NR. Cannabis dependence and psychotic symptoms in young people. Psychol Med 2003;33:15-21.

35. Fergusson DM, Horwood LJ, Ridder EM. Tests of causal linkages between cannabis use and psychotic symptoms. Addiction 2005; 100:354-66.
36. Erard R, Luisada PV, Peele R. The PCP psychosis: prolonged intoxication or drug-precipitated functional illness? J Psychedelic Drugs 1980;12:235-51.
37. Jansen KL. A review of the nonmedical use of ketamine: use, users and consequences. J Psychoactive Drugs 2000;32:419-33.
38. Hambrecht M, Häfner H. Do alcohol or drug abuse induce schizophrenia? Nervenarzt 1996;67:36-45.
39. Dalmau A, Bergman B, Brismar B. Psychotic disorders among inpatients with abuse of cannabis, amphetamine and opiates. Do dopaminergic stimulants facilitate psychiatric illness? Eur Psychiatry 1999;14:366-71.
40. Cami J, Farré M, Mas M et al. Human pharmacology of 3,4-methylenedioxymethamphetamine ("ecstasy"): psychomotor performance and subjective effects. J Clin Psychopharmacol 2000;20:455-66.
41. Cole JC, Sumnall HR. Altered states: the clinical effects of Ecstasy. Pharmacol Ther 2003;98:35-58.
42. Kahan VL. Paranoid states occurring in leaded-petrol handlers. J Ment Sci 1950;96:1043-7.
43. Schenk VW, Stolk PJ. Psychosis following arsenic (possibly thallium) poisoning. A clinical-neuropathological report. Psychiatr Neurol Neurochir 1967;70:31-7.
44. Dubovsky AN, Arvikar S, Stern TA et al. The neuropsychiatric complications of glucocorticoid use: steroid psychosis revisited. Psychosomatics 2012;53:103-15.
45. Feinberg M. The problems of anticholinergic adverse effects in older patients. Drugs Aging 1993;3:335-48.
46. Peters NL. Snipping the thread of life. Antimuscarinic side effects of medications in the elderly. Arch Intern Med 1989;149:2414-20.
47. Schor JD, Levkoff SE, Lipsitz LA et al. Risk factors for delirium in hospitalized elderly. JAMA 1992;267:827-31.
48. Marcantonio ER, Juarez G, Goldman L et al. The relationship of postoperative delirium with psychoactive medications. JAMA 1994;272:1518-22.
49. Schrestha S, Alao A. Isoniazid-induced psychosis. Psychosomatics 2009;50:640-1.
50. Tran TM, Browning J, Dell ML. Psychosis with paranoid delusions after a therapeutic dose of mefloquine: a case report. Malar J 2006;5:74.
51. Alisky JM, Chertkova EL, Iczkowski KA. Drug interactions and pharmacogenetic reactions are the basis for chloroquine and mefloquine-induced psychosis. Med Hypotheses 2006;67:1090-4.
52. Gustafson Y, Berggren D, Brännström B et al. Acute confusional states in elderly patients treated for femoral neck fracture. J Am Geriatr Soc 1988;36:525-30.
53. Meador KJ. Cognitive side effects of antiepileptic drugs. Can J Neurol Sci 1994;21:S12-6.
54. Inouye SK, Charpentier PA. Precipitating factors for delirium in hospitalized elderly persons. Predictive model and interrelationship with baseline vulnerability. JAMA 1996;275:852-7.
55. Bowen JD, Larson EB. Drug-induced cognitive impairment. Defining the problem and finding solutions. Drugs Aging 1993; 3:349-57.
56. Martin NJ, Stones MJ, Young JE et al. Development of delirium: a prospective cohort study in a community hospital. Int Psychogeriatr 2000;12:117-27.
57. Snavely S. The neurotoxicity of antibacterial agents. Ann Intern Med 1984;101:92-104.
58. Almeida OP, Xiao J. Mortality associated with incident mental health disorders after stroke. Aust N Z J Psychiatry 2007;41:274-81.
59. Kumral E, Oztürk O. Delusional state following acute stroke. Neurology 2004;62:110-3.
60. Nagaratnam N, O'Neile L. Delusional parasitosis following occipito-temporal cerebral infarction. Gen Hosp Psychiatry 2000; 22:129-32.
61. Beniczky S, Kéri S, Vörös E et al. Complex hallucinations following occipital lobe damage. Eur J Neurol 2002;9:175-6.
62. Nye E, Arendts G. Intracerebral haemorrhage presenting as olfactory hallucinations. Emerg Med 2002;14:447-9.
63. Starkstein S, Robinson R. Post-stroke hallucinatory delusional syndromes. Neuropsychiatry Neuropsychol Behav Neurol 1992; 5:114-8.
64. Lyketsos CG, Steinberg M, Tschanz JT et al. Mental and behavioral disturbances in dementia: findings from the Cache County Study on Memory in Aging. Am J Psychiatry 2000;157:708-14.
65. Sachdev PS, Keshavan MS. Secondary schizophrenia. Cambridge: Cambridge University Press, 2010.
66. Waggoner RW, Bagchi BK. Initial masking of organic brain changes by psychic symptoms: clinical and electroencephalographic studies. Am J Psychiatry 1954;110:904-10.
67. Klotz M. Incidence of brain tumors in patients hospitalized for chronic mental disorders. Psychiatr Q 1957;31:669-80.
68. Larson C. Intracranial tumors in mental hospital patients a statistical study. Am J Psychiatry 1940;97:49-58.
69. Mulder DW, Daly D. Psychiatric symptoms associated with lesions of temporal lobe. JAMA 1952;150:173-6.
70. Selecki BR. Intracranial space-occupying lesions among patients admitted to mental hospitals. Med J Aust 1965;1:383-90.
71. Malamud N. Psychiatric disorder with intracranial tumors of limbic system. Arch Neurol 1967;17:113-23.
72. Galasko D, Kwo-On-Yuen PF, Thal L. Intracranial mass lesions associated with late-onset psychosis and depression. Psychiatr Clin North Am 1988;11:151-66.
73. Edwards-Lee T. Focal lesions and psychosis. In: Bogousslavsky J, Cummings JL (eds). Behavior and mood disorders. Cambridge: Cambridge University Press, 2000:419-36.
74. Davison K, Bagley CR. Schizophrenia-like psychoses associated with organic disorder of the central nervous system: a review of the literature. In: Herrington RN (ed). Current problems in neuropsychiatry. Ashford: Headley Brothers, 1969:1-45.
75. Hollister LE, Boutros N. Clinical use of CT and MR scans in psychiatric patients. J Psychiatry Neurosci 1991;16:194-8.
76. Benes F. Why does psychosis develop during adolescence and early adulthood? Curr Opin Psychiatry 2003;16:317-9.
77. Feinberg I. Schizophrenia: caused by a fault in programmed synaptic elimination during adolescence? J Psychiatr Res 1982; 17:319-34.
78. Benes FM. Myelination of cortical-hippocampal relays during late adolescence. Schizophr Bull 1989;15:585-93.
79. Weinberger DR, Lipska BK. Cortical maldevelopment, anti-psychotic drugs, and schizophrenia: a search for common ground. Schizophr Res 1995;16:87-110.
80. Mizukami K, Sasaki M, Suzuki T et al. Central nervous system changes in mitochondrial encephalomyopathy: light and electron microscopic study. Acta Neuropathol 1992;83:449-52.
81. Suzuki T, Koizumi J, Shiraishi H et al. Psychiatric disturbance in mitochondrial encephalomyopathy. J Neurol Neurosurg Psychiatry 1989;52:920-2.
82. Amemiya S, Hamamoto M, Goto Y et al. Psychosis and progressing dementia: presenting features of a mitochondriopathy. Neurology 2000;55:600-1.
83. Huang CC, Chu NS. Wilson's disease: clinical analysis of 71 cases and comparison with previous Chinese series. J Formos Med Assoc 1992;91:502-7.
84. Rathbun J. Neuropsychological aspects of Wilson's disease. Int J Neurosci 1996;85:221-9.
85. Wilson S. Progressive lenticular degeneration. BMJ 1912; 2:1645.4.
86. Lindenbaum J, Healton EB, Savage DG et al. Neuropsychiatric disorders caused by cobalamin deficiency in the absence of anemia or macrocytosis. N Engl J Med 1988;318:1720-8.

87. Hutto BR. Folate and cobalamin in psychiatric illness. Compr Psychiatry 1997;38:305-14.
88. Evans DL, Edelsohn GA, Golden RN. Organic psychosis without anemia or spinal cord symptoms in patients with vitamin B12 deficiency. Am J Psychiatry 1983;140:218-21.
89. Masalha R, Chudakov B, Muhamad M et al. Cobalamin-responsive psychosis as the sole manifestation of vitamin B12 deficiency. Isr Med Assoc J 2001;3:701-3.
90. Payinda G, Hansen T. Vitamin B(12) deficiency manifested as psychosis without anemia. Am J Psychiatry 2000;157:660-1.
91. Levitt AJ, Joffe RT. Vitamin B12 in psychotic depression. Br J Psychiatry 1988;153:266-7.
92. Godfrey PS, Toone BK, Carney MW et al. Enhancement of recovery from psychiatric illness by methylfolate. Lancet 1990; 336:392-5.
93. Prakash R, Gandotra S, Singh LK et al. Rapid resolution of delusional parasitosis in pellagra with niacin augmentation therapy. Gen Hosp Psychiatry 2008;30:581-4.
94. Rudin DO. The major psychoses and neuroses as omega-3 essential fatty acid deficiency syndrome: substrate pellagra. Biol Psychiatry 1981;16:837-50.
95. Hare EH. The origin and spread of dementia paralytica. J Ment Sci 1959;105:594-626.
96. Sobhan T, Rowe HM, Ryan WG et al. Unusual case report: three cases of psychiatric manifestations of neurosyphilis. Psychiatr Serv 2004;55:830-2.
97. Klaus MV, Amarante L, Beam TR. Routine screening for syphilis is justified in patients admitted to psychiatric, alcohol, and drug rehabilitation wards of the Veterans Administration Medical Center. Arch Dermatol 1989;125:1644-6.
98. Hutto B. Syphilis in clinical psychiatry: a review. Psychosomatics 2001;42:453-60.
99. Taycan O, Ugur M, Ozmen M. Quetiapine vs. risperidone in treating psychosis in neurosyphilis: a case report. Gen Hosp Psychiatry 2006;28:359-61.
100. Güler E, Leyhe T. A late form of neurosyphilis manifesting with psychotic symptoms in old age and good response to ceftriaxone therapy. Int Psychogeriatr 2011;23:666-9.
101. Sewell DD. Schizophrenia and HIV. Schizophr Bull 1996; 22:465-73.
102. Dolder CR, Patterson TL, Jeste DV. HIV, psychosis and aging: past, present and future. AIDS 2004;18 (Suppl. 1):S35-42.
103. Harris MJ, Jeste DV, Gleghorn A et al. New-onset psychosis in HIV-infected patients. J Clin Psychiatry 1991;52:369-76.
104. Shedlack KJ, Soldato-Couture C, Swanson CL. Rapidly progressive tardive dyskinesia in AIDS. Biol Psychiatry 1994;35:147-8.
105. Yolken RH, Dickerson FB, Fuller Torrey E. Toxoplasma and schizophrenia. Parasite Immunol 2009;31:706-15.
106. Torrey EF, Yolken RH. Schizophrenia and toxoplasmosis. Schizophr Bull 2007;33:727-8.
107. Kramer W. Frontiers of neurological diagnosis in acquired toxoplasmosis. Psychiatr Neurol Neurochir 1966;69:43-64.
108. Freytag HW, Haas H. Psychiatric aspects of acquired toxoplasmosis. A case report. Nervenarzt 1979;50:128-31.
109. Brown AS. Epidemiologic studies of exposure to prenatal infection and risk of schizophrenia and autism. Dev Neurobiol (in press).
110. Brown AS, Begg MD, Gravenstein S et al. Serologic evidence of prenatal influenza in the etiology of schizophrenia. Arch Gen Psychiatry 2004;61:774-80.
111. Mortensen PB, Nørgaard-Pedersen B, Waltoft BL et al. Toxoplasma gondii as a risk factor for early-onset schizophrenia: analysis of filter paper blood samples obtained at birth. Biol Psychiatry 2007;61:688-93.
112. Babulas V, Factor-Litvak P, Goetz R et al. Prenatal exposure to maternal genital and reproductive infections and adult schizophrenia. Am J Psychiatry 2006;163:927-9.
113. Brown AS, Cohen P, Harkavy-Friedman J et al. Prenatal rubella, premorbid abnormalities, and adult schizophrenia. Biol Psychiatry 2001;49:473-86.
114. Patten SB, Svenson LW, Metz LM. Psychotic disorders in MS: population-based evidence of an association. Neurology 2005; 65:1123-5.
115. Feinstein A. The neuropsychiatry of multiple sclerosis. Can J Psychiatry 2004;49:157-63.
116. Feinstein A, Boulay du G, Ron MA. Psychotic illness in multiple sclerosis. A clinical and magnetic resonance imaging study. Br J Psychiatry 1992;161:680-5.
117. Ropacki SA, Jeste DV. Epidemiology of and risk factors for psychosis of Alzheimer's disease: a review of 55 studies published from 1990 to 2003. Am J Psychiatry 2005;162:2022-30.
118. Paulsen JS, Ready RE, Stout JC et al. Neurobehaviors and psychotic symptoms in Alzheimer's disease. J Int Neuropsychol Soc 2000;6:815-20.
119. Jeste DV, Finkel SI. Psychosis of Alzheimer's disease and related dementias. Diagnostic criteria for a distinct syndrome. Am J Geriatr Psychiatry 2000;8:29-34.
120. Rapoport MJ, van Reekum R, Freedman M et al. Relationship of psychosis to aggression, apathy and function in dementia. Int J Geriatr Psychiatry 2001;16:123-30.
121. Gustafson L, Brun A, Passant U. Frontal lobe degeneration of non-Alzheimer type. Baillieres Clin Neurol 1992;1:559-82.
122. Edwards-Lee T, Miller BL, Benson DF et al. The temporal variant of frontotemporal dementia. Brain 1997;120 (Pt 6):1027-40.
123. Logsdail SJ, Toone BK. Post-ictal psychoses. A clinical and phenomenological description. Br J Psychiatry 1988;152:246-52.
124. Kanner AM, Stagno S, Kotagal P et al. Postictal psychiatric events during prolonged video-electroencephalographic monitoring studies. Arch Neurol 1996;53:258-63.
125. Slater E, Beard AW, Glithero E. The schizophrenia-like psychoses of epilepsy. Br J Psychiatry 1963;109:95-150.
126. Qin P, Xu H, Laursen TM et al. Risk for schizophrenia and schizophrenia-like psychosis among patients with epilepsy: population based cohort study. BMJ 2005;331:23.
127. Standage KF, Fenton GW. Psychiatric symptom profiles of patients with epilepsy: a controlled investigation. Psychol Med 1975;5:152-60.
128. Katsigiannopoulos K, Georgiadou E, Pazarlis P et al. Psychotic disorder as a manifestation of Graves' disease. Psychosomatics 2010;51:449-50.
129. Chen T-S, Wen M-J, Hung Y-J et al. A rare storm in a psychiatric ward: thyroid storm. Gen Hosp Psychiatry 2012;34:210.e1–4.
130. Brownlie BE, Rae AM, Walshe JW et al. Psychoses associated with thyrotoxicosis – "thyrotoxic psychosis". A report of 18 cases, with statistical analysis of incidence. Eur J Endocrinol 2000;142:438-44.
131. Rizvi AA. "Thyrotoxic psychosis" associated with subacute thyroiditis. South Med J 2007;100:837-40.
132. Kobayashi N, Tajiri J, Takano M. Transient psychosis due to painless thyroiditis in a patient with anxiety disorder: a case report. J Med Case Reports 2011;5:534.
133. Sathya A, Radhika R, Mahadevan S et al. Mania as a presentation of primary hypothyroidism. Singapore Med J 2009;50:e65-7.
134. Tor PC, Lee HY, Fones CSL. Late-onset mania with psychosis associated with hypothyroidism in an elderly Chinese lady. Singapore Med J 2007;48:354-7.
135. Hall R. Psychiatric effects of thyroid hormone disturbance. Psychosomatics 1983;24:7-11.
136. Heinrich TW, Grahm G. Hypothyroidism presenting as psychosis: myxedema madness revisited. Prim Care Companion J Clin Psychiatry 2003;5:260-6.
137. Irwin R, Ellis PM, Delahunt J. Psychosis following acute alteration of thyroid status. Aust N Z J Psychiatry 1997;31:762-4.

138. Schofield A, Bracken P. Thyroid-induced psychosis in myxoedema. Ir Med J 1983;76:495-6.
139. Papa A, Bononi F, Sciubba S et al. Primary hyperparathyroidism: acute paranoid psychosis. Am J Emerg Med 2003;21:250-1.
140. Watson LC, Marx CE. New onset of neuropsychiatric symptoms in the elderly: possible primary hyperparathyroidism. Psychosomatics 2002;43:413-7.
141. Velasco PJ, Manshadi M, Breen K et al. Psychiatric aspects of parathyroid disease. Psychosomatics 1999;40:486-90.
142. Denko JD, Kaelbling R. The psychiatric aspects of hypoparathyroidism. Acta Psychiatr Scand 1962;38 (Suppl. 164):1-70.
143. Ang AW, Ko SM, Tan CH. Calcium, magnesium, and psychotic symptoms in a girl with idiopathic hypoparathyroidism. Psychosom Med 1995;57:299-302.
144. Lisanby S, Kohler C, Swanson C. Psychosis secondary to brain tumor. Semin Clin Neuropsychiatry 1998;3:12-22.
145. Illes J, Kirschen M, Edwards E et al. Incidental findings in brain imaging research: what should happen when a researcher sees a potential health problem in a brain scan from a research subject? Science 2006;311:783-84.
146. Lawrie SM, Abukmeil SS, Chiswick A et al. Qualitative cerebral morphology in schizophrenia: a magnetic resonance imaging study and systematic literature review. Schizophr Res 1997;24;25:155-66.
147. Keshavan MS, DeLisi LE, Seidman LJ. Early and broadly defined psychosis risk mental states. Schizophr Res 2011;126:1-10.
148. Shenton ME, Whitford TJ, Kubicki M. Structural neuroimaging in schizophrenia: from methods to insights to treatments. Dialogues Clin Neurosci 2010;12:317-32.
149. Keshavan MS, Tandon R, Boutros NN et al. Schizophrenia, "just the facts": what we know in 2008. Part 3: Neurobiology. Schizophr Res 2008;106:89-107.
150. Tandon R, Keshavan MS, Nasrallah HA. Schizophrenia, "just the facts": what we know in 2008. 2. Epidemiology and etiology. Schizophr Res 2008;102:1-18.
151. Trimble M. The psychoses of epilepsy. New York: Raven, 1991.
152. Torta R, Keller R. Behavioral, psychotic, and anxiety disorders in epilepsy: etiology, clinical features, and therapeutic implications. Epilepsia 1999;40 (Suppl. 10):S2-20.
153. Ando N, Morimoto K, Watanabe T et al. Enhancement of central dopaminergic activity in the kainate model of temporal lobe epilepsy: implication for the mechanism of epileptic psychosis. Neuropsychopharmacology 2004;29:1251-8.
154. Shashi V, Keshavan MS, Howard TD et al. Cognitive correlates of a functional COMT polymorphism in children with 22q11.2 deletion syndrome. Clin Genet 2006;69:234-8.
155. Shashi V, Kwapil TR, Kaczorowski J et al. Evidence of gray matter reduction and dysfunction in chromosome 22q11.2 deletion syndrome. Psychiatry Res 2010;181:1-8.
156. Kates WR, Antshel KM, Faraone SV et al. Neuroanatomic predictors to prodromal psychosis in velocardiofacial syndrome (22q11.2 deletion syndrome): a longitudinal study. Biol Psychiatry 2011;69:945-52.
157. Olney JW, Farber NB. Glutamate receptor dysfunction and schizophrenia. Arch Gen Psychiatry 1995;52:998-1007.
158. Moghaddam B. Bringing order to the glutamate chaos in schizophrenia. Neuron 2003;40:881-4.
159. Harrison PJ, Law AJ, Eastwood SL. Glutamate receptors and transporters in the hippocampus in schizophrenia. Ann N Y Acad Sci 2003;1003:94-101.
160. González-Burgos G, Hashimoto T, Lewis DA. Alterations of cortical GABA neurons and network oscillations in schizophrenia. Curr Psychiatry Rep 2010;12:335-44.
161. Potvin S, Stip E, Sepehry AA et al. Inflammatory cytokine alterations in schizophrenia: a systematic quantitative review. Biol Psychiatry 2008;63:801-8.
162. Dalmau J, Tüzün E, Wu H-Y et al. Paraneoplastic anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian teratoma. Ann Neurol 2007;61:25-36.
163. Dalmau J, Rosenfeld MR. Paraneoplastic syndromes of the CNS. Lancet Neurol 2008;7:327-40.

The Breivik case and what psychiatrists can learn from it

INGRID MELLE

K.G. Jebsen Centre for Psychosis Research, Institute of Clinical Medicine, University of Oslo and Oslo University Hospital, Nydalen, 0424 Oslo, Norway

In the afternoon of July 22, 2011, Norwegian Anders Behring Breivik killed 77 persons, many of them children and youths, in two separate events. On August 24, 2012, he was sentenced to 21 years in prison. Breivik went through two forensic evaluations: the first concluded that he had a psychotic disorder, thus being legally unaccountable, whereas the second concluded that he had a personality disorder, thus being legally accountable. This article first describes Breivik's background and his crimes. This is followed by an overview of the two forensic evaluations, their methods, contents and disagreements, and how these issues were handled by the court in the verdict. Finally, the article focuses on some lessons psychiatrists can take from the case.

Key words: Breivik's case, forensic psychiatry, psychiatric diagnosis, psychiatry and the media

July 22, 2011 was a quiet, gray and humid day in Oslo. As most Fridays in the middle of the Norwegian summer holiday, offices closed early and locals left the city to the tourists. At 15.25, Anders Behring Breivik detonated a 950-kg fertilizer-based car bomb in the downtown government quarter, killing eight persons and injuring nine severely. Only around 200 persons of the usual 1900 were still at work this late in the afternoon, and there was thus a short-lived relief that casualties were lower than initially feared. However, two hours later, reports of gunfire at the summer camp for the Norwegian labor party's youth organization came pouring in. Breivik had traveled directly from the bombsite to the small island of Utøya and gained access to the island ferry masquerading as a police officer. He almost immediately started shooting at the 600 persons trapped on the island and killed 69 persons, 59 of these born in or after 1990. Using hollow-point, expanding ammunition, he also caused many severe and disfiguring injuries. Survivors report that he went back to previous victims, shooting them repeatedly, and that several times he persuaded those hidden to come forward by saying he was a policeman. Survivors also report that he at times was laughing and shouting while shooting.

After 50 minutes, Breivik called the police saying: "Yes, hello, my name is Commander Anders Behring Breivik from the Norwegian anti-communist resistance movement. I'm on Utøya for the moment. I want to give myself up". Met with the standard follow-up questions of where he was calling from, he broke the connection and went back shooting. Twenty minutes later he surrendered to an armed police force with his arms over his head. In his first interrogation at Utøya, Breivik again presented himself as the commander of the Knights Templars Norway, an organization he claimed held 15–80 "Knights" in Europe in 2008, with himself having a central position in the Norwegian branch. He further told the police that his acts that day were "part of plan B", and "the unfortunate with what happened was that the people on the island were category C traitors". In the organization's view, there were category A, B, and C traitors. He had "the right to kill category A and B traitors, but not a mandate to kill category C traitors".

BACKGROUND

Breivik was born in Oslo in 1979. Both parents had children from previous relationships. They married shortly before his birth and moved abroad, where his father worked for the Norwegian foreign services. His parents divorced in 1980 and he grew up with his mother and half-sister in Oslo, with limited contact with his father outside holiday visits. His mother asked for help from the Child Welfare Services twice because she found him a difficult child, and in 1983 Breivik was examined by the Child Psychiatric Services. They evaluated his rearing situation as so problematic that he was in danger of developing more severe psychopathology and recommended foster care. However, the Child Welfare Services decided against, and after a short period of home supervision they closed the case in 1984.

Breivik went to schools in his affluent local area, where he was part of a group who later described him as somewhat shy, but sociable and loyal. As a teenager he was preoccupied with his physical appearance, worked out frequently, used anabolic steroids and had cosmetic nose surgery in his early twenties. Albeit intelligent, he dropped out of high school before final exams. He started out several companies, including sale of false diplomas over the Internet. Breivik presented himself as a successful businessman in this period, claiming he earned millions, whereas the police estimate that he was paid 4.5 million NOK (around 600,000 Euros) for false diplomas and sales on the stock exchange.

In 2006, he was declared bankrupt and moved in with his mother. His friends report changes in his behavior from this time onward. He became increasingly withdrawn, used

most of his time to play World of Warcraft online, and cut contact with his friends, who worried that he might suffer from gambling addiction. In 2009, he founded a farming company and in the spring of 2011 he rented a farm in a rural area outside Oslo, which made it possible for him to buy large amounts of fertilizers without attracting suspicion. This background information differs from Breivik's description in the document he posted online on July 22, 2011, his so-called Manifesto. There is general agreement that the main parts of its 1500 pages are cut-and-paste from other sources, to some extent extremist groups and right-wing bloggers, but also Karl Marx, Tony Blair, Osama bin Laden, and George W. Bush. In addition to outlining his extreme views on multicultural societies, Islam and Marxism, Breivik here presents an edited version of his own development. However, information from others casts considerable doubt around its accuracy. There is also significant doubt about the existence of the Knights Templars organization Breivik repeatedly claims was the initiator of his attacks. Other right-wing groups allegedly involved denied having any knowledge about it, and investigations by Norwegian and other police forces found no indications that the organization even exists.

BREIVIK'S PSYCHIATRIC EVALUATIONS

The current Norwegian criminal code has a maximum prison sentence of 21 years, with no additions for multiple victims. In the case of particularly serious acts, the offender can be sentenced to additional protective detention. Offenders found "not legally accountable" are sentenced to compulsory treatment. It is the court's obligation to evaluate if an accused person is legally accountable (1) and two forensic psychiatric experts are usually appointed to conduct a psychiatric evaluation. To what extent the report meets prerequisite formal requirements is evaluated by the Norwegian Board of Forensic Medicine, a part of the Norwegian Civil Affairs Authority. The report is presented to the court, which decides whether it will follow the advice of the appointed experts. Up to its presentation, the report is confidential under the Criminal Procedure Code, and disregard of confidentiality is punishable. Based on a legal tradition going back to Norway's first unified national code of law issued in 1274, the code reflects the view that offenders with severe mental disorders are legally unaccountable for their acts and should not be punished. The current definition of legal insanity, introduced in 1929 and last revised in 2002, states that a person is not criminally accountable if psychotic, unconscious, or severely mentally retarded at the time of the crime. "Psychotic" is here simply defined as "a condition that meets the criteria in the current diagnostic manuals". Therefore, the Norwegian law does not follow the stricter M'Naghten rule used by many other countries. This rule only accepts legal unaccountability in cases where the person is perceived "from disease of the mind, not to know the nature and quality of the act he was doing; or, if

he did know it, that he did not know he was doing what was wrong" (2).

The first pair of court-appointed psychiatrists had 13 interviews covering a total of 36 hours with Breivik, in addition to hearing or viewing all police interrogations and interviewing his mother. They combined unstructured talks with structured diagnostic interviews, including the Mini-International Neuropsychiatric Interview, the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) and the Positive and Negative Syndrome Scale (PANSS). The first interview was done on August 10, while Breivik was still in full isolation. The psychiatrists were initially asked to conduct their interviews through a glass partition, for security reasons, but declined. Because of time-consuming safety precautions seeing Breivik face-to-face, both psychiatrists were present during the interviews, even if separate interviews are recommended.

On November 29, 2011, the psychiatrists reported to the court that Breivik was psychotic while planning and implementing his acts and during the evaluation. As later explained during the trial, this conclusion was based on central contents of Breivik's thought system. He told them in the interviews that he had "precedence as the ideological leader for the Knights Templars organization, with the mandate of being both a military order, a martyr organization, a military tribunal, judge, jury and executioner". He thought he was a pioneer in a European civil war, and compared his situation to that of Tsar Nicolas of Russia and Queen Isabella of Spain. He believed that it was likely (with somewhat varying degrees of likelihood) that he could be the new regent in Norway following a coup d'état. He said he decided who should live and who should die in Norway. This responsibility was felt as real, but also a heavy burden. He believed that a considerable proportion of the Norwegian population (several hundred thousands) supported his deeds. If he became the new regent, he would take the name Sigurd the Crusader the Second (Sigurd the Crusader was a Norwegian medieval king who reclaimed parts of Portugal from Muslim rule). He claimed that he had given 5 million NOK to the fight. He thought he would be given the responsibility for deporting several hundred thousands of Muslims to North Africa. He believed there was an ongoing ethnic cleansing in Norway and feared for his life. He thought the events he was a part of could start a nuclear third world war. He worked with solutions to improve the Norwegian ethnic genetic pool, make illnesses extinct, and reduce the divorce rate. He thought about reservations for indigenous Norwegians, DNA testing, and factories for mass deliveries of babies. He believed that the house of Glucksburg (current Norwegian royal house) would be removed through revolution in 2020. As an alternative to recruiting a new regent from the leadership of the Knights Templars, one could make DNA tests of the remains of King Olav the Saint (the Viking

King who introduced Christianity to Norway) and then choose the one with best genetic likeness to be the new king.

The psychiatrists saw these as grandiose delusions with bizarre and paranoid qualities that went far beyond conspiracy notions about an Islamist take-over of Europe. Thus, they did not consider him psychotic by mistaking his extremist, racist, right-wing views as delusional, but because they thought he had grandiose delusions regarding *his own role* in this extremist universe. Although his political opinions unfortunately are shared by others, he stood alone in his claims of an exalted role in the alleged Knights Templars organization, or even in the claims of this organization's existence. In addition, Breivik claimed he had exceptional personal abilities, for instance knowing what other people – including his evaluators – thought, without fully explaining them how.

The two psychiatrists perceived his language as stilted and technical, using common words in new contexts mixed with unusual words, which he said he had made himself and that the psychiatrists perceived as neologisms. There were otherwise no signs of grossly disorganized speech or actions. He usually displayed restricted, but sometimes also inappropriate affect when talking about his killings, which he called "the executions of traitors". He got animated when talking about his shooting rampage and about his Manifesto. The psychiatrists saw this as an example of affective flattening with incidents of incongruent affect. There were no outward signs of depression, mania, auditory hallucinations or ideas of reference, influence phenomena or ideas of thought insertion. He had taken anabolic steroids in several periods up to July 22, combined with large doses of ephedrine, caffeine, and aspirin on the actual day. Blood samples taken at his arrest showed these substances in amounts that most likely could exacerbate, but not directly cause, mental symptoms. Based on Breivik's symptomatology, in particular the presence of bizarre grandiose delusions, the psychiatrists concluded that he had schizophrenia, paranoid type.

However, they had to wait more than 6 months before they could explain the basis for this conclusion. Meanwhile, the notion that Breivik might not be legally accountable for his acts caused extensive public discussion (3). Parts of the public were angry because they felt cheated from punishing him and worried that he might be freed too early. Many had difficulties understanding the concept of legal unaccountability – how could he be "not guilty"? Shortly, the report was leaked by one of the supportive councilors for the victims and the summary made accessible on the web by Norwegian newspapers, causing a new wave of discussions. Although several in the field of psychiatry saw the report as confidential and declined commenting it before trial, others started analyzing and criticizing excerpts of the report. Professionals were cited stating that a diagnosis of schizophrenia could be refuted based on the absence of pathognomonic symptoms, in this case particularly the lack of clear-cut Schneiderian first-rank delusions or auditory hallucinations. Breivik's lack of severe disorganization and his good planning abilities were also raised as counterarguments for a diagnosis of schizophrenia (4).

The discussion was fueled by intense media coverage, with repeated requests for a new evaluation by major newspapers and politicians, including the head of the Parliament's Standing Committee on Justice. The dispute was not appeased by the report's formal approval by the Norwegian Board of Forensic Medicine. Instead, this caused a flurry of conspiratory theories, from notions about collegial "cover-up" to outright suggestions that the Norwegian authorities had an interest in keeping the public in dark about the presence of right-wing extremists in their country. While Breivik's defense lawyer initially stated that the conclusion of the first evaluation did not surprise him, the most adamant protests came from Breivik himself. He did not want an "insanity defense" and would not evade responsibility or avoid trial. On the contrary, the mass murders were done with the explicit *intent* of achieving a heavily media-covered trial. The preparations for the trial thus turned into an effort for him to be declared sane, stating he would prefer death penalty to compulsory treatment (5).

In January 2013, the Oslo district court appointed a second pair of psychiatrists for a re-evaluation. This was performed in late February to early March, that is, 6 months after the first. By that time, Breivik had undergone weekly consultations with the prison's psychiatric treatment team since September. He was no longer in isolation and had access to the first psychiatric report and to details of the media discussions about his mental health. The main part of the new evaluation was based on the same format and the same instruments as the first, with the exception of the psychiatrists meeting Breivik separately. An inpatient observation was also performed in the prison by trained psychiatric personnel.

As in the first evaluation, neither the new pair of evaluators nor the observation staff saw any signs of gross disorganization or outward signs of auditory hallucinations. They also agreed with the first in that they were seeing a man with pathological self-aggrandizement. The main difference was that Breivik this time toned down the importance of the Knights Templars, described himself as a "foot-soldier" doing his duty and suggested that he earlier on had exaggerated his own role. The psychiatrists stated that he had "ideas of heightened self-worth, power and knowledge that may be reminiscent of what is observed in the case of delusional disorders". "Not least the ideas concerning the Knights Templars appear peculiar. He has however rationalized this and has explained that it is a *willed idea*". Regarding negative symptoms, they focused mainly on his social withdrawal, interpreted as a natural consequence of planning a terrorist attack. To what extent Breivik's apparent

indifference to his victims and sometimes incomprehensible affective displays could be a sign of affective disturbance was not discussed. Based on this, the psychiatrists concluded that Breivik's symptoms were due to a severe narcissistic personality disorder combined with pseudologia fantastica (pathological lying) (6), and that he was psychotic neither during their interviews nor at the time of his crimes, thus being legally accountable.

Thus, the main difference between the two evaluations is that 9 months after the attacks Breivik appeared more open to alternative explanations concerning his own role, which made the reality testing regarding his grandiose notions appear less impaired.

THE COURT TRIAL AND THE VERDICT

The trial in Oslo District Court took place from April 16 to June 22, 2012. Many TV stations and newspapers used "expert commentators" focusing on Breivik's state of mind throughout the trial, sometimes attempting to discern a diagnosis based on his appearance in court. In addition to the court-appointed psychiatrists, other psychiatrists and psychologists were called by Breivik's defense team or by the coordinating councils for the victims to testify. These included Breivik's prison psychiatrist and several of the most active media critics of the first evaluation report. The trial ended with the prosecution recommending that Breivik should be confined to psychiatric care, and the defense arguing that Breivik should be considered sane but acquitted as his actions were in self-defense.

The verdict was given on August 24, and, rather extraordinarily for a first-level court verdict, was not appealed. The court found Breivik accountable, and sentenced him to 21 years in preventive custody with a minimum time of 10 years. The court took as the basis for its verdict the second psychiatric report and the evaluations of other mental health professionals, including witnesses called by Breivik. The ruling starts out debating Breivik's possible diagnoses. As the diagnosis of schizophrenia in the first evaluation was based on the presence of bizarre delusions, the discussion focuses on that concept. The court here follows the ICD-10 definition ("persistent delusions of other kinds that are culturally inappropriate and completely impossible, e.g. being able to control the weather, or being in communication with aliens from another world") and refers to the concrete characterization of bizarreness given by the second pair of psychiatrists ("delusions involving phenomena which lie outside the realm of natural science"). Thus, they conclude that Breivik's absurd grandiose notions are non-bizarre and state that experts on right-wing ideologies should have been consulted before deciding that his perceptions of grandeur were culturally implausible. The court follows up with a series of common-sense alternative explanations of Breivik's statements and behaviors. His claim that he knew what other people were thinking could as likely be based on his experience as a telephone salesman; his withdrawal and suspiciousness could be a consequence of his terrorist plans; his odd choice of words could be explained as part of an online war-games/right-wing cultural sphere. The ruling recognizes Breivik's emotional bluntness but argues that his affective outbursts, such as crying over his own propaganda film in court, counts against an affective disturbance, in disregard of the clinical knowledge that flat and inappropriate affect are not mutually exclusive. It thereby concludes that Breivik does not meet (ICD-10) criteria for schizophrenia, apparently unaware that he still would meet the DSM-IV criteria.

In the next step discussing delusional disorder, the court follows the second evaluation which states that Breivik's ability to argue, present nuanced statements and be corrected, combined with an ability to keep plans concealed, rules out delusional disorder. The basis for this, in the court's view, is that persons with ideas of a psychotic nature will have a prominent urge to assert perceived injustice and would not be able to keep up good impulse control during the interviews. It is also unlikely that a person with a psychotic disorder would be able to dissimulate over time. The court finds support for the notion that Breivik is not psychotic from the reports of his treating psychiatrist and the psychiatric councilor to the prison governor, who both view his statements as expressions of a personality disorder with the more peculiar grandiose beliefs as primitive defense mechanisms. The court particularly emphasizes the 3-week round-the-clock observation by the hospital staff, engaging Breivik in small talk, preparing meals or playing jig-saw puzzles. However, the observers never challenged Breivik on his grandiose views or the existence of the Knights Templars organization, a task they considered part of the police work.

The court does not discuss in detail the second pair of psychiatrists' main diagnosis of a narcissistic personality disorder. However, its comments on the lack of necessity for also evaluating the DSM-IV general duration and severity criteria indicate that it may have missed the point that personality disorders are not cross-sectional diagnoses.

WHAT CAN PSYCHIATRISTS LEARN FROM THE BREIVIK CASE?

The Breivik case has received considerable international attention (7,8), and several aspects have relevance for psychiatrists.

The most baffling aspect of the verdict is the neglect of Breivik's role in shaping others' impressions. The witness reports taken as support for Breivik being non-psychotic are rather disconcerting, in their repeated descriptions of his politeness, his consideration, his skills in playing jig-saw puzzles, and his use of laughter as a "way of coping". Together with the commonsensical explanations for Breivik's unusual behavior, the verdict

conveys a picture that is very difficult to match with the descriptions given in the first evaluation. Moreover, the picture is very difficult to match with the survivors' description of a laughing killer of youths, regardless whether his motives were based on delusions, narcissistic rage, or evil.

The court reports clearly illustrate the odd effect Breivik seems to have had on all his evaluators, including the first, in generating reluctance to explore what might lie behind some of his strange utterances. As an illustration, when asked if he ever was in doubt about Breivik's sanity, one of the witnesses stated that he was that once, when Breivik in a discussion suggested that in the future people's brains could be directly linked to a computer, thus circumventing the need for expensive schooling. Instead of asking Breivik to extrapolate, the witness stated that he "rapidly *said to himself* that this was not a psychotic notion but rather a vision of the future".

The Breivik case shows the importance of the context in which psychiatric evaluations are made. In fact, the court interpreted diagnostic disagreement, in particular regarding the presence of bizarre delusions, as "differing interpretations of similar observations", ignoring the time difference between the two observations and the different situations in which they took place. It also highlights that a source of confusion may be represented by some subtle, but relevant, differences between the ICD-10 and the DSM-IV, and the fact that the ICD-10 is often adopted as a diagnostic system while the SCID, based on the DSM-IV, is sometimes used for diagnostic assessments. Moreover, it underscores that diagnostic criteria should not be regarded as rules of law, but as pragmatic definitions meant to capture symptoms and syndromes central to an illness, and that their use requires knowledge about the illness in question, and an understanding of the underlying clinical phenomena (9).

An important lesson from the Breivik case is that the complexity of forensic evaluations should lead professionals to be cautious about how they express themselves when taking position publicly. In the current case, diagnostic disagreement was front-page news. This conflict added momentum to the newspapers' claims about psychiatric failure, actively supported by persons or groups holding general antiexpert or specific antipsychiatry views. However, a poll indicated that the Breivik case has not changed the Norwegian public's view on forensic psychiatry, probably reflecting their acceptance of the implicit difficulties in forensic evaluations. The question on the front of most people's mind has not been diagnostic details, but what it "really means to be not accountable due to a mental disorder". This question cuts to the core of a dilemma that has occupied lawyers, philosophers, and psychiatrists for a long time, and to which there are no simple solutions. The evaluation of what went on in a person's mind while committing a crime will, de-

spite technical innovations, in the end continue to rely on personal evaluations and interpretations.

Psychiatrists involved in high-profile cases should expect significant public interest and media pressure. All psychiatrists involved in the Breivik case were followed by the media to a problematic extent, ranging from telephones around the clock to journalists contacting their families or listening in on closed meetings. As several of the larger Norwegian newspapers and the Norwegian public broadcasting company were extremely critical to the content and acceptance of the first report, those held responsible were exposed to harsh critique, including claims of incompetence, bias, and paranoia. However, despite claims made by the media, Norwegian psychiatrists were not unanimously critical to the first evaluation report. Based on the number of professionals refusing to comment on the evaluation report before the trial, it is obvious that many felt restricted by confidentiality issues. Also, although the main critics were not actively engaged in treatment or research on psychotic disorders, the main support to the first evaluation came from professionals working with psychotic disorders, familiar with the difficulties in evaluating uncooperative patients, and the diversity and fluctuations of clinical presentations. Especially researchers, familiar with using both the ICD-10 and the DSM-IV, were rather surprised by the heavy emphasis put on ICD-10 descriptions for reports otherwise relying on SCID as the main diagnostic instrument.

Another important lesson from the Breivik case is that psychiatrists, when engaging in public debates about mental disorders, should remember that these debates hold considerable interest for persons diagnosed with these disorders. One of the most problematic aspects of the Breivik case has been the notions about schizophrenia conveyed by the participating professionals. For instance, claiming that Breivik cannot be psychotic because he has no behavioral signs suggests that psychiatrists think that having schizophrenia always will be discernible from the person's behavior. Even more provoking for well-functioning persons with the disorder, some holding complicated jobs, are professionals' repeated claims that Breivik cannot suffer from schizophrenia as he shows good cognitive abilities.

The impact on Norwegian society of Breivik's acts has been substantial. The camp was a meeting place for youth from all over the country and everybody knows someone affected by the events. All Norwegians are thus glad that the case was not appealed and the victims are spared for another round with having Breivik's face displayed on all bulletin boards, giving him even more of the publicity he craved. However, the case will surely be followed by a re-evaluation of parts of the Norwegian criminal code, and we might hope that those involved will remember that "great cases like hard cases make bad laws".

References

1. Grondahl P. Scandinavian forensic psychiatric practices – an overview and evaluation. Nordic J Psychiatry 2005;59:92-102.
2. Syse A. Punishment, treatment and fair retribution. Tidsskr Nor Laegeforen 2012;132:841-3.
3. BBC News Europe. Anders Behring Breivik trial: day by day. www.bbc.co.uk.
4. Spiegel Online International. Experts disagree on psychological state of Norwegian killer. www.spiegel.de.
5. BBC News World. Norway massacre: Breivik declared insane. www.bbc.co.uk.
6. Newmark N, Adityanjee KJ. Pseudologia fantastica and factitious disorder: review of the literature and a case report. Compr Psychiatry 1999;40:89-95.
7. Wessely S. Anders Breivik, the public, and psychiatry. Lancet 2012;379:1563-4.
8. Lockey DJ. The shootings in Oslo and Utoya island July 22, 2011: lessons for the International EMS community. Scand J Trauma Resusc Emerg Med 2012;20:4.
9. Nordgaard J, Revsbech R, Sæbye D et al. Assessing the diagnostic validity of a structured psychiatric interview in a first-admission hospital sample. World Psychiatry 2012;11:181-5.

The Breivik case and "conditio psychiatrica"

Josef Parnas

Psychiatric Center Hvidovre and Center for Subjectivity Research, University of Copenhagen, Denmark

In Denmark, a Scandinavian country linguistically proximate to Norway, the Breivik case (BC) stirred a similar, media-based, diagnostic debate, involving lay public, psychiatrists, and other professionals. The BC merits a commentary, not related to the judicial/forensic specifics, but concerning the current status and problems of psychiatric diagnosis and profession that the account of this case (1) so emphatically brings forth.

More than 30 years ago, psychiatry, attempting to match somatic medicine in its scientific aspirations, underwent an "operational revolution", introducing criteria-based diagnoses and "operational definitions" of such criteria. The BC shows, quite dramatically, that such criteria are not, in fact, *operational* in the original sense of specifying *action rules* (2), intended to link the psychiatric concepts with their counterparts in reality (*operations*, as in: X is *harder* than Y because X *can make a scratch* on Y, but not vice versa). None of the divisive issues in the BC was disambiguated through an appeal to operational action rules. What "operational" criteria actually amount to is no more than simple, lay-language descriptions of symptoms and signs. Moreover, the operational project came at a price. It radically abridged, simplified and compressed the then existing corpus of clinical knowledge into diagnostic manuals accessible to *grand publique*, because written in lay-language and stripped of theoretical and psychopathological reflection. These manuals have been for long the main source of clinical knowledge for psychiatrists in training (3) and it is mistakenly assumed (4,5) that a structured interview, asking preformulated questions in a fixed sequence, is an adequate methodology for obtaining psycho-

diagnostic information, even in sensitive situations, where the patient can be expected to dissimulate (1). This "death" of psychopathology (3) has created an intellectual hypodensity that blurs the professional borders of psychiatry, thereby welcoming any opinion as a voice *a priori* worthy of attention, equal, and legitimate. Unfortunately, operational revolution also failed to fulfil its motivating promise of a breakthrough to actionable etiological knowledge. "A gaping disconnect" is now widely recognized between the impressive progress in genetics and neurosciences and "its almost complete failure" to elucidate the causes and guide the diagnosis and treatment of psychiatric disorders (6,7).

As the BC illustrates, psychiatry will continue to crucially depend on the distinctions in the phenomenal realm, that is, domains of experience, expression, behaviour, rationality, and so forth. However, at the same time, the BC reveals important problems there. The discussion of Breivik's potential psychosis/delusions appears to have revolved around apparently mutually independent issues (e.g., are his views really shared; how to consider his affect and isolation; how to view his peculiar linguistic expressions; is surveillance of behaviour an adequate substitute for the knowledge of his inner world; etc.). It seems forgotten that falsity of a thought content is *not* a definitive feature of delusion. Jaspers emphasized that his triad of falsity, conviction, and incorrigibility (imported into the current diagnostic criteria) was not *defining* but *merely suggestive* of delusion (8). Delusion usually involves alterations in the patient's subjective framework with its interconnecting perspectives on himself, world, and others (8,9). Delusion is, therefore, typically identified in a larger temporal, situational, and experiential context. The collateral information feeds into that context, which also entails considerations of "double book-keeping", ability to dissimulate, and (ir)rationality of the transition between belief and action.

The BC discussion reveals an implicit epistemological tension in the very conception of psychiatric diagnosis, a tension apparently only vaguely perceived by the discussing parties and rarely explicitly addressed in the literature. It is a tension between an operational, cross-sectional approach of "symptom counting", without a guiding hierarchy and intelligibility principle (10), and a more prototypical perspective, which articulates a psychopathological Gestalt, emerging from the interactions between the whole and its reciprocally implicative aspects (11,12). The BC also illustrates a universal interpersonal human attitude, often interfering with clinical tasks, the so-called "principle of charity" (13). This is a subconscious, automatic, compensating tendency to make one's interlocutor appear as more rational than he actually is, for example, by smoothening out the bumps of his reasoning, filling up the gaps of his logic, and normalizing the instances of his flagrant irrationality.

It is worthwhile to recall that the BC-type debate is not unique to our times. Foucault devoted a seminar to a quite similar public diagnostic discussion in 1835 (14). It concerned a suspected insanity of Pierre Rivière, a just literate peasant, who murdered his mother, sister, and brother and was able to present in his defence a 100-page account of his life and motivations.

Psychiatry was, is, and will continue to be an object of intense societal attention and extra-scientific pressures. Only rigorous psychopathological standards can empower psychiatry to fulfill its clinical obligations and resist or modify the external pressures.

References

1. Melle I. The Breivik case and what psychiatrists can learn from it. World Psychiatry 2013;12:16-21.
2. Hempel CG. Explanation and other essays in the philosophy of science. New York: Free Press, 1965.

3. Andreasen N. DSM and the death of phenomenology in America: an example of unintended consequences. Schizophr Bull 2007;33:108-12.
4. Nordgaard J, Revsbech R, Sæbye D et al. Assessing the diagnostic validity of a structured psychiatric interview in a first-admission hospital sample. World Psychiatry 2012;11:181-5.
5. Nordgaard J, Sass LA, Parnas J. The psychiatric interview: validity, structure and subjectivity. Eur Arch Psychiatry Clin Neurosci (in press).
6. Frances AJ, Widiger T. The psychiatric diagnosis: lessons from DSM-IV past and cautions for the DSM-5 future. Annu Rev Clin Psychol 2012; 8:109-30.
7. Hyman SE. Psychiatric drug discovery: revolution stalled. Sci Transl Med 2012; 155:1-5.
8. Jaspers K. General psychopathology. London: John Hopkins University Press, 1963.
9. Spitzer M. On defining delusions. Compr Psychiatry 1990;31:377-97.
10. McHugh PR. Rendering mental dis-orders intelligible: addressing psychiatry's urgent challenge. In: Kendler K, Parnas J (eds). Philosophical issues in psychiatry II: nosology. Oxford: Oxford University Press, 2012: 42-53.
11. Parnas J. The core Gestalt of schizophrenia. World Psychiatry 2012;11:67-9.
12. Parnas J, Sass LA, Zahavi D. Rediscovering psychopathology: the epistemology and phenomenology of the psychiatric object. Schizophr Bull (in press).
13. Davidson D. Inquiries into truth and interpretation. Oxford: Clarendon Press, 1974.
14. Foucault M. Moi, Pierre Rivière, ayant égorgé ma mère, ma sœur et mon frère. Un cas de parricide au XIX siècle. Paris: Gallimard, 1973.

Should mental disorders be regarded as brain disorders? 21st century mental health sciences and implications for research and training

DEREK BOLTON

Institute of Psychiatry and Centre for the Humanities and Health, King's College London, South London and Maudsley NHS Foundation Trust, London, UK

Two recent papers focus our attention on developments in mental health sciences and their implications, respectively, for research, by Insel et al (1), and for the psychiatry profession, by White et al (2). Both papers headline the classification problem and both propose to regard mental disorders as brain disorders. While this proposal is not new (3,4), what is striking is that these two recent formulations are plainly not reductionist. By this, I mean that they do not suppose that neural dysfunctions are the only causes of mental disorders, but rather recognize developments in mental health sciences showing that causes or risks of mental disorders may operate at many levels, including the genetic and the neural, the individual, the family environment, and the social context. Crucially, this view of multifactorial or multilevel view of causation (or risk) acknowledges and is intended to accommodate the fact that interventions at these various levels may affect onset and course, playing parts in primary prevention and management and treatment after. This is one aspect of developments in mental health sciences outlined by both papers; another, and the emphasis in both papers, is the massive recent developments in the fields of genetics and neuroscience.

There is something of a tension between these two aspects of the new mental health sciences. On the one hand, the importance of genetics and neuroscience can suggest that the important causal pathways are intra-organism, within the brain in particular, pulling toward reductionism, a plausible interpretation of the proposal that mental disorders are brain disorders. On the other hand, if the causal pathways really do run up and down the biopsychosocial system, in and out of the organism, then the brain is part of the system involved, so also may be the mental life of the patient and their life circumstances – a nonreductionist view, not well expressed by the proposal that mental disorders are brain disorders.

One way to examine this apparent tension is by considering the "biopsychosocial model" in the context of the new sciences. This model has recently come under criticism from Ghaemi (5) in its original version due to Engel, in which "all three levels, biological, psychological, and social, must be taken into account in every health care task" (6), a proposition which Ghaemi (5) understands as meaning that the three levels "are all, more or less equally, relevant, in all cases, at all times". Ghaemi rejects the biopsychosocial model in this sense and also rejects its traditional opposite,

biological reductionism, proposing to explore other options between these two extremes. In the brief characterization of new developments in the mental health sciences above, the intention was to say that causal pathways and hence interventions may involve all the three "levels" – the biological, the psychological, and the social – but this was not meant to imply that all the three levels were always causally involved, let alone always involved "equally".

The Research Domain Criteria (RDoC) framework proposed by Insel et al (1) has a matrix in which there are columns labeled genetic, molecular, cellular, neural circuitry, individual, family environment, and social context; in the rows, there are conditions that may be diagnostic or transdiagnostic. The authors say (1, p. 749): "Importantly, all of these levels [in the columns] are seen as affecting both the biology and psychology of mental illness. With the RDoC approach, independent variables for classification might be specified from any of these levels of analysis, with dependent variables chosen from one or more other columns". However, there is no implication here that all these levels of analysis will always have causal relevance, still less equal causal relevance – regardless of what conditions are entered in the rows. Depending on the condition, genetic risk may be more or less important, for example, as may be the potential for psychological therapy to make any sustainable difference to the primary problem, or the causal role of social factors and potential for effective intervention in this domain. To make the point at one extreme: some conditions that might go into the rows of the RDoC framework will have no ticks under any boxes indicating causal processes at levels other than, for instance, the genetic or the neural, such as Huntington's disease or concussion, that is, no psychological or social factors make any difference (though they may do if the row had "adjustment to"). That is to say, reductionism might be right in some cases and in some cases it is already known to be right; in other cases, the psychosocial might be more important, account for more of the variance in incidence or outcomes, than, for instance, genetic factors. In short, the new sciences for which RDoC provides a framework make discriminations between conditions in these respects.

A related aspect of the view of causation in the new sciences is that they emphasize the interplay between the internal biology, the environment, and individual differences. Causal interplay occurs in normative development and in the development and course of health conditions, in psychiatric conditions and in some general medical conditions such as

cardiovascular disease. The new sciences of genetics and gene–environment interactions through the life course can be expected to increase this emphasis on interactions, and it will require research attention to be given not only to genes and the brain but also to environmental impacts and their timing in interaction with internal processes. In this context, the research effort needs to span the biological, the psychological/behavioral, and the environmental/social context, and their interactions – not limited to a "brain science" that studies only what is inside the skull.

The question of the importance of biological factors in mental disorders and especially the proposition that mental disorders should be regarded and classified as brain disorders is often coupled with challenges facing the psychiatry profession (e.g., 2,4). Many recent within-psychiatry papers have referred to challenges facing the profession, including clarifying the distinctive task of medical psychiatry within multiprofessional mental health care provision and improving the too low rates of recruitment of medical graduates into psychiatry (2,4,7-10). The new sciences as indicated above generally reaffirm the position of psychiatry as a medical specialty by blurring the differences between mental health and physical health conditions: the same kinds of multifactorial pathways may be operating in both. Nevertheless, while this is correct, the common assumption is that mental health conditions involve much of the psychosocial, evidenced for example in many of the "rapid responses" in the *BMJ* (see www.bmj.com) following the publication of the White et al analysis (2). The RDoC framework (1) actually provides a way in which medical and psychiatric conditions could be compared, and let me make a suggestion: if you put 10 typical physical health conditions in the first 10 rows of the framework and 10 typical mental health conditions in the next 10 rows, and fill in the cells in each column with processes known to be causal and amenable to effective intervention in the condition (after onset) – then the density of the resulting matrix would be greater in the upper left and the lower right quadrants. Once they have onset, physical health conditions are more amenable to internal medical procedures and not much to psychosocial; conversely, mental health conditions are more amenable to psychosocial interventions. My only excuse for flying such a kite without book length examination is that it at least represents common prejudice. In any case, it would all likely look different again – the density patterns would shift – if the rows had "primary prevention of"; and it would shift around again if the rows had "person's attitude to their illness", or "attitude to risk", or "adjustment to/quality of life in", that is, factors relevant to decent health care.

Insofar as psychiatry has particular expertise in the management of psychosocial factors as well as internal biological factors, it is somewhat unlike the rest of medicine, in particular, unlike much of internal medicine, and this is probably one of the problems in defining medical psychiatry's distinctive role among the other psychosocial professions – clinical psychology and social work – and in medical graduate recruitment to the profession. However – and this is the main point I wish to make in this connection – these professional divisions and the educational traditions they embody do not make much sense from the point of view of the new mental health sciences. These new sciences do not work with ideological battles between the biological, the psychological, and the social, the old parallel universes with poor communication between them; rather they work with all of these factors and the diversity of interplay between them.

Whether our current professional boundaries and trainings are fit for the purpose of accommodating, assimilating, and applying the vast fields and subtleties of these new sciences is seriously open to doubt. Yet this is what we need, I suggest, to align future mental health services with the science. What is to be looked forward to – and I raise this issue as a clinical psychologist, not a psychiatrist – is not so much reestablishing psychiatry as a medical speciality, going over again a 100-year-old problematic, but the construction of a new curriculum covering genetics, neuroscience, psychology, and social determinants of health, for a new profession of consultants in mental health for the 21st century. This might provide better care for patients, relative to expenditure, and might also solve the recruitment problem.

References

1. Insel T, Cuthbert B, Garvey M et al. Research Domain Criteria (RDoC): toward a new classification framework for research on mental disorders. Am J Psychiatry 2010;167:748-51.
2. White PD, Rickards H, Zeman AZJ. Time to end the distinction between mental and neurological illnesses. BMJ 2012;344:e3454.
3. Bucknill JC. The diagnosis of insanity. Br J Psychiatry 1856;2:229-45.
4. Baker M, Menken M. Time to abandon the term mental illness. BMJ 2001;322:937.
5. Ghaemi N. The rise and fall of the biopsychosocial model. Br J Psychiatry 2009;195:3-4.
6. Engel GL. The biopsychosocial model and the education of health professionals. Ann N Y Acad Sci 1978;310:169-87.
7. Craddock N, Antebi D, Attenburrow MJ et al. Wake-up call for British Psychiatry. Br J Psychiatry 2008;193:6-9.
8. Pichot P. The history of psychiatry as a medical profession. In: Gelder MG, Lopez-Ibor JJ, Andreasen N (eds). New Oxford textbook of psychiatry, 2nd ed. Oxford: Oxford University Press, 2009:17-27.
9. Katschnig H. Are psychiatrists an endangered species? Observations on internal and external challenges to the profession. World Psychiatry 2010; 9:21-8.
10. Ghaemi N. Taking disease seriously: beyond "pragmatic" nosology. In: Kendler K, Parnas J (eds). Philosophical issues in psychiatry. II: nosology. Oxford: Oxford University Press, 2012:42-52.

Understanding and addressing religion among people with mental illness

KENNETH I. PARGAMENT[1,2], JAMES W. LOMAX[3]

[1]Institute for Spirituality and Health at the Texas Medical Center, Houston, TX; [2]Department of Psychology, Bowling Green State University, OH 43403-0228; [3]Menninger Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, Houston, TX, USA

This article reviews recent advances in the domain of psychiatry and religion that highlight the double-edged capacity of religion to enhance or damage health and well-being, particularly among psychiatric patients. A large body of research challenges stereotyped views of religion as merely a defense or passive way of coping, and indicates that many people look to religion as a vital resource which serves a variety of adaptive functions, such as self-regulation, attachment, emotional comfort, meaning, and spirituality. There is, however, a darker side to religious life. Researchers and theorists have identified and begun to study problematic aspects of religiousness, including religiously-based violence and religious struggles within oneself, with others, and with the divine. Religious problems can be understood as a by-product of psychiatric illness (secondary), a source of psychiatric illness (primary), or both (complex). This growing body of knowledge underscores the need to attend more fully to the potentially constructive and destructive roles of religion in psychiatric diagnosis, assessment, and treatment. In fact, initial evaluative studies of the impact of spiritually integrated treatments among a range of psychiatric populations have shown promising results. The article concludes with a set of recommendations to advance future research and practice, including the need for additional psychiatric studies of people from diverse cultures and religious traditions.

Key words: Religion, spirituality, religious resources, religious coping, religious struggles, religiously-based violence, religious assessment, spiritually integrated treatment

The last 20 years have witnessed a sharp rise of scientific interest in the links between religion and psychological functioning (1). In contrast to commonly held stereotypes about religion and mental health, a significant body of theory and research indicates that religion is a source of strength and resilience for many people, including those with serious psychiatric disorders (2). It remains true, however, that religion can be problematic for some patients.

This article begins by placing the current status of the field in historical context, noting the history of troubled relations that has marked psychiatry and religion. We then review recent advances in the domain of psychiatry and religion that highlight the double-edged capacity of religion to enhance or damage health and well-being, particularly among psychiatric patients. This article concludes by considering the implications of this literature for psychiatric assessment and treatment.

A TROUBLED HISTORY BETWEEN PSYCHIATRY AND RELIGION

There is a long history of suspicion and, at times, antagonism between psychiatry and religion. Freud famously stated that religion represents a "defense against childish helplessness" and went on to conclude that "surely [such] infantilism is destined to be surmounted" (3). Negative religious attitudes of this kind within psychiatry and other mental health disciplines were commonplace for much of the 20th century. Religion was often stereotyped as defensive or regressive in character, fostering a passive retreat from problems, the outright denial of pain and suffering, or florid symptomatology. For example, DSM-III contained a disproportionate number of examples of religious expressions of major psychopathology, such as religious hallucinations and delusions (4). Oftentimes, religious issues were simply ignored, as illustrated by infrequent references to religion within major psy-

chiatric texts and journals (5). Some religious groups reciprocated with negative attitudes of their own toward psychiatry and discouraged or prohibited adherents from seeking out psychiatric treatment (6).

The tensions between psychiatry and religion are rooted in a number of factors. These include: perceptions that the world views grounded in science and faith are fundamentally irreconcilable (7); considerably lower levels of religiousness among psychiatrists than in the general population (8), leading psychiatrists to underestimate the salience of religion to patients; and a lack of psychiatric education and training to help practitioners understand and address patients' religious resources and problems (9).

This picture has begun to change in recent years, reflecting the growth of theory and research on religion, psychological functioning, and psychiatric disorders as well as a recognition of the need for a culturally sensitive approach

to psychiatric care. The field is now moving to a more nuanced understanding of religion, one which is cognizant of its double-sided capacity to support and strengthen people grappling with serious mental illness or exacerbate their pain and suffering.

RELIGION AS RESOURCE

Empirical studies show that religion is one of the first resources people and their loved ones turn to when faced with serious illness (2). For example, according to one study of outpatients with serious mental illness in Los Angeles, more than 80% reported that they used religion to cope with their illness and 65% indicated that religion helped reduce the severity of their symptoms (10). Similar findings have been reported in other countries. In a study comparing European patients with psychosis to a nonpsychiatric control group, the psychiatric patients reported a larger number of religious beliefs and practices that offered comfort during times of stress (11). In another study of Hindu family caregivers of patients with schizophrenia in a public hospital in India, 90% reportedly coped by praying to God, and 50% reported that religion was a source of solace, strength, and guidance (12).

Several empirical studies also challenge overly simplistic views of religion as a passive way of coping or a source of denial (2). For example, higher levels of religiousness have been associated with greater feelings of empowerment and self-efficacy among patients with serious mental illness (13), and more active attempts to resolve problems among women with ovarian cancer (14). In contrast to the view that religiousness promotes avoidance and denial, studies have linked greater religious involvement among women to a shorter length of time to schedule a visit with a physician after noticing symptoms of breast cancer (15) and less reliance on thought suppression and denial as ways of coping among HIV-seropositive African American women (16). One interesting study

found that, while higher levels of religiousness were unrelated to reports of the *presence* of pain among cancer patients, religiousness was related to reports of lower *levels* of pain (17). These findings suggest that religion may be involved in the reinterpretation rather than denial of pain.

Theory and research have also shown that religion serves a variety of adaptive functions for people, including those with serious mental illness. A content analysis of semistructured interviews with Swiss patients with serious mental illness revealed several functions of religion: meaning, comfort, self-respect, self-confidence, compassion, hope, love, and acceptance (18). Although theorists have debated about the most central purposes of religion, part of the power of this phenomenon may lie in its ability to meet a diverse array of human needs.

Self-regulation

Building on Freud's view that religion helps control undesirable sexual and aggressive urges, more recent theorists have emphasized the evolutionary advantages of religiously-based mechanisms that foster regulation of human impulses and desires (19). Consistent with this theory, a number of empirical studies have shown robust ties between religiousness and greater behavioral restraint with respect to substance use, crime and delinquency, suicidality, and sexual promiscuity.

People with serious mental illness may find the self-regulation afforded by religion particularly valuable. In one study of Swiss patients with schizophrenia or schizoaffective disorder, greater use of religious coping was predictive of fewer negative symptoms and better social functioning and quality of life 3 years later (20).

Attachment and connectedness

Sociologist E. Durkheim maintained that religion is most importantly about providing people with connectedness and identity: "The idea of society", he said, "is the soul of religion" (21). His thesis has been supported by several

studies documenting that individuals who are more involved in public religious expressions (e.g., worship attendance) report a larger network of social relationships and greater social support (22). However, it is important to note that individuals with serious mental illness often feel alienated from their religious institutions or other people more generally as a result of stigmatization or a history of insecure attachments (23). Religion may offer some compensation to these individuals through more secure attachments to sacred beings (e.g., God, Jesus, higher powers) who are perceived as more available and more accessible than their mortal counterparts (24).

Several studies have linked individual's perceptions of a secure relationship with God to lower levels of psychological distress. Relevant here too are the frequent reports of continued attachment to a loved one who has died. In one study of bereaved parents of pediatric cancer patients, 88% reported a continued connection with their deceased child (25). One grieving mother said: "I talk to him all the time. I 'keep him up on' what's going on at home and with all of us. I feel the strongest connection at the cemetery. I imagine his spirit in the trees behind his grave. When I begin to talk to him the wind almost always rustles the leaves, which tells me he's there" (25).

Emotional comfort

Freud and other theorists have maintained that religion functions in large part to allay the individuals' anxieties in a world of powerful forces that point to human frailty and finitude. Many studies have supported this theoretical perspective (22). For example, higher levels of religiousness have been associated with lower levels of depression especially among people facing more severe life stressors (26), less complicated grief among family caregivers of patients with dementia (27), and less anxiety and perceived stress among patients with panic disorder (28).

Beliefs in an afterlife appear to play a particularly key role in reducing basic

existential anxieties. Experimental studies in the domain of terror management theory have revealed that reminders of personal mortality increase beliefs in an afterlife, and beliefs in an afterlife reduce the anxieties associated with dying (29).

Meaning

Based on his extensive anthropological field studies, C. Geertz concluded that the most critical function of religion is meaning making (30). He wrote: "the effort is not to deny the undeniable – that there are unexplained events, that life hurts, or that rain falls upon the just – but to deny that there are inexplicable events, that life is unendurable, and that justice is a mirage". When 2000 people were surveyed about why they were religious, the most common response was "religion gives meaning in life" (31).

One study of hospice care providers identified several forms of benevolent religious reframing that lent meaning to the experience, such as appraisals that caregiving offers an opportunity to grow spiritually or represents a part of God's plan or will (32). These appraisals were associated with more positive outcomes and purpose in life. In a longitudinal study of parents who had suffered the violent death of a child, those who made more use of religious resources were able to find more meaning in the loss of their child 5 years later (33). Working with Flemish patients with chronic pain, another group of researchers reported that praying was associated with greater pain tolerance, not pain severity; the relationship between prayer and tolerance was mediated by positive reappraisals of the pain (34).

Spirituality

To the religiously minded, the most important purpose of religion is spirituality itself. The world's religious traditions insist that the search for the sacred takes priority over more temporal matters, though these faiths also offer a way to reconcile the spiritual with

the human. Psychologist and theologian P. Johnson captured this sentiment: "it is the ultimate Thou whom the religious person seeks most of all" (35). From this perspective, people grappling with serious illness are motivated to sustain themselves spiritually as well as psychologically, socially, and physically (36). Empirical studies have shown that people are generally quite successful in maintaining their faith in difficult times. For example, in one investigation of individuals who had experienced multiple traumas, 73% reported no religious change after the second event (37).

RELIGION AS PROBLEM

A number of theorists have attempted to move beyond global, stereotyped views of religion to delineate more specific aspects of religion that may be responsible for its troubling links with personal and social pathology. For example, Allport (38) articulated several characteristics of an "immature religious sentiment": simplistic and undifferentiated; fanatical and impulsive; lacking influence over conduct and values; intolerant and incomplete; and internally conflicted and fragmented. Pruyser (39) said that the "seamy side" of faith is marked by a sacrifice of the intellect, regressive fantasies, an inability to tolerate freedom, and neurotic coping styles. According to Pargament (40), an unhealthy spirituality is "dis-integrated"; it is ill-equipped to deal with the full range of internal and external life demands because it lacks comprehensiveness, depth, flexibility, dynamism, balance, and coherence.

Two aspects of problematic religiousness have received particular attention in recent years: religious struggles and religiously-based violence.

Religious struggles

Although people with serious mental illness are more likely to report that religion is a resource than a source of problems, a significant minority indicate that their faith contributes to their sense of anger, guilt, suffering, and despair

(26). In recognition that religion can be distressing, DSM-IV-TR included under "other conditions that may be a focus of clinical attention" the category of religious and spiritual problems, such as loss or questioning of faith, spiritual emergency, and new religious movements (41).

Several researchers have begun to examine a related concept, religious struggles, defined as questions, tensions, and conflicts about spiritual issues within oneself (intrapsychic), with others (interpersonal), and with higher powers or God (divine) (42). For example, one young woman described divine struggles related to her bipolar illness: "I'm suffering, really suffering. My illness is tearing me down, and I'm angry at God for not rescuing me, I mean really setting me free from my mental bondage. I have been dealing with these issues for ten years now and I am only 24 years old. I don't understand why he keeps lifting me up, just to let me come crashing down again".

It is important to distinguish between religious struggles that are the end-result of psychopathology and those that lead to psychopathology (43). Secondary religious struggles are elicited by major traumas, including the diagnosis of a serious mental illness, which can shake or shatter an individual's most fundamental values and worldview. In this vein, according to one national survey of people in the United States, a wide variety of forms of psychopathology were associated with higher levels of religious struggles (44). Primary religious struggles trigger subsequent psychological problems. Several longitudinal studies have tied religious struggles to declines in mental health, physical health, and even greater risk of dying (45). Finally, in complex religious struggles, the problematic form of religiousness is both a cause and a consequence of psychopathology.

Religion-based violence

In spite of its capacity to foster compassion, humanness, and understanding, throughout the ages religion has at times been a source of abuse, persecution, terrorism, and genocide. These problems

continue today across the world, as illustrated by religiously-based terrorism, clergy sexual abuse, and religiously-supported genocide. To take one among unfortunately many examples, in 2001, a Belgian court convicted two Benedictine nuns, Sisters G. Mukangango and J. Kisito, of participating in the massacre of more than 7600 Tutsis at the Sovu convent in Butare (46).

No single explanation is likely to offer a complete understanding of a process as complex as religious violence. Theorists, however, have suggested several psychological and social causal factors: a) felt experiences of shame and humiliation that may be evoked by present or historic events; b) a splitting of the world into all-good and evil-demonic camps, based on an inability to tolerate ambivalence and ambiguity; c) submission to an overly idealized, yet humiliating institution based on a fear of abandonment; d) threats to ideals, ideologies, or institutions that evoke narcissistic rage; e) an insistence on total perfection and purification; f) doctrinal linkages between violence and purification; and g) sexual repression (47). Particular attention has been paid by some theorists to the critical role of religious leaders in fomenting violence. For example, Olsson (48) maintains that destructive cult leaders manifest a malignant form of narcissistic personality disorder in which, by virtue of their ability to manipulate and inflict pain and suffering on others, they are able to assuage their own history of rejection and abandonment and support their inflated sense of themselves.

Empirical research in this critically important area is in short supply. However, some studies have provided support for these theoretical perspectives. For example, in one set of experimental studies, exposure to divinely sanctioned violence in the scriptures was tied to increases in aggressiveness, especially for more religious individuals. The authors (49) concluded that "to the extent religious extremists engage in prolonged, selective reading of the scriptures, focusing on violent retribution toward unbelievers instead of the overall message of acceptance and understanding, one might expect to see increased brutality". Other

studies have found that people who demonstrate an extrinsic religious orientation, one in which religion is used as a tool to serve nonreligious as opposed to spiritual ends, are more likely to demonstrate prejudice (50). These findings might also well apply to the problem of clergy sexual abuse, in which ministers hide behind the cloak of religion to gain access to vulnerable populations for their own destructive purposes. Still other research has suggested that people who believe that their sacred values are under attack are more willing to engage in aggressive, extremist responses (51).

IMPLICATIONS FOR PSYCHIATRIC PRACTICE

In recent years, theory and research within psychiatry and related mental health fields have challenged negative stereotypes about religion and led to a more nuanced view that recognizes the double-sided capacity of religion to foster both problems and solutions, distress and relief, among people with serious mental illness. Signs of the Janus-like character of religion can even be found within the same individual. In one study of patients dealing with religious delusions (52), many individuals reported that their faith was both a help and a hindrance, as illustrated in the following quote by a patient experiencing a malevolent spiritual aura: "The auras say 'we will catch him' and 'we will kill him', and they make me feel external pain. I spoke to the priest about the auras, and he helped me to find the courage to fight. God loves me and comforts me. With the help of God, I am winning against the auras".

A more differentiated view of religion holds significant implications for assessment and treatment.

Diagnosis and assessment

Recent advances in this area of study point to the need to attend more fully to the religious dimension in the process of assessment (40). Religion should be routinely included in the list of potential resources patients may

draw on in dealing with their illness and other life stressors. Patients should be asked whether they have religious resources they can access to help them cope with their problems, just as they are asked about other resources, such as their family and social network, hobbies, and exercise. Conversely, it is important to examine whether religion is problematic for the patient. A simple question, such as "Have your problems affected your religiousness or spirituality?", can open the door to an exploration of religious struggles or other religious problems.

In the process of assessing religious problems, it is important to consider whether the problems are secondary to the psychiatric symptomatology, primary sources of psychiatric distress, or complex. This basic distinction will have significant implications for subsequent treatment. In many cases, problems in the spiritual realm such as religious hallucinations and delusions can be best understood as secondary to a primary psychiatric illness; religion in these instances may simply be the idiom or language through which the illness is expressed (53). Treatment of the psychiatric illness among people with secondary religious problems may result in spiritual as well as emotional improvement. However, in other cases in which religion is in itself a primary source of distress, a focus on the psychiatric illness may not be sufficient to produce significant change. Spiritually integrated treatments that address the religious dimension of the problem may be needed to facilitate progress.

It is important to add that a spiritually sensitive approach to diagnosis and assessment calls for a full appreciation of the patient's cultural context. Whether a religious belief is delusional and problematic generally cannot be determined on the basis of the content of the belief (54). As Miller and Kelley noted (55), "in some African communities, a person would be considered insane *not* to believe that the spirits of the dead actively influence an individual's life". In some cases, it is appropriate and necessary to consult with

members of a patient's religious subculture to determine whether a set of religious beliefs are normative within that particular context. More generally, psychiatric diagnosis and assessment must incorporate an understanding of the beliefs, practices, and values that define the patient's world.

Treatment

Religion speaks to highly sensitive issues that lie at the core of the individual's identity, commitments, values, and worldview. This holds true for people with serious mental illness. Patients are unlikely to engage in a conversation about the deepest side of themselves unless their psychiatrist demonstrates an openness to, interest in, and appreciation of the patient's religiousness. Case studies have illustrated how practitioners can provide this kind of sensitive, spiritually affirming care to patients without commenting on the ontological reality of the patient's experience, something that lies beyond the knowledge or authority of mental health professionals (56). In this sense, psychiatrists can approach the patient's religiousness in the same fashion as other dimensions of life. Speaking to this point from a psychodynamic perspective, Rizzuto (57) stated: "The analyst owes the patient a full analytic experience in which his [or her] private religious world is explored with the same attentiveness and respectful exploration as the rest of his [or her] psychic life". Several practitioners have reported that patients welcome this type of spiritually sensitive and integrative approach to treatment (58,59).

One of the most important questions is whether a spiritually integrated approach to treatment is, in fact, as effective or even more effective than traditional methods of care. Several studies have been conducted on this question, and the initial results are promising. One set of studies has examined the effectiveness of treatments that encourage the patient to draw more fully on his/her religious resources. Spiritually integrated treatments have shown better results than various comparative treatments in a number of groups, including: Muslim patients from Malaysia diagnosed with generalized anxiety disorder, dysthymic disorder, and major depression (60); depressed patients and patients with schizophrenia in Australia (61); women from the Church of the Latter Day Saints diagnosed with eating disorders in an inpatient setting (62); Jews dealing with subclinical worry and anxiety (63); and treatment-resistant opioid-dependent patients in the United States (64). Encouraging findings have also been reported in a few evaluative studies on the effects of spiritually integrated treatments that address religious problems among patient populations in the United States, including military veterans dealing with post-traumatic stress disorder symptoms (65) and women who have been sexually abused (66).

PROMISING DIRECTIONS

In spite of the knowledge that has been gained on the relationship between religion and serious mental illness, questions continue to outnumber answers. There are several promising areas for further work in this domain (67). First, most of the research and practice in this area has focused on Western cultures. The findings from this particular context have to be extended to other religious groups, countries, and cultures. Second, further studies are needed which can help psychiatrists identify and distinguish between religious resources and religious problems across religious groups and cultures as well as psychiatric diagnoses. Third, longitudinal research is called for that helps disentangle the complex interplay between religion and psychopathology; specifically, the ways mental illness may elicit secondary religious problems and/or lead people to seek out solace and support from their faith, and the ways religious resources and primary religious problems may ameliorate and/or exacerbate the trajectory of illness. Studies of religiousness defined globally by a few indicators such as frequency of prayer or religious affiliation should give way to more specific investigations of particular forms of religious expression among people grappling with specific forms of mental illness. For instance, when might religious conversion play a protective role against psychotic illness; conversely, when might it foster serious psychological problems? Do religious and nonreligious delusions and hallucinations have a different set of etiological factors and consequences? Fourth, additional studies are needed to answer key questions about the religious dimension of psychiatric care. Do spiritually integrated treatments enhance the effects of standard psychiatric treatment? What impact, if any, does religious similarity or dissimilarity between doctor and patient have on treatment process and outcome? What effects does psychiatric medication have on the religious problems, beliefs, practices, and experiences of patients? Fifth, for this area of work to advance, effective models of psychiatric training need to be developed, tested, and implemented (68,69). Finally, although theory, research, and practice in the area of religion and serious mental illness is still in its early stages, it is becoming clear that excellence in mental health care will involve the creation of respectful, collaborative relationships between psychiatry and the leaders and members of religious communities.

References

1. Koenig HG, King D, Carson V. Handbook of religion and health, 2nd ed. New York: Oxford, 2012.
2. Pargament K. The psychology of religion and coping: theory, research, practice. New York: Guilford, 1997.
3. Freud S. The future of an illusion. New York: Norton, 1927/1961.
4. Richardson JT. Religiosity as deviance: negative religious bias and misuse of the DSM-III. Deviant Behav 1993;1:1-21.
5. Weaver AJ, Samford JA, Larson DB et al. A systematic review of research on religion in four major psychiatric journals: 1991–1995. J Nerv Ment Dis 1998;186:187-90.
6. Bobgan M, Bobgan D. Psychoheresy: the psychological seduction of Christianity. San Francisco: East Gate, 1987.
7. Barbour JG. Myths, models and paradigms: a comparative study in science and religion. New York: Harper & Row, 1974.
8. Shafranske EP. Religious involvement and professional practices of psychiatrists

and other mental health professionals. Psychiatr Ann 2000;30:1-8.

9. Larson D, Lu F, Swyers J. A model curriculum for psychiatry residence training programs: religion and spirituality in clinical practice. Rockville: National Institute for Healthcare Research, 1997.

10. Tepper L, Rogers SA, Coleman EM et al. The prevalence of religious coping among patients with persistent mental illness. Psychiatr Serv 2001;52:660-5.

11. Neeleman J, Lewis G. Religious identity and comfort beliefs in three groups of psychiatric patients and a group of medical controls. Int J Soc Psychiatry 1994; 40:124-34.

12. Rammohan A, Rao K, Subbakrishna DK. Religious coping and psychological well-being in carers of relatives with schizophrenia. Acta Psychiatr Scand 2002;105: 356-62.

13. Yangarber-Hicks N. Religious coping styles and recovery from serious mental illness. J Psychol Theol 2004;32:305-17.

14. Canada AL, Parker PA, deMoor JS et al. Active coping mediates the association between religion/spirituality and quality of life in ovarian cancer. Gynecol Oncol 2006;101:102-7.

15. Friedman LC, Kalkidas M, Elledge R et al. Medical and psychosocial predictors of delay in seeking medical consultation for breast symptoms in women in a public sector setting. J Behav Med 2006;19:327-34.

16. Prado G, Feaster DJ, Schwartz SJ et al. Religious involvement, coping, social support, and psychological distress in HIV-seropositive African American mothers. AIDS Behav 2004;8:221-35.

17. Yates JW, Chalmer BJ, St. James P et al. Religion in patients with advanced cancer. Med Ped Oncol 1981;9:121-8.

18. Mohr S, Brandt PY, Borras L et al. Toward an integration of religiousness and spirituality into the psychosocial dimension of schizophrenia. Am J Psychiatry 2006;163:1952-9.

19. McCullough ME, Willoughby BLB. Religion, self-regulation, and self-control: associations, explanations, and implications. Psychol Bull 2009;135:69-93.

20. Mohr S, Perroud N, Gillieron C et al. Spirituality and religiousness as predictive factors of outcomes in schizophrenia and schizo-affective disorders. Psychiatry 2010;186:177-82.

21. Durkheim E. The elementary forms of the religious life. New York: Free Press, 1965.

22. Pargament KI, Cummings J. Anchored by faith: religion as a resilience factor. In: Reich J, Zautra AJ, Hall JS (eds). Handbook of adult resilience. New York: Guilford, 2010:193-212.

23. Huguelet P, Mohr S, Borras L et al. Spirituality and religious practices among outpatients with schizophrenia and their clinicians. Psychiatr Serv 2006;57:366-72.

24. Granqvist P, Kirkpatrick LA. Religion, spirituality and attachment. In: Pargament KI, Exline JE, Jones J (eds). APA handbook of psychology, religion, and spirituality (Vol. 1). Washington: APA Press, 2013:139-56.

25. Sormanti M, August J. Parental bereavement: spiritual connections with deceased children. Am J Orthopsychiatry 1997;6: 460-9.

26. Smith TB, McCullough ME, Poll J. Religiousness and depression: evidence for a main effect and the moderating influence of stressful life events. Psychol Bull 2003;129:614-36.

27. Hebert RS, Dang Q, Schulz R. Religious beliefs and practices are associated with better mental health in family caregivers of patients with dementia: findings from the REACH study. Am J Geriatr Psychiatry 2007;15:292-300.

28. Bowen R, Baetz M, D'Arcy C. Self-rated importance of religion predicts one-year outcome of patients with panic disorder. Depress Anxiety 2006;23:266-73.

29. Soenke M, Landau MJ, Greenberg J. Sacred armor: religion's role as a buffer against the anxieties of life and the fear of death. In: Pargament KI, Exline JE, Jones J (eds). APA handbook of psychology, religion, and spirituality (Vol. 1). Washington: APA Press, 2013:105-22.

30. Geertz C. Religion as a cultural system. In: Banton M (ed). Anthropological approaches to the study of religion. London: Tavistock, 1966:1-46.

31. Clark WH. How do social scientists define religion? J Soc Psychol 1958;47:143-7.

32. Mickley JR, Pargament KI, Brant CR et al. God and the search for meaning among hospice caregivers. Hospice J 1998;13:1-17.

33. Murphy SA, Johnson LC, Lohan J. Finding meaning in a child's violent death: a five-year prospective analysis of parents' personal narratives and empirical data. Death Studies 2003;27:381-404.

34. Dezutter J, Wachholtz A, Corveleyn J. Prayer and pain: the mediating role of positive re-appraisal. J Behav Med 2011; 34:542-9.

35. Johnson P. Psychology of religion. Nashville: Abingdon, 1959.

36. Dein S, Pargament KI. On not praying for the return of an amputated limb: conserving a relationship with God as the primary function of prayer. Bull Menninger Clin 2012;76:235-59.

37. Falsetti SA, Resick PA, Davis JL. Changes in religious beliefs following trauma. J Trauma Stress 2003;16:391-8.

38. Allport GW. The individual and his religion: a psychological interpretation. Oxford: Macmillan, 1950.

39. Pruyser PW. The seamy side of current religious beliefs. Bull Menninger Clin 1977;41:329-48.

40. Pargament KI. Spiritually integrated psychotherapy: understanding and addressing the sacred. New York: Guilford, 2007.

41. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed., text revised. Washington: American Psychiatric Association, 2000.

42. Pargament KI, Murray-Swank N, Magyar G et al. Spiritual struggle: a phenomenon of interest to psychology and religion. In: Miller WE, Delaney W (eds). Judeo-Christian perspectives on psychology: human nature, motivation, and change. Washington: APA Press, 2005:245-68.

43. Pirutinsky S, Rosmarin DH, Pargament KI et al. Does negative religious coping accompany, precede, or follow depression among Orthodox Jews? J Affect Disord 2011;132:401-5.

44. McConnell K, Pargament KI, Ellison C et al. Examining the links between spiritual struggles and symptoms of psychopathology in a national sample. J Clin Psychol 2006;62:1469-84.

45. Exline J. Religious and spiritual struggles. In: Pargament KI, Exline JE, Jones J (eds). APA handbook of psychology, religion, and spirituality (Vol. 1). Washington: APA Press, 2013:459-76.

46. Waller JE. Religion and evil in the context of genocide. In: Pargament KI, Exline JE, Jones J (eds). APA handbook of psychology, religion, and spirituality (Vol. 1). Washington: APA Press, 2013:477-94.

47. Jones JE. Blood that cries out from the earth: the psychology of religious terrorism. Oxford: Oxford University Press, 2008.

48. Olsson PA. Malignant pied pipers of our time: a psychological study of destructive cult leaders from Rev. Jim Jones to Osama bin Laden. Baltimore: PublishAmerica, 2005.

49. Bushman BJ, Ridge RD, Das E et al. When God sanctions killing: effect of scriptural violence on aggression. Psychol Sci 2007;18:204-7.

50. Donahue MJ. Intrinsic and extrinsic religiousness: review and meta-analysis. J Pers Soc Psychol 1985;48:400-19.

51. Abu Raiya H, Pargament KI, Mahoney A et al. When Muslims are perceived as a religious threat: examining the connection between desecration, religious coping, and anti-Muslim attitudes. Basic Appl Soc Psychol 2008;30:311-25.

52. Mohr S, Borras L, Betrisey C et al. Delusions with religious content in patients with psychosis: how they interact with spiritual coping. Psychiatry 2010;73: 158-72.

53. Greenberg D, Witztum E. Sanity and sanctity: mental health work among the

ultra-orthodox in Jerusalem. New Haven: Yale University Press, 2001.

54. Pierre J. Faith or delusion? At the crossroads of religion and psychosis. J Psychiatr Pract 2001;7:163-72.

55. Miller L, Kelley BS. Relationships of religiosity and spirituality with mental health and psychopathology. In: Paloutzian RF, Park CL (eds). Handbook of the psychology of religion and spirituality. New York: Guilford, 2005:460-78.

56. Lomax JW, Kripal JJ, Pargament KI. Perspectives on 'sacred moments' in psychotherapy. Am J Psychiatry 2011;168:12-8.

57. Rizzuto AM, Shafranske E. Addressing religion and spirituality in treatment from a psychodynamic perspective. In: Pargament KI, Mahoney A, Shafranske E (eds). APA handbook of psychology, religion, and spirituality,Vol. 2. Washington: APA Press, 2013:125-46.

58. Stanley MA, Bush AL, Camp ME et al. Older adults' preferences for religion/spirituality in treatment for anxiety and depression. Aging Mental Health 2011; 15:334-43.

59. Kehoe N. Wrestling with our inner angels: faith, mental illness, and the jour-ney to wholeness. San Francisco: Jossey-Bass, 2009.

60. Azhar MZ, Varma SL, Dharap AS. Religious psychotherapy in depressive patients. Psychother Psychosom 1995;63: 165-73.

61. D'Souza R, Rich D, Diamond I et al. An open randomized controlled trial using a spiritually augmented cognitive behavioural therapy for demoralization and treatment adherence in patients with schizophrenia. Presented at the 37th Royal Australian and New Zealand College of Psychiatrists Congress, Brisbane, April 2002.

62. Richards PS, Berrett ME, Hardman RK et al. Comparative efficacy of spirituality, cognitive, and emotional support groups for treating eating disorder inpatients. Eat Disord 2006;41:401-15.

63. Rosmarin DH, Pargament KI, Pirutinsky S et al. A randomized controlled evaluation of a spiritually integrated treatment for subclinical anxiety in the Jewish community, delivered via the Internet. J Anxiety Disord 2010;24:799-808.

64. Avants SK, Beitel M, Margolin A. Making the shift from "addict self" to "spiritual self": results from a Stage I study of spiritual self-schema (3-S) therapy for the treatment of addiction and HIV risk behavior. Mental Health, Religion, and Culture 2005;8:167-77.

65. Harris JI, Erbes CR, Engdahl BE et al. The effectiveness of a trauma focused spiritually integrated intervention for veterans exposed to trauma. J Clin Psychol 2011;67:1-14.

66. Murray-Swank N, Pargament KI. God, where are you? Evaluating a spiritually-integrated intervention for sexual abuse. Mental Health, Religion, and Culture 2005;8:191-203.

67. Koenig HG. Schizophrenia and other psychotic disorders. In: Peteet JR, Lu FG, Narrow WE (eds). Religious and spiritual issues in psychiatric diagnosis: a research agenda for DSM-V. Arlington: American Psychiatric Association, 2011:31-51.

68. Blass DM. A pragmatic approach to teaching psychiatry residents the assessment and treatment of religious patients. Acad Psychiatry 2007;31:25-31.

69. Puchalski CM, Larson DB, Lu FG. Spirituality in psychiatry residency training programs. Int Rev Psychiatry 2001;13: 131-8.

Religiosity, a personality trait to be reckoned within psychiatry

HERMAN M. VAN PRAAG[1,2]

[1]University of Groningen, Utrecht, Maastricht, The Netherlands; [2]Albert Einstein College of Medicine, New York, NY, USA

Pargament and Lomax provide a lucid, cogent, succinct account of the interface between psychiatry and religion. However, they do not make a clear distinction between religiosity and religion. I do, and consider this point to be an important one, theoretically as well as practically.

I define *religiosity* as affinity for the religious root-idea. That idea entails that, apart from the world perceptible with our senses, another world exists, a supra-natural world. Men of faith feel the urge to reach out for that metaphysical world. They want to provide life with a vertical dimension, are receptive to the concept of God and know feelings, thoughts, experiences that are linked with that concept.

"Thinking upwards" – a term borrowed from de Ryk (1) – is no gratuitous business. It leads to something. It gains one access to a metaphysical world. A world beyond the horizon, a world completely irrational, impervious to rational, logical analysis. That world does not remain empty. Humans cannot easily manage a vacuum. They tend to fill it up. In case of a silence falling in a conversation, with words; in case of a view on a metaphysical universe, with undeterminable forces influencing one's personal life as well as that of the community of which one is part. The forces imagined to operate in this metaphysical universe may remain vague, unformed. In that case I speak of *spirituality*. Alternatively, the forces fancied are brought together in one, omnipresent, omnipotent, all-embracing mythical being, called God. In that case, I speak of *religiosity*.

That conception, that effigy, that likeness of God acquires a variety of functions. In the first place, it is a symbol; a symbol of unlimited crea-tivity and ultimate morality. As such it becomes for men of faith a role model, a touchstone for one's own behavior. The word symbol, meta-phor, lacks any denigrating connota-tion. A metaphor is the very means to express verbally something actually impossible to catch in words. It pro-vides an image of something ineffable and unimaginable.

Second, both fatherly and motherly qualities are projected in the God-ef-figy; in an idealized form. In the expe-riential world of the faithful, God is both advisor and supreme protector. He steps in in times of emotional up-heaval and provides solace or resigna-tion, if so needed.

In the third place, God, as perceived by the faithful, has expectations. He expects man (to phrase it in a Jewish fashion) to sanctify life. God encroaches on man's conscience; integrates with his conscience. In that capacity, God warns him when he threatens to go astray and stirs up guilt feelings if backsliding occurs nevertheless.

Finally, God provides men of faith with a destination, or better: He expects men to single out a destination. A desti-nation with an altruistic character; one that promises to contribute to the well-being of a society; on whatever level (social, cultural, scientific), or less lofty but no less important, by trying to make the best of it, in one's family, the work place, or in social life.

Thus, in the life of the faithful, the image of God functions as an important support system. It enlightens a life, pro-vided that religiosity has come to frui-tion without coercion, without pressure from without. It enlightens in the dual sense of that word. Life becomes lighter, less hard to live. It provides light, making it easier to find purpose and meaning in life, so that at the end of the journey one can say: it all made sense. I made a difference. The God-ef-figy is certainly not the sole provider of meaning, but no doubt an important one.

Religion, on the other hand, refers to a set of religious doctrines; actually to a philosophy, a way of interpreting the human existence, with the God-idea as focal point. Religion provides the urge "to think upward" with con-tent and form. Religion is, so to say, the formalized, structured, and often, unfortunately, codified expression form of religiosity. Religiosity is the infra-structure, religion the superstructure.

Religion is presented in various frames. On one extreme, one finds a, what I have called, coagulated, codi-fied set of rulings one is obliged to believe or to practice; rulings that, often, inhibit rather than encourage reflection, are apt to induce feelings of sin and shame, instead of generat-ing joy of living.

On the other extreme, one finds a view of life that captivates; is without difficulty incorporated in one's life; prompts discussion; stimulates reflec-tion as to purpose and meaning of one's life; a system that provides no certainties, only possibilities.

Religion may enrich a life or cor-rupt it. One may reject the system or embrace it, partly or entirely. It can be an influence for good or for ill. Over the centuries, it has often been an influence for evil. This has gotten religion a bad reputation.

This commentary concerns religios-ity. It was shown in all Western coun-tries that religiosity is a frequent phe-nomenon. It is expressed in many dif-ferent ways: belief in a personal god; belief in an omnipotent, abstract princi-ple; belief in after-life; belief in immor-tality of the soul; belief in a divine influ-ence in one's life, and many others. Church and church authorities lost prestige; religiosity appears to still enjoy

a strong presence. This evidence does suggest that religiosity is to be considered as a normal component of the human personality.

Other data support this conclusion. Religiosity is a feature that is biologically anchored. It is, to begin with, in part genetically determined (2). Second, several types of evidence indicate that the brain contains systems that are the root of religious experiences and considerations (2–5). If they are activated by electromagnetic currents, religious experiences occur, even in nonbelievers. Neuronal activity in those regions vary with the intensity and depth of religious devotion.

Does that mean that religiosity is caused biologically, that is no more than the product of some overactive neuronal circuits? No, that is not the right conclusion. It means that the brain is *intermediary* between religious needs and the gratification of those needs. The religious needs are of psychological origin, their gratification is made possible by the brain, by neurobiological processes.

I conclude: religiosity is an *attribute* of the human mind. Religion is a *product* of the human mind. Religion can be disposed of. Religiosity is there, ingrained in our very being. It is there to stay (6).

This being so, psychiatrists cannot ignore religiosity, whatever their own belief system may be. Careful personality analysis is core diagnostic business. Religiosity should not be missing in this undertaking (7). Did it play a role in the occurrence of the mental condition, and if so, was its influence aggravating or mitigating? Can the vertical dimension be utilized in psychological treatment, and if so, who is the designated therapist, the pastor or the psychiatrist, or should it be a tandem operation?

In short, religiosity matters in psychiatry. Psychiatrists cannot afford professional blind spots.

References

1. de Rijk LM. Religie, normen, waarden (Religion, norms, values). Amsterdam: Bert Bakker, 2008.
2. van Praag HM. Seat of the divine: a biological "proof of God's existence"? In: Verhagen PJ, van Praag HM, López-Ibor JJ, Jr. et al (eds). Religion and psychiatry. Beyond boundaries. New York: Wiley, 2010: 523-39.
3. Beauregard M, O'Leary D. The spiritual brain. New York: Harper, 2007.
4. Perroud N. Religion/spirituality and neuropsychiatry. In: Huguelet P, Koenig HG (eds). Religion and spirituality in psychiatry. Cambridge: Cambridge University Press, 2009:48-64.
5. Inzlicht M, McGregor I, Hirsh JB et al. Neural markers of religious conviction. Psychol Sci 2009;20:385-92.
6. Norenzayan A, Shariff AF. The origin and evolution of religious prosociality. Science 2008; 322:58-62.
7. Verhagen PJ, Cox JL. Multicultural education and training in religion and spirituality. In: Verhagen PJ, Van Praag HM, López-Ibor JJ, Jr. et al (eds). Religion and psychiatry. Beyond boundaries. New York: Wiley, 2010:587-613.

Religion and mental health: the contribution of anthropology

Simon L. Dein

Department of Mental Health Sciences, University College London, Charles Bell House, London WC1 7EY, UK

Pargament and Lomax provide a comprehensive overview of the rapidly growing literature on religion and mental health. Their account is balanced, describing the positive and negative impacts of religion and spirituality on wellbeing. In this commentary, I focus on some areas where my approach, which derives from social anthropology, may differ from Pargament and Lomax, with the intention of furthering discussion about future research on religion and mental health.

Several of the areas mentioned in the review (e.g., meaning, violence, and conflict) have attracted considerable anthropological attention. As an anthropologist, I agree that the findings cited need to be extended to other religious groups, cultures, and countries. Religious factors always operate in cultural frameworks. For example, the lived experience of Islam may differ in communities with different cultures. Gender hierarchy and political organization may differ in two Islamic societies, contributing to the experience of stressors. This has both theoretical and clinical implications.

Anthropology has much to contribute to this debate, with its emphasis on in-depth descriptions of individual experiences, and its key methodology – participant observation – which allows for long-term engagement with a cultural group and facilitates understanding of how religion and mental health impact on each other over time (1). Furthermore, it permits emic understanding of the ways that concepts such as religion, spirituality, coping, belief, and mental illness are culturally constructed. Future work on religion and mental health needs to take stock of lay meanings of terms such as religion, spirituality, and health rather than imposing professional definitions. Data from anthropological fieldwork can be deployed in rendering existing measurement scales culturally sensitive.

The extant literature on religion and mental health has predominantly focused on belief rather than experience. Anthropologists have generally taken issue with the notion of belief, especially when discussing religious convictions (2). It is debatable whether the notion of belief used in Protestant Christianity can easily be applied to other faith groups. And the relations between belief, knowledge, and faith are often vaguely defined in the current literature.

Experience comprises both cognitive and affective elements. The cognitive emphasis to date has ignored the important role of emotional factors in maintaining belief. R. Firth (3) conceptualized religious belief as a set of ideas more or less integrated by reason but held with conviction that they are true – that they are meaningful in relation to reality. He points out that we can distinguish elements of knowledge, emotion, and volitional activity acting on belief. It is the element of emotion in whatever kind of experience that gives a basis to belief and provides it with a strong flavour of reality.

While I would accord with Pargament and Lomax's (who themselves quote the anthropologist C. Geertz) assertion that religion provides a source of meaning for believers, social anthropology moves beyond individual beliefs and provides a broader perspective through looking at the ways in which power structures and religious hierarchies engender specific convictions and the ways that these convictions are contested by different factions in communities.

Anthropologists have long recognized the importance of specific religious experiences in maintaining wellbeing in communities and the role of rituals in engendering such experiences. There is wealth of anthropological data examining the role of rituals in engendering mental health. Yet the existing scholarly work has not examined the mental health implications of specific rituals. For instance, despite the fact that much of the contemporary literature has focused on Christianity, there is a dearth of work examining Christian rituals such as Baptism, the Eucharist, and laying on of hands in terms of facilitating wellbeing. Religious experience and rituals and their impact on wellbeing should be a future target of research in this area (4).

Furthermore, long-term immersion in a religious community facilitates the collection of detailed phenomenological descriptions of religious experiences, such as hearing God's voice, and allows an understanding of how they are interpreted in diverse cultural contexts (5). It allows to better understand the similarities and differences between normative religious experience and psychopathologic states.

One area that Pargament and Lomax discuss – religious hallucinations – has attracted some anthropological discussion. Anthropologist T. Luhrmann (6) points out that hallucinations are a vivid illustration of the way culture affects our most fundamental mental experience and the way that mind is shaped by both cultural invitation and biologic constraint.

The anthropological literature has indicated that conceptualizations of wellbeing include physical, emotional and spiritual dimensions, and similarly healing often reflects the reestablishment of a harmonious relationship with whatever communities label as the transcendent through diverse rituals. As the social anthropologist V. Turner points out in his work among the Ndembu (7), healing does as much to repair the social fabric as it does to repair the body, mind, and soul of sufferers. Thus, the impact of religion/spirituality on mental health should encompass measures of social harmony and social integration.

Finally, in terms of treatment, there is by now a growing literature on cross-cultural psychotherapy and the benefits of matching patient and therapist (8). This literature demonstrates that, while sometimes cultural matching enhances psychotherapeutic efficacy, other factors such as gender and social class are equally important in determining outcomes. These findings could be extended to research on spiritually integrated therapy, looking at the ways in which religious matching may enhance or diminish outcomes.

References

1. Dein S, Cook CC, Koenig H. Religion, spirituality, and mental health: Current controversies and future directions. J Nerv Ment Dis 2012;200:852-5.
2. Tambiah S. Magic, science, religion and the scope of rationality. New York: Cambridge University Press, 1990.
3. Firth R. Religion: a humanist interpretation. London: Routledge, 1996.
4. Dein S. Judeo-Christian religious experience and psychopathology: the legacy of William James. Transcult Psychiatry 2010;47:523-47.
5. Dein S, Littlewood R. The voice of God. Anthropol Med 2007;14:213-28.
6. Luhrmann T. Hallucinations and sensory overrides. Ann Rev Anthropol 2011;40: 71-85.
7. Turner V. The forest of symbols: aspects of Ndembu ritual. Ithaca: Cornell University Press, 1967.
8. Kareem J, Littlewood R. Intercultural therapy: themes, interpretations and practice. Oxford: Blackwell, 1992.

Spiritual assessment as part of routine psychiatric evaluation: problems and implications

PHILIPPE HUGUELET[1],
OLFA MANDHOUJ[2]

[1]Division of General Psychiatry, Department of Mental Health and Psychiatry, University Hospitals of Geneva, 1207 Geneva, Switzerland; [2]INSERM U699, Hôpital André-Mignot, Le Chesnay, Paris, France

Pargament and Lomax provide a comprehensive and clear review of the way religion may impact on symptoms and outcome in patients with mental disorders, both in its positive and negative aspects. Their work is predominantly based on the important research conducted by Pargament and colleagues on religious and "meaning-making" coping. They propose that many patients use religion both as a coping resource and as an explanatory model for their

disorder (1). This can have positive consequences (e.g., "I think my illness is God's punishment for my sins. It gives meaning to what happened to me, so it is less unjust") or negative ones (e.g., "I have been praying for years. I am still ill because I don't believe in God's healing power enough. I am a bad person") (2).

Negative aspects of religion can be expressed through "religious struggle", a complex concept for many clinicians. Spiritual struggle can be intrapersonal, interpersonal, and/or related to the patient's representation of God. The fact that religious struggle may be the cause or the consequence of psychopathology (or both) has to be considered while assessing this phenomenon.

Pargament and Lomax's paper should help clinicians to disentangle the various ways in which religion can be used: as a resource for coping with the illness, as a source of emotional comfort (e.g., to relieve anxiety) or meaning, and as a means of self-regulation and better social functioning. Clinicians should possess some familiarity with these concepts before performing spiritual assessment, which should be part of routine psychiatric evaluation (3). However, they will discover that more than one of the above aspects are often present in any given patient at a given time (3). For instance, a patient with schizophrenia may produce delusions with religious content ("The devil speaks to me . . .") while at the same time being capable to relieve his/her anxiety by praying to God. It is important to be aware that screening questions can be used in a thorough assessment of all aspects of a patient's religiosity (3,4).

After the spiritual assessment has been completed, how should we then proceed? The issues that arise should be addressed first of all as part of supportive psychotherapy, by fostering positive coping and dealing with its negative forms (e.g., spiritual struggle). In some cases, however, issues may become more complex, depending on the cultural context in which treatment is provided. In some areas where there are no psychiatrists, traditional healers will try to help patients with mental disorders in the context of "integrated care" involving both spiritual healing and more secular techniques (with more or less success). Conversely, in most European countries, psychiatrists work from a layman's position. Hence, issues arising from spiritual assessment may be considered as pertaining to chaplains and/or religious representatives. Half-way between these two positions, there are places where matters are intertwined. In the United States, for example, some clinicians may pray with their patients, a position which would not be considered as acceptable in most public psychiatric facilities in Europe.

The complexity of this field is also related to the existence of a "gray zone", sometimes grounded in the domain of psychotherapy, where some intrapsychic material may be considered either as "theological" or as a cognitive process warranting psychological intervention or as both of the above (for instance, the guilt a woman feels following abortion, which is partially grounded in religious considerations).

So what should we do? On the one hand, psychiatrists should adapt their practice to what society asks of them. On the other hand, there are contexts in which "local care" is inconsistent with effective conventional treatments (such as antipsychotics for psychotic symptoms). In some areas, patients first consult their local practitioner and then turn to more traditional medicine when they figure out that the former does not bring improvement (5). In these situations, clinicians should better not compete with these approaches but find ways of applying different models in a complementary fashion (6). This may be frustrating when considering the time – and money – that is likely to be lost in this sequence of steps. However, there is actually no other choice: people embedded in a culture involving religious explanatory models for their disorders will frequently look for traditional care before trying more conventional psychiatry. The role of psychiatrists is to take the cultural context of their patients into account, which may involve building a collaborative relationship with members of religious communities when necessary.

In conclusion, we would like to emphasize that: a) spiritual assessment should be part of the investigation of the cultural context of any patient consulting in a psychiatric facility, according to WPA recommendations (7); b) spiritual assessment is likely to reveal psychological and social issues that are relevant to treatment; and c) these issues may have psychotherapeutic, psychiatric, and social implications, depending on the patient's characteristics and the local psychiatric and cultural context.

References

1. McCabe R, Priebe S. Explanatory models of illness in schizophrenia: comparison of four ethnic groups. Br J Psychiatry 2004;185:25-30.
2. Huguelet P, Mohr S, Gillieron C et al. Religious explanatory models in patients with psychosis: a 3-year follow-up study. Psychopathology 2010; 43:230-9.
3. Huguelet P, Mohr S, Betrisey C et al. A randomized trial of spiritual assessment of outpatients with schizophrenia: patients' and clinicians' experience. Psychiatr Serv 2011;62:79-86.
4. Anandarajah G, Hight E. Spirituality and medical practice: using the HOPE questions as a practical tool for spiritual assessment. Am Fam Physician 2001;63: 81-9.
5. Campion J, Bhugra D. Experience of religious healing in psychiatric patients in South India. Soc Psychiatry Psychiatr Epidemiol 1997;32:215-21.
6. Khan I, Pillay K. User' attitudes toward home and hospital treatment: a comparative study between South Asian and white residents of the British Isle. J Psychiatr Ment Health Nurs 2003;10:137-46.
7. Mezzich E. Psychiatry for the Person: articulating medicine's science and humanism. World Psychiatry 2007;6: 65-7.

Religion and health: the more we know the more we need to know

ALEXANDER MOREIRA-ALMEIDA

Research Center in Spirituality and Health, School of Medicine, Universidade Federal de Juiz de Fora, Brazil; WPA Section on Religion, Spirituality and Psychiatry

In the last few decades, largely due to a growing body of robust empirical evidence, there has been an increasing recognition of the interconnections between religiousness and health. These data have challenged previous views, mostly based on theoretical perspectives, that religiousness is a vestige of a primitive psychological development, usually associated with immature defenses and psychiatric symptoms. Most data from epidemiologic studies indicate that religious involvement is associated with better health outcomes at a population level (1). However, there has been little discussion on the implications these findings might have for routine clinical practice, and little research on the negative side of the connection. Pargament and Lomax's paper highlights these two shortcomings.

The authors mention the "troubled history between psychiatry and religion". Undoubtedly, there have been conflicts between religion and psychiatry, and more generally between religion and science. However, it is important to be aware that, from a wider historical perspective, the relationship between science and religion has been more of synergy than antagonism. It seems that the conflict was predominantly confined to the period from mid-19th century to the end of the 20th century (2). Indeed, from ancient times spiritual issues and care for the sick have been interconnected, and it would be worthwhile to conduct new and in-depth studies of the historical connections between spirituality and the care of mentally disturbed people. This historical understanding would be very important to put recent developments in perspective.

While the interaction between religiousness and health has been well established, the understanding of its mechanisms has been a challenge. The recognition of the "active ingredients" of religiousness is of special interest for the clinician, since they may help in the development of new treatment and preventive approaches. A review of four proposed psychosocial mediators (health practices, social support, psychosocial resources, and belief structures) has found that the empirical evidence is "mixed and inconsistent" (3). Pargament and Lomax discuss some promising mechanisms. In order to advance knowledge on this issue, it could be fruitful to investigate how spirituality may promote the development of "salutogenic" factors more than the decrease of "pathogenic" factors. It might also be useful to focus on the mechanisms that may be specific to spirituality, not being present in secular contexts. Qualitative studies are an important but still largely neglected source of new hypotheses for mediators.

The understanding of religion's dark side needs much more investigation. This applies in particular to religious violence. It seems that religion, more than a cause of violence between groups, may often be a marker, a proxy for a wider network of sociocultural characteristics. Several conflicts that have been hastily presented as religious might be better understood as having economic, ethnic, or other more mundane sources (4). Religion, as any other powerful idea like science, health, social justice, and freedom, may be and has been used as a justification for violence and intolerance.

In addition to the important issue of when a religious problem is primary or secondary to a mental disorder, it is also imperative to improve the differentiation between healthy spiritual experiences that resemble psychiatric symptoms and mental disorders with religious content (5). This is an essential but still largely unexplored area.

Further to the exploration of spiritually integrated treatment, as emphasized by the authors, a better understanding of the mechanism and impact of spiritual treatments is needed. Millions of people suffering from mental disorders around the globe seek help from spiritual treatments alone or in conjunction with psychiatric treatments (6). One important example that deserves further in-depth investigation is religious/spiritual treatment for substance abuse. This kind of investigation may help us to clarify which practices are harmful (and should be avoided) and which may be effective (and should be better studied and used).

In addition to the studies on spiritually integrated treatment showing better outcomes compared to regular treatments, reviewed by Pargament and Lomax, there are other investigations that found a similar level of efficacy of the two treatments (7). Nevertheless, spiritually integrated treatment may foster a better acceptance of psychological/psychiatric treatments among religious patients. Some specific profiles of patients may especially benefit from these integrated approaches. It is also worth noting that, according to some evidence, spiritually integrated treatment may be adequately delivered by nonreligious clinicians (8).

In summary, Pargament and Lomax's review has the merit of bringing to the attention of a wide psychiatric audience some important aspects of religion that are relevant to clinical practice. It is possible to reach some conclusions on the basis of the available evidence: a) there is a frequent and significant connection between

religiousness and mental health; b) this relationship is usually positive, but there are also harmful ties; and c) there is a need of taking into consideration patients' spirituality in order to provide an adequate and culturally competent clinical care.

On the basis of this, the psychiatric community has some educational and research challenges. From the educational viewpoint, it is appropriate to make clinicians aware of the body of knowledge available in the field. From a research perspective, there is a need to: a) expand studies to a more diverse geographical and cultural base; b) conduct more studies in psychiatric populations; c) explore the impact of spiritual and spiritually integrated treatments; d) investigate the mechanisms through which religious involvement and spiritual-related treatments may affect health; e) study spiritual experiences, their roots and differentiation from mental disorders; and f) develop clinical applications of the currently available epidemiologic data about the interconnection between religion and health.

In other to address these challenges, the WPA has a Section on Religion, Spirituality and Psychiatry, that includes and welcomes members from any religious/philosophical background from all over the world (www.religionandpsychiatry.com). This section has promoted several initiatives such as symposia in international congresses, published the newsletter *Psyche and Spirit: connecting psychiatry and spirituality*, and edited a comprehensive book (9).

References

1. Koenig HG, King DE, Carson VB. Handbook of religion and health, 2nd ed. New York: Oxford University Press, 2012.
2. Numbers RL. Galileo goes to jail and other myths about science and religion. Cambridge: Harvard University Press, 2009.
3. George LK, Ellison CG, Larson DB. Explaining the relationships between religious involvement and health. Psychological Inquiry 2002; 13:190-200.
4. Cavanaugh WT. The myth of religious violence: secular ideology and the roots of modern conflict. New York: Oxford University Press, 2009.
5. Moreira-Almeida A, Cardena E. Differential diagnosis between non-pathological psychotic and spiritual experiences and mental disorders: a contribution from Latin American studies to the ICD-11. Rev Bras Psiquiatr 2011;33 (Suppl. 1): S21-36.
6. Schumann C, Stroppa A, Moreira-Almeida A. The contribution of faith-based health organisations to public health. International Psychiatry 2011; 8: 62-3.
7. Worthington EL, Hook JN, Davis DE et al. Religion and spirituality. In: Norcross JC (ed). Psychotherapy relationships that work, 2nd ed. New York: Oxford University Press, 2011:402-19.
8. Post BC, Wade NG. Religion and spirituality in psychotherapy: a practice-friendly review of research. J Clin Psychol: In Session 2009; 65:131-46.
9. Verhagen PJ, van Praag HM, López-Ibor JJ et al (eds). Religion and psychiatry. Beyond boundaries. Chichester: Wiley-Blackwell, 2010.

Religion and psychiatry: from conflict to consensus

MARILYN BAETZ

Department of Psychiatry, University of Saskatchewan, Saskatoon, SK, S7N 0W8, Canada

Pargament and Lomax present religion as a "double-edged sword", serving both as a resource and a challenge to psychiatry. This analogy may be useful in guiding further research and moving to a position of consensus a field that has been sometimes conflict-laden. Not only psychiatry has had difficulty with religion, but also religion – with its focus on mind, body and spirit – has had difficulty with psychiatry. Optimal restoration of mental health in a patient requires an ability by the psychiatrist to assemble evidence for treatment at levels ranging from cells to communities. This evidence is best informed by credible research.

Factors to be considered in research aimed at understanding religion as a resource or challenge to psychiatric treatment include the following:

- *Operationalization of the definition of spirituality*. Typical measures of spirituality tend to use "common denominators", such as "I feel a connection to all life" (1), which may not accurately capture its essence. Such measures have informed research to date, but have been "impure", in that they measure psychological wellbeing and existential or social constructs as much as they measure spirituality (2). Accordingly, conclusions based on these studies may be misleading. Furthermore, spirituality is generally measured by self-report assessment, a process that requires a level of insight in the respondents to assess some aspects about themselves against an unknown standard. Attempts to find objective validators, such as behaviors, related to spirituality are much more challenging. Here, the risk is in measuring the outcome of a spiritual life (i.e., peace or joy) rather than the spiritual essence itself, which leads to faulty conclusions.

- *Measuring religiosity in a multifaith context*. Each religion claims exclusivity in certain domains. Thus, using all-encompassing or generic measures while ignoring the individual tenets of specific religions serves

only to compromise the measure. It would be useful to develop questions that explore what is considered core in each religion, particularly as it relates to mental health. Religious beliefs, attitudes, experiences, commitment, and maturity may be common themes, but each religion and even denominations within the same religion will have different sets of "standards" or ideals for each of these. For example, Judaism tends to emphasize God as all powerful, while Catholicism tends to emphasize God as all merciful. Different conceptions of God are associated with adaptive or maladaptive ways of religious coping (3).

- *Elucidating the role of culture in religious expression*. Culture may inform the way religion and spirituality are interpreted by the individual and subsequently how questions are answered. For instance, in some countries "religion" is seen as outdated and almost a pejorative term, while in others "spirituality" is seen as a mystic new age phenomenon. Studies conducted among African Americans or in the Southern United States have a completely different cultural overlay than in other areas of the United States, Canada, or Europe. In the former, there are much higher baseline rates of stated religious beliefs, and different measures may be required to actually determine the integration of religion or spirituality by the individual as opposed to the prevailing culture. As an example, religious service attendance may reflect internalized religious commitment, or compliance with social norms, or a combination of both.

- *Longitudinal studies*. Longitudinal studies have been advocated during the last decade, as a plethora of cross-sectional data was emerging. Cross-sectional data have been inconsistent, often showing different relationships between depression and religion, or depression and spiritual self-assessment. Causality cannot be determined, and the issue of whether psychiatric illness causes one to turn to or away from spirituality is left in question. Good longitudinal data on the relationship of depression and religious observance or commitment have begun to address both how depression may impact religiosity and how religiosity may impact depression (2). Longitudinal studies on anxiety disorders, affect dysregulation, exacerbation or reduction of guilt or shame, or serious chronic mental illness would also be enlightening. Cross-sectional epidemiologic studies have shown, anyway, that religion and spirituality are significant determinants of mental health to a similar extent as gender or income. This indicates that religion should be a variable considered in psychiatric epidemiological studies. There is also solid evidence (4) suggesting that religion is not just another measure of social support.

- *Research on which aspects of religion or spirituality are sustained during a psychiatric episode or how they might be supported*. A shift is required from treating mental disorder as an end goal to a patient-centered perspective in which the aim is spiritual and psychological growth. Consistent with this, Kohlberg (5) and Piaget (6) envision moral development as a sequence of stages starting with the self as the "be-all and end-all" to universal ethical principles.

Religion is unique in that it provides a link to the past and to the future. In a society where governments focus on short-term expediency, many things are thrown away, families are volatile, institutions are unstable, and cultures are less "pure" because of migration, religion clearly provides a longitudinal perspective. To understand psychiatric disorders, a longitudinal perspective, including consideration of religion and spirituality, is also needed. In those individuals where religion and mental health problems intertwine, the better the understanding psychiatrists have of potentials and pitfalls around their patients' religious or spiritual beliefs (or loss of them), the more able they will be to help restore balanced mental health.

References

1. Underwood L, Teresi J. The Daily Spiritual Experience Scale: development, theoretical description, reliability, exploratory factor analysis, and preliminary construct validity using health-related data. Ann Behav Med 2002;24:22-33.
2. Koenig HG, King DE, Carson VB. Handbook of religion and health. Oxford: Oxford University Press, 2012.
3. Phillips RE, Stein CH. God's will, God's punishment, or God's limitations? Religious coping strategies reported by young adults living with serious mental illness. J Clin Psychol 2007;63:529-40.
4. Krause N. Exploring the stress-buffering effects of church-based and secular social support on self-rated health in late life. J Gerontol B Psychol Sci Soc Sci 2006; 61:S35-43.
5. Kohlberg L. Essays on moral development. San Francisco: Harper & Row, 1981.
6. Piaget J. The moral judgment of the child. New York: Free Press, 1965.

Are we doing harm by omission? Addressing religiosity of the mentally ill

Samuel George Hansdak[1],
Raja Paulraj[2]

[1]Department of Medicine, Christian Medical College, Vellore, Tamil Nadu, India; [2]Herbertpur Christian Hospital of Emmanuel Hospital Association, Dehradun, Uttarakhand, India

In ancient days, the main hindrance in treating the mentally ill was prejudice related to the therapist's own religious views. Religious leaders used exorcisms to treat mental disorders, often through cruel, harsh, and barbarous methods. They believed demon possession was the main cause of mental illness. In modern days, we are experiencing a problem on the other side of the spectrum. In order not to impose religious beliefs on the patient, therapists are not addressing the issue of religiosity at all in the management of mental disorders. This is harmful as well, since there is often a spiritual component involved. We, as spiritually concerned medical workers, should attempt to reach a balance.

The role of religion may be perceived differently by psychiatrists and their patients (1). Psychiatrists are often less religious than their patients and may not appreciate the value of religion in helping patients cope with their illness. Furthermore, psychiatrists may experience religion through the pathological expressions of individuals with religious delusions, which may bias them against religion as a therapeutic resource. Third, psychiatrists may focus on the biologic components of mental illness and may view the religious component as subjective and not supported by empirical evidence. Finally, psychiatrists may believe that religion always causes dependence and guilt (2).

The possible negative effects of religion on mental health, or our personal past experiences with religion, should not prevent us from acknowledging a spiritual component in mental illness and at least offering resources for, if not personally suggesting, spiritual help. This includes collecting a spiritual history, supporting healthy religious beliefs, challenging unhealthy beliefs, praying with patients (in highly selected cases), and consultation with, referral to, or joint therapy with trained clergy (3).

Globally, we can see a vast difference between therapies in the East and the West, due to different overriding spiritual beliefs. In the West, monotheism and the heavy influence of individualism have produced much resistance to acknowledging spirituality in mental illness. In the East, perspectives are very different, due to wide acceptance of polytheism and firmly held beliefs that the causes of distress and disorders may in fact be spiritual in nature. This, of course, produces less resistance.

Pargament and Lomax rightly emphasize the need for further studies beyond the Western perspective. In some Eastern countries, spirituality and religion are part of daily life, and religiousness cannot be ignored in the therapy setting. As almost every physical ailment is associated with some religious beliefs, or lack thereof, treating a patient without addressing religiosity would be considered incomplete treatment.

In conclusion, when attempting to understand religion, and its influence, in the mentally ill, we need to ask some questions. Are we doing justice to our clients if we are not addressing the religious influence in their clinical presentation? Can we train ourselves to seriously confront our "take it easy" attitude toward spiritual influence in the mentally ill? Psychiatry and religion are the unfortunate enemies of yesterday and forgotten friends of today. How can we, in the mental health field, find a way to bridge the gap and acknowledge that they actually work in parallel with one another?

References

1. Curlin FA, Odell SV, Lawrence RE et al. The relationship between psychiatry and religion among US physicians. Psychiatr Serv 2007;58:1193-8.
2. Neeleman J, King MB. Psychiatrists' religious attitudes in relation to their clinical practice: a survey of 231 psychiatrists. Acta Psychiatr Scand 1993;88:420-4.
3. Koenig HG. Religion and mental health: what should psychiatrists do? Psychiatr Bull 2008;32:201-3.

Religion and mental health: a double-edged sword or a life-giving medicine?

David Greenberg

Herzog Hospital and Hebrew University, Jerusalem, Israel

Pargament and Lomax present religion as a double-edged sword: on the one side, succor, self-regulation, social support, meaning, and spirituality; on the other side, struggles and violence. In this commentary, I would

like to discuss the meaning of such a perception for religionists and the implications for mental health services.

The attitude of religious codes to the impact of the religious life is expressed clearly in the following quotation from the Babylonian Talmud, a central text of orthodox Judaism: "The Rabbis teach: 'It is written, 'And you shall put the words of my *Torah* in your hearts' (Deuteronomy 11:18) and the word 'you shall put' can also mean 'a perfect medicine', for the *Torah* can be compared to a life-giving medicine' ".

The effect of religion is perceived to be entirely positive. Is this an absence of insight? Would members of a religion find the observations of Pargament and Lomax offensive? How would they respond to the specific claims of the effects of struggles and the occurrence of religiously motivated violence?

During the last three decades of providing mental health services to the ultra-orthodox Jewish community of Jerusalem, there have been many opportunities to consider: is religion a double-edged sword or a life-giving medicine? Evaluating the religious background of new referrals, we found an overrepresentation of newly orthodox Jews, and concluded initially that their religious change had precipitated their disturbance. On reevaluating the data, however, the majority had a history of mental health problems prior to their religious change, the change brought several years of relief, and then the earlier problems reemerged (1).

Our attention then focused on obsessive-compulsive disorder (OCD). Is OCD more common in this population, encouraged by the demand and praise accorded the punctilious? Epidemiological studies of OCD in a range of cultures have not measured degree of religiosity, although urban versus rural studies did not suggest OCD was more prevalent in the rural, more traditional societies that were likely to be more religious.

Studying ultra-orthodox patients with OCD, we noted that the content of the religious OCD symptoms was similar to nonreligious populations. Further, despite the centrality of religious observance in their lives, most had religious and nonreligious symptoms (2). When asked if they saw a link between their OCD and their religion, their responses covered the whole gamut of possibilities, from blaming their religious practice, blaming their educators, to understanding it was their own problem ("I realize this is *my* problem, as it says: 'The *Torah* was not given to angels', the *Torah* is for fallible humans") and that if they were not religious, it would just appear in a different form. The overall impression is that the religious form of OCD ("scrupulosity") is not caused by religion, but assumes a religious form reflecting the content and values of the religious lives of the sufferers (3).

The ideal method for approaching these questions is via large-scale epidemiological studies. Unfortunately, the reticence of the ultra-orthodox community to participate in secular projects and consequent high refusal rate led the organizers of a recent study in Israel to exclude this community *ab initio* (4).

Religion is based on belief, and beliefs, by definition, have no objective proof. Doubts in matters of religious belief are a normal component of adolescence and early adulthood, which is the period in life when people are most likely to undergo religious change, either by abandoning or increasing their religious observance (5). For a religious person, religious doubts create anxiety. As adolescents become adults, their cognitive processes mature, and they are likely to learn to tolerate the ambivalence of doubt. As such, doubt is normative. As with all normative processes, it has a range of expressions and degrees. Similarly, the emotional response to doubt is varied and will be influenced by serious life events, such as severe mental illness, in which the sufferer may either find succor in religion, or ask "why me?" and "why did He create a world with such problems?" that will inevitably lead to doubts (6). It is unclear, however, why the blame for the human capacity for doubt is to be placed at religion's door.

Religiously motivated violence is problematic in the present context. The world news brings daily proof that it exists, rarely the act of individuals but of organizations and countries that are motivated by religious beliefs. As the largest scale murders of the last century have shown, however, this is not the prerogative of religion alone. As organized and ideologically based acts, it is an unfortunate truth that there is no role for mental health services for the individual in their prevention.

References

1. Witztum E, Greenberg D, Dasberg H. Mental illness and religious change. Br J Med Psychol 1990; 63:33-41.
2. Greenberg D, Shefler G. Obsessive compulsive disorder in ultra-orthodox Jewish patients: a comparison of religious and non-religious symptoms. Psychol Psychother 2002;75:123-30.
3. Greenberg D, Huppert JD. Scrupulosity: a unique subtype of obsessive-compulsive disorder. Curr Psychiatry Rep 2010;12: 282-9.
4. Levinson D, Paltiel A, Nir M et al. The Israel National Health Survey: issues and methods. Isr J Psychiatry Relat Sci 2007;44:85-93.
5. Ozorak EW. Social and cognitive influences on the development of religious beliefs and commitment in adolescence. J Sci Study Relig 1989;28:448-63.
6. Webb M, Charbonneau AM, McCann RA et al. Struggling and enduring with God, religious support, and recovery from severe mental illness. J Clin Psychol 2011;67:1161-76.

Disentangling complexities in the relationship between religion and psychiatry

JUAN J. LÓPEZ-IBOR JR[1-5],
MARÍA INÉS LÓPEZ-IBOR[2,3,5],
SARA GONZÁLEZ-VIVES[1,3,4],
JULIA GARCÍA-ALBEA[1,3,4]

[1]Instituto de Psiquiatría y Salud Mental, Hospital Clínico San Carlos, Madrid; [2]Departamento de Psiquiatría, Facultad de Medicina, Universidad Complutense, Madrid; [3]CIBERSAM, Madrid; [4]Instituto de Investigación Sanitaria, Hospital Clínico San Carlos, Madrid; [5]Fundación Juan José López-Ibor, Madrid, Spain

One of the consequences of modernity has been the separation between science and religion. This split has had positive consequences, such as the progress of scientific research and the separation of Church and State in developed societies. On the other hand, religion and science have started to ignore or despise each other, and this has had an impact especially on research and practice in psychiatry as this discipline deals with the most "spiritual" of the diseases, the *Geisteskrankheiten* (literally, "diseases of the spirit-mind"). Religiosity has often been regarded either as a manifestation of mental disorder or as irrelevant to clinical practice.

Pargament and Lomax's article is a precise and comprehensive summary of the new perspectives on the relationship between religion and mental illness. We want to highlight here two important aspects.

The first has implications for everyday practice. According to Pargament and Lomax, religion may simply be the idiom through which mental illness is expressed. Indeed, delusions *per se* are not just a by-product of a brain disease, but the expression of an attempt to cope meaningfully with incomprehensible experiences. Many years ago, Sarró (1) studied in depth the themes of delusions and came to the conclusion that they correspond to the mythologems described by anthropologists in ancient myths and religions. In a similar way, Schneider (2) regarded the delusions of severe depressive states as the expression of the primeval fears of human beings: the fears of not going to be able to survive, to fall ill, or to be condemned for eternity. Considering the delusional manifestations of abnormal states of consciousness, we came to the same conclusion (3): the themes coincide with the everlasting preoccupations of human beings (where do we come from, is there a new life after death, why two sexes, and so on). What is morbid is not the preoccupation, the theme of the delusion, but the fact that it emerges untimely and out of context. In this light, the relatively common presence of religious contents in delusions should not appear surprising.

The second aspect is a more general one and concerns the role of religion in the establishment of individual and collective identities and the dangers involved in this process, including religiously based violence and religious struggles within oneself, with others, and with the divine. Recently, we have considered (4) the origin of identification from an anthropologic perspective. According to Lovaglia et al (5), human evolution is the consequence of the pressures precipitated by xenophobia among primates in the past several millions of years. This xenophobia is manifested in deadly raids against individuals of other groups of the same primate kind. The immediate consequences are both intergroup conflicts and intragroup cooperation. According to the theory of social identity (6), the sense of self depends on the identification with a group. Once the identification has been produced, the individuals attribute pleasant and desirable characteristics to the group members and repellent and unpleasant characteristics to individuals outside the group. Religion may be involved in this process but just as one of the many possible elements. The xenophobia may even be oriented toward the body and the evils that come with it: for instance, anorexia nervosa can be interpreted as an identity disorder grounded on the wish to dominate and subdue the body (ascetism) and, therefore, as a sacred disease.

Pargament and Lomax's article should be welcome as a very significant contribution to the clarification of the complex relationship between religion and mental illness. Other initiatives worth mentioning are those of the WPA Section on Religion, Spirituality, and Psychiatry (www.religionandpsychiatry.com), the WPA publication *Psychiatry and Religion: Beyond Boundaries* (7), and the *Handbook of Religion and Health* (8). A growing interest on the religious side in topics concerning psychiatry should also be noticed. The López Ibor chair on Mystical Studies and Mental Health founded by the Order of the Carmelites in Ávila (Spain) is a good example (www.citesavila.org; www.fundacion lopezibor.es).

References

1. Mediavilla Sánchez JL. Mito y delirio. Cartas de Ramón Sarró. Oviedo: KRK, 2001.
2. Schneider K. Clinical psychopathology. New York: Grune and Stratton, 1959.
3. García Albea J, González Vives S, Tejeira C et al. Psicosis inducidas por estados excepcionales de conciencia. Actas Esp Psiquiatr (in press).
4. López-Ibor JJ, Ortiz T, López-Ibor MI. Perception, experience and body identity. Actas Esp Psiquiatr 2011;39(Suppl. 3):113-8.
5. Lovaglia MJ, Barron C, Houser JA. Social development and human evolution: managing the in-group boundary. Theory Workshop 2003;31.
6. Hogg MA, Terry DJ, White KM. A tale of two theories: a critical comparison of identity theory with social identity theory. Soc Psychol Q 1995;58:255-69.
7. Verhagen PJ, van Praag HM, López-Ibor JJ et al (eds). Psychiatry and religion: beyond boundaries. Oxford: Wiley-Blackwell, 2010.
8. Koenig HG, King DE, Carson VB (eds). Handbook of religion and health. New York: Oxford University Press, 2012.

A "complex" subject matter asks for a clear lead

PETER J. VERHAGEN

GGZ Centraal, Harderwijk, the Netherlands; WPA Section on Religion, Spirituality and Psychiatry

Pargament and Lomax did the readers of *World Psychiatry* and the WPA community a splendid service with their comprehensive and well-ordered paper. They managed to clear the way for proper discussion and innovative study and action toward training, continuing medical education, and clinical practice. They clearly are not out to provoke or to tread on someone's corns. Their review is well-balanced, without finery, and therefore can and should be read closely. And indeed, the empirical data are self-evident.

Nevertheless, the subject matter is complex, as indicated with subtlety in the title of this Forum. Why is this the case? Are those data not as self-evident as I assume? Of course, as the authors explain briefly, the relationship between psychiatry and religion has a troubled history. However, the field is moving, they write, to "a more nuanced understanding of religion" with regard to the promising and damaging forces religious and spiritual beliefs can have.

It is often encouraging to be optimistic and to have positive expectations, as the authors seem to have, but I think the after effects of that troubled history remain a matter of concern. The issue of "psychiatry and religion" is still (and only?) emphasized by prominent scholars who are under suspicion of being strongly involved in faith or any spiritual tradition. In other words, it is supposed they have a conflict of interests, and therefore their contributions are essentially labeled as opinion based. Despite the evidence provided by research, the clinical relevance of data is still contested. Furthermore, the whole topic of "psychiatry and religion" is perceived as a threat to appropriate therapeutic boundaries in clinical practice (1,2).

Several colleagues do struggle with the double-sided face of religion. Hesitating as they are, they admit that religion and spirituality might be helpful for certain patients, but at the same time they tend to underscore the negative and harmful, guilt-inducing effects they have witnessed over the years.

Given this "complex" state of affairs, WPA could give a lead. According to the WPA Section on Religion, Spirituality and Psychiatry, it would be a major accomplishment if WPA would do so. In 2006, this WPA Section and the Psychiatry and Spirituality Special Interest Group of the UK Royal College of Psychiatrists started working on a statement that would delineate WPA's vision on "psychiatry and religion" in psychiatric practice, research, and training worldwide. A first version of this statement was published in a WPA volume (3). In the meantime, the Special Interest Group continued its work within the Royal College, and in August 2011 a position statement was approved (4).

Is the topic to be considered worthy of such attention? The WPA Section thinks it is according to WPA's own criteria. Pargament and Lomax's paper supports this view. In the first place, the topic is relevant to the further development of psychiatry around the world. The transformation of religion instead of its disappearance and the place spirituality occupies are significant to psychiatry. A renewed impulse is needed for empirical and conceptual research into the distinction between religious and spiritual experiences, on the one hand, and pathological phenomena on the other. Religiosity and spirituality can be corrupted, but cannot be regarded in themselves as morbid conditions (5). In addition, research is needed into the significance and effectiveness of religious and spiritual healing practices around the world. There are important differences in the way these practices are approached, interpreted, and evaluated, depending on cultural and subcultural contexts, values and sources. Second, the importance of the topic is supported by an overwhelming amount of empirical evidence, although mental health professionals, psychiatrists in particular, are often not aware of that evidence. Psychiatric training should be updated in order to get psychiatrists ready to readjust their attitude and to deepen their knowledge. Third, the topic is relevant to mental health as a political and public theme and is likely to get a high public visibility. Fourth, the topic is in line with the trend toward a more personalized clinical practice and the increasing attention to transcultural aspects of psychiatry. If it is true that the individual needs to be the focal point of clinical attention and has to be understood in his/her cultural context, then the dimension of religion and spirituality cannot be ignored. So, the absence of a statement on this issue may be detrimental to psychiatry and to psychiatric patients.

For all these reasons, the WPA Section on Religion, Spirituality and Psychiatry holds the view that the topic of psychiatry and religion concerns psychiatry worldwide and that consequently a statement deserves priority. We call upon WPA to take this lead.

References

1. Poole R, Higgo R. Spirituality and the threat to therapeutic boundaries in psychiatric practice. Mental Health Religion & Culture 2011;14:19-29.
2. Verhagen PJ. Controversy or consensus? Recommendations on psychiatry, religion and spirituality. Asian J Psychiatry 2012; 5:355-7.
3. Verhagen PJ, Cook CCH. Epilogue: Proposal for a World Psychiatric Association consensus or position statement on spirituality and religion in psychiatry. In: Verhagen PJ, Van Praag HM, López-Ibor JJ et al (eds). Religion and psychiatry. Beyond boundaries. Chichester: Wiley-Blackwell, 2010:615-31.
4. Royal College of Psychiatrists. Recommendations for psychiatrists on spirituality and religion. www.rcpsych.ac.uk.
5. Sims A. Is faith delusion? Psyche Geloof [Psyche Faith] 2011;22:64-71.

When does depression become a disorder? Using recurrence rates to evaluate the validity of proposed changes in major depression diagnostic thresholds

Jerome C. Wakefield[1-4], Mark F. Schmitz[3,5]

[1]Silver School of Social Work, New York University, 1 Washington Square North, New York, NY; [2]Department of Psychiatry, School of Medicine, New York University, New York, NY; [3]InSPIRES (Institute for Social and Psychiatric Initiatives–Research, Education and Services), New York University, New York, NY; [4]Division of Clinical Phenomenology, Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, NY; [5]School of Social Work, Temple University, Philadelphia, PA, USA

High community prevalence estimates of DSM-defined major depressive disorder (MDD) have led to proposals to raise MDD's diagnostic threshold to more validly distinguish pathology from normal-range distress. However, such proposals lack empirical validation. We used MDD recurrence rates in the longitudinal 2-wave Epidemiologic Catchment Area Study to test the predictive validity of three proposals to narrow MDD diagnosis: a) excluding "uncomplicated" episodes (i.e., episodes that last no longer than 2 months and do not include suicidal ideation, psychotic ideation, psychomotor retardation, or feelings of worthlessness); b) excluding mild episodes (i.e., episodes with only five to six symptoms); and c) excluding nonmelancholic episodes. For each proposal, we used lifetime MDD diagnoses at wave 1 to distinguish the group proposed for exclusion, other MDD, and those with no MDD history. We then compared these groups' 1-year MDD rates at wave 2. A proposal was considered strongly supported if at wave 2 the excluded group's MDD rate was not only significantly lower than the rate for other MDD but also not significantly greater than the no-MDD-history group. Results indicated that all three excluded groups had significantly lower recurrence rates than other MDD (uncomplicated vs. complicated, 3.4% vs. 14.6%; mild vs. severe, 9.6% vs. 20.7%; nonmelancholic vs. melancholic, 10.6% vs. 19.2%, respectively). However, only uncomplicated MDD's recurrence rate was also not significantly greater than the MDD occurrence rate for the no-MDD-history group (3.4% vs. 1.7%, respectively). This low recurrence rate resulted from an interaction between uncomplicated duration and symptom criteria. Multiple-episode uncomplicated MDD did not entail significantly elevated recurrence over single-episode cases (3.7% vs. 3.0%, respectively). Uncomplicated MDD's general-distress symptoms, transient duration, and lack of elevated recurrence suggest it may generally represent nonpathologic intense sadness that should be addressed in treatment guidelines and considered for exclusion from MDD diagnosis to increase the validity of the MDD/normal sadness boundary.

Key words: Major depression, recurrence, uncomplicated depression, diagnosis, validity, severity, melancholic depression

Establishing a valid diagnostic boundary between major depressive disorder (MDD) versus intense normal sadness or mild adjustment disorder that generally does not require intervention has proven challenging. The problem is that nonpathological reactions to major losses and stressors possess many of the same general-distress symptoms as depressive disorder (1,2).

The DSM's five-out-of-nine-symptoms-for-2-weeks descriptive MDD diagnostic criteria were originally formulated to distinguish depressive conditions from other mental disorders among severely ill populations for research purposes (3). No empirically grounded rationale exists for the current boundary in distinguishing between disorder and normal distress in community populations (4,5). Professional and public concerns about the validity of MDD criteria have been provoked by dramatically rising community prevalence estimates of DSM-specified MDD as the methodology of epidemiologic studies improves, from 2–3% some decades ago (6) to current estimates of half or more of all individuals meeting criteria over a lifetime (7-9). This diagnostic conundrum takes on additional importance at a time of increased depression screening in schools and physicians' offices, making the validity of diagnostic criteria relevant to everyone in the community, not just those presenting for treatment.

In light of these concerns, some depression researchers argue that the diagnostic threshold currently set by DSM criteria for MDD is too low, yielding false-positive diagnoses that misclassify considerable numbers of normally distressed individuals as having MDD (2,10-12). However, specific proposals for raising the MDD diagnostic threshold remain unsupported by persuasive empirical evidence regarding validity.

Crystallizing the need for a scientific approach to the MDD boundary question, Maj (13,14) recently challenged depression researchers to undertake the difficult task of establishing an empirically based answer to the question, When does depression become a mental disorder? We address Maj's challenge by empirically evaluating the predictive validity of three suggestions derived from the literature for narrowing the category of major depression: a) extend the DSM-IV's (15) "bereavement exclusion" to all MDD, thus excluding from MDD diagnosis all "uncomplicated" episodes (i.e., episodes lasting no longer than 2 months and lacking suicidal ideation, psychotic symptoms, psychomotor retardation, and feelings of worthlessness); b) exclude all mild episodes (i.e., episodes having only five or six symptoms) from MDD diagnosis; and c) equate MDD with melancholic depression, thus excluding from diagnosis all depression that is nonmelancholic.

To evaluate these proposals, we examined recurrence rate as a predictive validator of disorder. No single outcome variable can entirely resolve the question of where to place the diagnostic threshold between normal sadness and MDD to optimally prevent false positives while avoiding false negatives. However, substantially elevated recurrence rates are characteristic of MDD in both clinical and community samples (16-23), justifying the standard conceptualization of MDD as an episodic and chronic condition (24-26) for which prevention of recurrence is a major treatment goal (25,27-29). Moreover, recurrence is often interpreted as the prime evidence allowing one to infer that there exists a latent dysfunction that repeatedly generates the MDD symptoms, thus verifying the presence of a disorder rather than a normal emotional response (16,30).

In this study, we examine two progressively stronger indicators that a group's recurrence diverges from standard MDD. First, we examine whether subgroups of MDD proposed for exclusion have lower recurrence rates than other MDD, suggesting a difference in kind of condition worthy of further investigation. Second – and much more stringently – we examine whether the groups have rates that are not significantly elevated over the occurrence of MDD in the general population, calling into question the presence of pathology. Many variables are known to predict differentially lower MDD recurrence rates (16-18,20). However, no study has identified a substantial subset of MDD that does not have an elevated risk for recurrence above general population risk levels, with the one limited exception of uncomplicated bereavement-related depression (23,31). The lack of elevated recurrence runs contrary to the standard characterization of MDD as a highly recurrent condition in which the end of a depressive episode means "recovery from the episode, not from the illness per se" (30). Quite aside from diagnostic boundary issues, variables indicating lack of elevated recurrence rates or substantially lower recurrence rates would be of potential importance to clinicians in judging prognosis, evaluating the risk/reward ratio of treatments having potential negative side effects, and deciding about the timing of treatment termination.

METHODS

Sample

To evaluate the proposals for revising the MDD threshold, we used longitudinal community data from the 2-wave Epidemiologic Catchment Area Study (ECA, 32). We selected the ECA dataset for this analysis because it is the only currently publicly available nationally representative American longitudinal dataset with the information needed to perform the study's analyses.

The ECA's sampling and data collection are described extensively in other publications (32). Briefly, respond-

ents (ages 18 to 98 years) at five sampled sites (Baltimore, Durham, Los Angeles, St. Louis, and New Haven) were interviewed face-to-face twice, approximately 12 months apart (waves 1 and 2), between 1980 and 1985. Lifetime and 1-year DSM-III (33) diagnostic data were obtained at both the waves. Our analytic sample includes only cases with valid responses at both waves (n=18,943). Data were weighted to account for selection and nonresponse effects and to match age, sex, and race distributions in the 1990 US census, in order to provide nationally representative estimates.

Major depression

The present study compares ECA respondents with no history of MDD at wave 1 (n=18,239; 52.1% female, 68.1% white, 68.2% high school degree or more, average age 43.0 years old) to various subgroups of those with histories of MDD at wave 1 (n=704; 73.3% female, 77.9% white, 81.5% high school degree or more, average age 37.0 years).

The ECA's DSM-III-based diagnostic criteria for MDD required ever having 2 weeks or more of sadness accompanied by at least four other symptoms. Symptoms due to physical injury or use of drugs, medication, or alcohol were excluded. Psychotic and organic episodes were also excluded. In addition, the ECA ensures clinical significance by requiring that each symptom must be adequately severe, defined as either told a doctor or another professional about the symptom, took medication more than once for the symptom, or the symptom interfered with life or activities a lot. This requirement, which relies heavily on professional help-seeking, is more demanding than the DSM-IV clinical significance requirement of distress or role impairment, which is known to have little impact on prevalence (34-36). Thus, one can expect the ECA sample to be more severe than a usual DSM-defined sample.

The ECA also imposed a syndromal severity requirement that is essentially identical to the symptom requirement in relying largely on help-seeking but applied to the overall syndrome. We suspended the syndrome-severity requirement, because it was largely redundant with the already demanding symptom-severity requirement, and it pushes the sample even further in a direction of greater severity. The suspension of the syndrome-severity criterion modestly increased the MDD sample size from 945 to 1065.

Previous studies that have attempted to examine the trajectory of depression have sometimes failed to distinguish between recurrence and chronicity as explanations of MDD at the follow-up interview, due to lack of information about the trajectory between interviews (16,37). Because our interest was specifically in measuring recurrence of MDD at wave 2 in those with remitted lifetime MDD at wave 1, we ensured an appropriate sample by eliminating from the MDD analytic sample all

those cases in which the respondent reported experiencing an ongoing episode at the time of the wave 1 interview. The elimination of wave 1 ongoing cases reduced the size of our primary MDD sample from 1065 to 704.

Primary exclusion proposals to be tested

The primary ideas for reducing the scope of MDD that are tested in the present study were: a) excluding "uncomplicated" episodes (i.e., episodes that last no longer than 2 months and do not include suicidal ideation, psychotic ideation, psychomotor retardation or feelings of worthlessness); b) excluding mild episodes (i.e., episodes with only five to six symptoms); and c) excluding nonmelancholic episodes.

In evaluating each of these proposals, multiple episode cases were classified in accordance with the classification of the individual's worst episode, for which we had detailed data. This was based on the assumption that if the worst episode was uncomplicated, or mild, or nonmelancholic, then it is unlikely that the individual's other episodes would be complicated, severe, or melancholic, respectively, because those would generally be judged worse.

Uncomplicated depression

This proposal extends to all potential MDD the exclusion for "uncomplicated" episodes that the DSM currently applies only to bereavement-related depression in the "bereavement exclusion" (criterion E of major depressive episode). Exclusion as an uncomplicated episode requires brief duration (no more than 2 months) and the absence of four specific symptoms (psychomotor retardation, suicidal ideation, psychotic ideation, sense of worthlessness) identified by the DSM as "uncharacteristic" of normal sadness.

Uncomplicated MDD episodes thus include only "general distress" symptoms that are common in normal reactions to stress, such as sadness, insomnia, loss of interest in usual activities, decrease in appetite, and difficulty in concentrating. The "uncomplicated" criteria were created to distinguish normal range from disordered depressive episodes during bereavement (38,39), and the proposal to be evaluated is that the distinction should be extended to MDD in general (40).

The DSM-IV also specifies that, in order to be excluded as uncomplicated, a case must lack marked impairment. We were unable to include this feature in our criteria due to lack of appropriate data, because of the way the ECA severity questions were structured with skip-outs. This deviation from DSM-IV increases the difficulty of supporting the "uncomplicated" hypothesis, because it allows episodes with more marked impairment – and thus more likelihood of disorder and recurrence – to qualify as excluded. However, this limitation is consistent with both the ICD's and

DSM's aspiration to separate role impairment from disorder diagnosis.

Mild (low-symptom) MDD

It has been suggested that the MDD threshold should be raised to seven symptoms from the current five (41,42) "to lower the risk of 'over-diagnosis' or pathologizing 'normal' depression or sadness" (42). This proposal is supported by findings over the years suggesting a discontinuity between MDD episodes with five to six and seven to nine symptoms (4,5,43,44). As Maj (13) notes, "when a point of rarity has been reported, it has usually corresponded to a threshold higher than that fixed by the DSM-IV". Moreover, previous studies suggest that severity in the form of number of symptoms predicts recurrence (16,18).

We considered that a narrower exclusion might yield better results. Thus, we also tested a variant proposal to exclude only cases with five symptoms, setting the MDD threshold at six symptoms.

Nonmelancholic MDD

The third proposal is to restrict MDD to the traditional category of melancholic depression, long considered the quintessential form of depressive disorder, and frequently targeted in depression scale development and research (45). The rest of what we presently call depression would be conceived as various other forms of dysphoria (e.g., "In reality, nonmelancholia is a mixture of dysphoria, anxiety, and depressive character"(45)). Maj (13) reviews some research suggesting that there is "a subtype [of MDD], grossly corresponding to DSM-IV major depression with melancholia, which may be qualitatively different from normal sadness" and argues that melancholia warrants further examination in reconsidering the normal/disordered boundary.

The ECA did not define melancholic depression and did not contain all the symptom questions relevant to diagnosis of DSM melancholic depression. As an approximation, we defined melancholic depression as episodes that included three or more from a list of five melancholic-like ECA symptom items: fatigue ("felt tired out all the time"), psychomotor retardation ("talked or moved more slowly than is normal for you"); agitation ("had to be moving all the time, that is, you could not sit still and paced up and down?"); decreased interest in sex ("your interest in sex was a lot less than usual"); and slowed thinking ("your thoughts came slower than usual or seemed mixed up"). Nonmelancholic depression was defined as any MDD cases that had less than three melancholic symptoms.

We considered that a narrower exclusion might yield better results. Thus, we also tested a variant proposal to exclude only cases with less than two melancholic

symptoms, setting the threshold for melancholic depression at two melancholic symptoms rather than three.

Hypothesis testing procedure

Testing of primary hypotheses

Using lifetime MDD diagnoses at wave 1, for each of our five proposals (i.e., the three primary proposals plus the two narrower variants), we identified the subgroup of MDD proposed for exclusion, all other MDD, and those with no history of MDD. We then compared 1-year MDD recurrence/occurrence rates at wave 2 (1 year later) for these three wave 1 groups and tested differences in rates for significance.

We considered a proposal for excluding a subgroup from MDD to be disconfirmed if the subgroup's wave 2 recurrence rate was not significantly different from the rate for other MDD; to be weakly confirmed as worth further exploration if the subgroup's recurrence rate was significantly and substantially lower than the rate for other MDD; and to be strongly supported if the subgroup had a recurrence rate that was not only significantly lower than the recurrence rate for other MDD but also not significantly higher than the 1-year wave 2 MDD occurrence rate for those who had no prior history of MDD at wave 1.

Component analysis of "uncomplicated depression"

In a post-hoc analysis, we examined whether the results for "uncomplicated depression" were due to just one of the two criteria (brief, no uncharacteristic symptoms) or to an interaction between the two. To examine this question, we recalculated recurrence outcomes separately for the two components.

Increasing the "uncomplicated" duration limit to 6 months

Recent evidence regarding bereavement-related depression (31,46,47) suggests that the current 2-month DSM-IV duration threshold for uncomplicated episodes may be too low and that more valid cut-points may exist at higher durations. In a post hoc analysis, we tested whether our results for uncomplicated MDD were preserved if the duration threshold was lengthened to 6 months.

Reanalysis using the unadjusted ECA MDD sample

As noted, we made two changes to ECA diagnostic criteria in deriving our sample: we eliminated cases that reported ongoing episodes at wave 1 and we suspended the syndrome-level severity requirement. Concerned that these changes inadvertently might have introduced a bias into the results, we reanalyzed the data using the unadjusted ECA MDD sample, applying the ECA's syndrome-severity requirement and including MDD cases with ongoing episodes at wave 1. This increased our MDD sample from 704 to 945 cases. Both the changes would be expected to inflate the recurrence rates of proposed-for-exclusion subgroups, increasing the difficulty of confirming the exclusion proposals.

Recurrence in multiple-episode versus single-episode cases

We examined whether the frequent finding in the literature that multiple-episode cases predict higher recurrence held for our sample and was similar in uncomplicated and other MDD cases.

Statistical analysis

All data were weighted and corrected for sampling design to approximate a representative sample of the American population. Statistical analyses used Stata 12 survey estimation procedures, which calculate weighted coefficients and used Taylor series linearization to calculate standard errors (48).

As we hypothesized specific directions of the tested relationships, the analyses included one-tailed t-tests of statistical significance in mean differences for the percentage of respondents reporting a 1-year MDD at wave 2, between the different categories of cases.

RESULTS

Evaluation of five proposals for narrowing MDD's scope

We evaluated five proposals for narrowing the scope of MDD, using recurrence as a validator. Only one proposal, to exclude uncomplicated MDD, passed our most stringent test. Among those with lifetime uncomplicated MDD at wave 1, the likelihood of MDD recurrence at wave 2 was 3.4%, which was not significantly greater than the likelihood of 1-year occurrence of MDD at wave 2 among those with no history of MDD at wave 1 (1.7%) and was significantly lower than the recurrence rate at wave 2 of all other MDD (14.6%) (Table 1).

The proposals to exclude mild cases (defined as MDD with five to six symptoms or in a narrower variant as five-symptom MDD) and nonmelancholic cases (defined as having no more than two melancholic symptoms or in a narrower variant as having not more than 1) failed to pass this strong test (Table 1). This was because the recurrence rates for these proposed excluded groups (ranging from 8.8% to 10.6%) were in every case

Table 1 Recurrence rates of three subgroups proposed for exclusion from MDD, compared to occurrence rates of those with no history of MDD and recurrence rates of other (nonexcluded) MDD

MDD subgroup proposed for exclusion	Subgroup's recurrence rate	Is the subgroup's recurrence rate different from the No History of MDD group's rate=1.7% (1.4%, 1.9%), [n=18,239]	Recurrence rate for All Other MDD (except the subgroup)	Is the subgroup's recurrence rate different from the recurrence rate for All Other MDD?
Uncomplicated (brief and no psychomotor retardation, suicidal ideation, or felt worthless/guilty)	3.4 (−0.4, 7.1) [n=88]	**Not** different from No History	14.6 (11.3, 17.9) [n=616]	Different from Other MDD
Low-symptom (five to six MDD symptom groups)	9.6 (6.6, 12.6) [n=487]	Different from No History	20.7 (14.1, 27.3) [n=217]	Different from Other MDD
Narrowed low-symptom (five MDD symptom groups)	8.8 (5.6, 11.9) [n=310]	Different from No History	16.2 (11.9, 20.4) [n=394]	Different from Other MDD
Nonmelancholic (at most two melancholic symptoms)	10.6 (7.3, 13.9) [n=503]	Different from No History	19.2 (12.8, 25.6) [n=201]	Different from Other MDD
Narrowed nonmelancholic (at most one melancholic symptom)	10.4 (5.2, 15.7) [n=279]	Different from No History	14.9 (11.3, 18.4) [n=425]	Different from Other MDD

Significant differences between groups tested using one-tailed t-test, p <0.05

significantly higher than the occurrence rate for those with no MDD history (1.7%). However, the recurrence rates for these groups were in every case significantly and substantially lower than the recurrence rates for other MDD (ranging from 14.9% to 20.7%), and to this extent, these proposals warrant further examination for implications about the MDD boundary.

Component analysis of "uncomplicated depression"

We explored whether the "uncomplicated" hypothesis's confirmation was due to just one of the two criteria (brief duration, no uncharacteristic symptoms) or an interaction between the two components. Although uncomplicated MDD (n=88) had a recurrence rate of 3.4%, brief cases (n=326) had a recurrence rate of 12.6%, and cases lacking the uncharacteristic symptoms (n=153) had a recurrence rate of 7.3% (Table 2). Both of the components' rates were significantly higher than the no-MDD-history group. Clearly, the dramatic result for uncomplicated MDD in which its recurrence rate is no higher than the general population's MDD rate is due to an interaction, with both duration and symptom requirements contributing to this result.

Reanalysis of the "uncomplicated MDD" proposal with 6-month duration limit

On the basis of the earlier studies, we hypothesized that increasing the duration criterion for "uncomplicated episode" to 6 months would not alter the results of recurrence-rate comparisons. This prediction was confirmed. When uncomplicated duration was allowed to be up to 6 months, the number of uncomplicated cases increased by about 38% to 121 cases, yet the results of recurrence comparisons remained the same. The recurrence rate for uncomplicated cases still did not differ significantly from no-MDD-history cases (5.7% vs. 1.7%), and was significantly lower than other MDD (14.7%).

Reanalysis using the unadjusted ECA MDD sample

To check whether the adjustments we made to the ECA MDD criteria might have inadvertently biased the results in our favor, we repeated the recurrence analyses using the unadjusted ECA MDD sample. The results of comparisons (not displayed) were identical to the results of our primary analyses. Once again, only uncomplicated MDD (n=95) passed our strongest test as a potential variant of normal sadness. Uncomplicated MDD had an unadjusted recurrence rate not significantly different from the no-MDD-history group's occurrence rate and significantly lower than the rate for other MDD (1.7%, 5.0%, and 17.3%, respectively). All other subgroups proposed for exclusion had recurrence rates (range 7.2% to 10.3%) significantly higher than no-MDD-history's 1.7% rate but significantly lower than other MDD cases (range 17.7% to 19.1%).

Because this analysis of wave 2 MDD recurrence includes cases that involved an ongoing MDD episode at wave 1, in effect it evaluates a combination of recurrence and chronicity. This expanded test still yields the same verdict that uncomplicated MDD is not different from no-MDD-history but is very different from other MDD.

Recurrence in single-episode versus multiple-episode cases

Many studies indicate that multiple-episode MDD cases predict substantially higher recurrence. We, thus,

Table 2 Follow-up analyses: recurrence rates of components for uncomplicated criteria compared to occurrence rates of those with no history of MDD and recurrence rates of other (nonexcluded) MDD

Component analysis of uncomplicated	Subgroup's recurrence rate	Is the subgroup's recurrence rate different from the No History of MDD group's rate=1.7% (1.4%, 1.9%), [n=18,239]	Recurrence rate for All Other MDD (except the subgroup)	Is the subgroup's recurrence rate different from the recurrence rate for All Other MDD?
Brief (2 months or less duration)	12.6 (8.9, 16.3) [n=326]	Different from No History	13.6 (9.2, 18.0) [n=378]	**Not** different from Other MDD
No uncharacteristic symptoms (psychomotor retardation, suicidal ideation, or felt worthless/guilty)	7.3 (2.6, 11.9) [n=154]	Different from No History	14.7 (11.2, 18.1) [n=550]	Different from Other MDD

Significant differences between groups tested using one-tailed t-test, p<0.05

examined whether recurrence for uncomplicated MDD was predominantly due to multiple-episode cases. Although the low recurrence rate limited this analysis, we found recurrences similarly distributed between single- and multiple-episode cases. Of 43 single-episode uncomplicated wave 1 cases, two had wave 2 1-year MDD (3.0%; 95% CI: -1.2, 7.3); of 45 multiple-episode uncomplicated wave-1 cases (median episodes=3), again two cases had 1-year wave-2 MDD (3.7%; 95% CI: −2.2, 9.6). In each instance, one wave 2 case was complicated and one uncomplicated. This pattern of data is consistent with the situational determination of depressive episodes, in which no enduring tendency to recurrence is sustained despite multiple past episodes.

The situation was strikingly different with respect to other (complicated) MDD cases, in which multiple-episode wave 1 MDD (median episodes=3) had a significantly and substantially higher 1-year wave 2 recurrence rate (19.1%; 95% CI: 12.2, 24.1) than did single-episode cases (7.2%; 95% CI: 3.3, 11.2). The multiple-episode uncomplicated cases' substantially lower recurrence rate as compared to other multiple-episode cases offers a hitherto unnoticed clinical differentiation. In sum, uncomplicated MDD multiple-episode histories do not predict higher recurrence than single-episode histories, whereas other MDD multiple-history episodes do predict higher recurrence.

DISCUSSION

Recurrence is a cardinal feature of depressive disorder and can be used to evaluate the plausibility of proposed changes to the MDD diagnostic threshold. Our analysis identified a subset of current MDD – namely, uncomplicated MDD – for which recurrence was no greater than background occurrence of MDD in the general population who never had MDD, a criterion frequently mentioned for recognizing normal intense suffering versus depressive disorder (23,31,49).

Another frequently mentioned criterion is the "similarity thesis", according to which the strongest evidence that a subgroup of MDD is a form of pathology is that it is similar on validators to other MDD (22,50). Our results strongly disconfirm the similarity thesis for uncomplicated versus other MDD in terms of recurrence rates. The lack of a difference in recurrence rate between multiple-episode and single-episode uncomplicated cases also marks uncomplicated MDD as importantly dissimilar from other MDD. The other hypotheses we examined displayed some differences from other MDD as well, but the differences were not as consistent or pronounced, so the status of these other proposals remains more ambiguous.

A series of additional analyses confirmed the stability of our results. We lowered the thresholds for the low-symptom and nonmelancholic proposals, reanalyzed all the proposals using the original ECA MDD sample rather than our adjusted sample, and tried a 6-month duration in the definition of "uncomplicated", all without changing the essential results. We explored the source of our "uncomplicated" result by analyzing each definitional component separately, and found that support for the "uncomplicated" hypothesis resulted from an interaction between the two components. Our results in shifting the uncomplicated duration threshold to 6 months suggest that further research should explore the proper duration threshold for MDD and for any potential "uncomplicated MDD" exclusion.

Our results underscore the heterogeneity of current DSM MDD (12) and offer findings regarding recurrence risk that are clinically relevant irrespective of the issue of MDD diagnostic boundaries. Monroe and Harkness (20) express concern that "research focusing on depression as a recurrent condition has generally failed to reveal any useful early indicators of risk for recurrence". Our results reveal several such indicators of differential recurrence risk.

An episode can be judged uncomplicated only if it does not persist past 2 months. This presents a challenge to the clinician who attempts to apply these results, because of the need to monitor the patient, using "watchful waiting" until the condition remits or the 2-month duration is reached. The challenge is, however, limited. About 78% of our MDD cases displayed one or more of the uncharacteristic symptoms and so would be diagnosed as MDD on that basis alone, without reference to duration (Table 2). Of the remaining 22% who displayed no uncharacteristic symptoms, about 60% (13% of the MDD sample) remitted by 2 months and qualified as "uncomplicated" cases, whereas the remainder persisted beyond the 2-month duration limitation for uncomplicated cases. Consequently, briefly monitoring such individuals may be crucial to optimal formulation of prognosis and diagnosis. In some instances, treatment for symptom relief may be advisable much sooner, whether the condition is judged normal or disordered. Further research should be aimed at identifying low-recurrence uncomplicated-type MDD cases with minimal use of the duration threshold, so that adequate assessment need not depend on monitoring over time.

Limitations of the ECA dataset and of our measures must be considered in interpreting our findings. A major limitation is the ECA's 1-year window for assessing recurrence. Further studies are needed to see if our results are replicated at longer follow-up periods. Another limitation is that diagnoses were based on respondents' fallible recollections of symptoms.

The ECA's DSM-III-based MDD diagnostic algorithm lacked the DSM-IV's clinical significance criterion, and the ECA's structure also made it impossible to apply the DSM's impairment criterion for uncomplicated MDD. However, the ECA's demanding symptom severity criteria more than compensated. Indeed, the ECA sample is likely more severe on average than DSM MDD, so one would expect this study to offer an especially demanding test of the examined exclusion proposals.

The DSM criteria for uncomplicated MDD require that there be no morbid preoccupation with worthlessness — a more demanding symptom level than the ECA "felt worthless" symptom that we used as a proxy. Also, the ECA worthlessness item included feeling guilty or sinful as alternatives. Thus, the ECA worthlessness item likely excluded somewhat fewer cases from MDD diagnosis than the stricter DSM criterion. It seemed preferable to use this weaker version rather than to ignore the worthlessness criterion altogether, given that since Freud such feelings of low self-esteem have been considered a crucial indicator that normal sadness may have transformed into pathology.

Because ECA severity relied partly on service contact, but the severity responses were not encoded in the ECA data set, we could not analytically distinguish treatment effects from MDD's inherent course. Thus, treatment might have influenced the course of these depressions and their recurrences. However, previous studies indicate that recurrence remains high in treated MDD samples and is not reduced to general population levels (25,27-29). Thus, treatment effects would be unlikely to explain why the uncomplicated group's recurrence is not only substantially less than other MDD but also not different from the general population. The severity indicator does reveal that uncomplicated MDD cases are often seen by professionals, thus that these uncomplicated cases are clinically relevant.

Recurrence as a validator has the advantage that it is logically independent of the criteria used to define uncomplicated, low-symptom, and nonmelancholic MDD, so its use to evaluate these proposals does not tautologically bias the results. Moreover, recurrence is relevant to distinguishing normality from disorder, whereas many other commonly used validating variables, from lack of social support to suppressed immunological function, correlate both with disorder and intense normal distress and so do not distinguish these hypotheses. Previous attempts to address perceived MDD overdiagnosis generally focused on enhanced clinical significance/impairment criteria to raise the MDD diagnostic threshold (51). This strategy proved both conceptually misguided and empirically ineffectual (34-36,52-55).

However, recurrence as a validator imposes some limitations. Strictly speaking, recurrence is neither necessary nor sufficient for disorder. It is not necessary because in principle there can be one-episode genuine depressive disorders that do not confer a liability to recurrence (20). It is not sufficient because recurrence is conceptually ambiguous. It may represent the reemergence of an underlying dysfunction and thus indicate disorder, but it also might indicate, for example, the repetition of an earlier normal reaction due to either a chronic stressor or the occurrence of a related new stressor (e.g., loss of a spouse may lead to financial problems that in turn cause loss of a home, with each new stressor triggering distress). Future studies would benefit from a more nuanced, contextualized approach to understanding recurrence.

An additional limitation is that, due to the nature of the ECA dataset, we were unable to distinguish stress-triggered from nontriggered "endogenous" depressive episodes, so "uncomplicated" was applied without context sensitivity. However, previous studies indicate that application of DSM MDD criteria to community samples yields cases in which the vast majority have episodes triggered by a stressor. For example, in the National Comorbidity Survey, only 5% of MDD cases reported no trigger for their depressive feelings (38). Moreover, previous analyses indicate that almost no endogenous cases satisfy "uncomplicated" criteria. Thus, this gap in ECA data likely did not substantially influence the present study's results.

In conclusion, using elevated recurrence rates to predictively validate disorder, our results indicate that eliminating uncomplicated episodes would increase the validity of MDD diagnosis. The results also confirm that less-severe forms of MDD, such as low-symptom and non-melancholic MDD, have lower recurrence rates than other MDD and warrant further exploration regarding the proper threshold for distinguishing MDD from normal sadness.

Uncomplicated MDD includes only symptoms common in normal distress, is transient, and, in our ECA sample, does not raise recurrence rates significantly above population levels. If these findings are replicated, there is no empirical rationale for generally distinguishing uncomplicated MDD from intense normal sadness. Consideration should thus be given to eliminating uncomplicated episodes as a class from MDD diagnosis. Exceptional cases that appear pathological despite their uncomplicated status could then be diagnosed under mood disorders not otherwise specified. Alternatively, treatment guidelines should emphasize stepped treatment, in light of the dramatic differences in prognosis between uncomplicated and other MDD.

References

1. Clayton P, Desmarais L, Winokur G. A study of normal bereavement. Am J Psychiatry 1968;125:168-78.
2. Regier DA, Kaelber CT, Rae DS et al. Limitations of diagnostic criteria and assessment instruments for mental disorders: implications for research and policy. Arch Gen Psychiatry 1998;55:109-15.
3. Kendler KS, Munoz RA, Murphy G. The development of the Feighner criteria: a historical perspective. Am J Psychiatry 2010;167:134-42.
4. Kendler KS, Gardner CO. Boundaries of major depression: an evaluation of DSM-IV criteria. Am J Psychiatry 1998;155:172-7.
5. Kessler RC, Zhao SY, Blazer DG et al. Prevalence, correlates, and course of minor depression and major depression in the National Comorbidity Survey. J Affect Disord 1997;45:19-30.
6. Klein DF, Thase M. Medication versus psychotherapy for depression: progress notes. Am Soc Clin Psychopharmacol 1997;8:41-7.
7. Eaton WW, Anthony JC, Gallo J et al. Natural history of Diagnostic Interview Schedule/DSM-IV major depression: the Baltimore Epidemiologic Catchment Area follow-up. Arch Gen Psychiatry 1997;54:993-9.
8. Eaton WW, Neufeld K, Chen LS et al. A comparison of self-report and clinical diagnostic interviews for depression: Diagnostic Interview Schedule and Schedules for Clinical Assessment in Neuropsychiatry in the Baltimore Epidemiologic Catchment Area follow-up. Arch Gen Psychiatry 2000;57:217-22.
9. Moffitt TE, Caspi A, Taylor A et al. How common are common mental disorders? Evidence that lifetime prevalence rates are doubled by prospective versus retrospective ascertainment. Psychol Med 2010;40:899-909.
10. Tyrer P. Are general practitioners really unable to diagnose depression? Lancet 2009;374:589-90.
11. Fink M, Taylor MA. Resurrecting melancholia. Acta Psychiatr Scand 2007;115(Suppl. 433):14-20.
12. Goldberg D. The heterogeneity of "major depression". World Psychiatry 2011;10:226-8.
13. Maj M. When does depression become a mental disorder? Br J Psychiatry 2011;199:85-6.
14. Maj M. Refining the diagnostic criteria for major depression on the basis of empirical evidence. Acta Psychiatr Scand 2011;123:317.
15. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR, 4th ed. Washington: American Psychiatric Association, 2000.
16. Burcusa SL, Iacono WG. Risk for recurrence in depression. Clin Psychol Rev 2007;27:959-85.
17. Colman I, Naicker K, Zeng Y et al. Predictors of long-term prognosis of depression. Can Med Assoc J 2011;183:1969-76.
18. Coryell W, Endicott J, Keller MB. Predictors of relapse into major depressive disorder in a nonclinical population. Am J Psychiatry 1991;148:1353-8.
19. Judd LL, Akiskal HS, Maser JD et al. A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. Arch Gen Psychiatry 1998;55:694-700.
20. Monroe SM, Harkness KL. Recurrence in major depression: a conceptual analysis. Psychol Rev 2011;118:655-74.
21. Mueller TI, Leon AC, Keller MB et al. Recurrence after recovery from major depressive disorder during 15 years of observational follow-up. Am J Psychiatry 1999;156:1000-6.
22. Zisook S, Shear K, Kendler KS. Validity of the bereavement exclusion criterion for the diagnosis of major depressive disorder. World Psychiatry 2007;6:102-7.
23. Mojtabai R. Bereavement-related depressive episodes: characteristics, 3-year course, and implications for DSM-5. Arch Gen Psychiatry 2011;68:920-8.
24. Greden JF. The burden of recurrent depression: causes, consequences, and future prospects. J Clin Psychiatry 2001;62(Suppl. 22):5-9.
25. Nierenberg AA, Petersen TJ, Alpert JE. Prevention of relapse and recurrence in depression: the role of long-term pharmacotherapy and psychotherapy. J Clin Psychiatry 2003;64(Suppl. 15):13-7.
26. Solomon DA, Keller MB, Leon AC et al. Multiple recurrences of major depressive disorder. Am J Psychiatry 2000;157:229-33.
27. Bockting CLH, ten Doesschate MC, Spijker J et al. Continuation and maintenance use of antidepressants in recurrent depression. Psychother Psychosom 2008;77:17-26.
28. Clarke GN, Rohde P, Lewinsohn PM. Cognitive-behavioral treatment of adolescent depression: efficacy of acute group treatment and booster sessions. J Am Acad Child Adolesc Psychiatry 1999;38:272-9.
29. Geddes JR., Carney SM, Davies C et al. Relapse prevention with antidepressant drug treatment in depressive disorders: a systematic review. Lancet 2003;361:653-61.
30. Frank E, Prien RF, Jarrett RB et al. Conceptualization and rationale for consensus definitions of terms in major depressive disorder: remission, recovery, relapse, and recurrence. Arch Gen Psychiatry 1991;48:851-5.
31. Wakefield JC, Schmitz MF. Recurrence of depression after bereavement-related depression: evidence for the validity of DSM-IV bereavement exclusion from the Epidemiologic Catchment Area Study. J Nerv Ment Dis 2012;200:480-5.
32. Robins LN, Regier DA. Psychiatric disorders in America. New York: Free Press, 1991.
33. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 3rd ed. Washington: American Psychiatric Association, 1980.
34. Spitzer RL, Wakefield JC. DSM-IV diagnostic criterion for clinical significance: does it help solve the false positives problem? Am J Psychiatry 1999;156:1856-64.

35. Wakefield JC, Schmitz MF, Baer JC. Does the DSM-IV clinical significance criterion for major depression reduce false positives? Evidence from the NCS-R. Am J Psychiatry 2010;167:298-304.

36. Zimmerman M, Chelminski I, Young D. On the threshold of disorder: a study of the impact of the DSM-IV clinical significance criterion on diagnosing depressive and anxiety disorders in clinical practice. J Clin Psychiatry 2004;65:1400-5.

37. Barkow K, Maier W, Ustun B et al. Risk factors for depression at 12-month follow-up in adult primary health care patients with major depression: an international prospective study. J Affect Disord 2003;76:157-69.

38. Maj M. Bereavement related depression in the DSM-5 and ICD-11. World Psychiatry 2012;11:1-2.

39. Wakefield JC, First MB. Validity of the bereavement exclusion to major depression: does the empirical evidence support the proposal to eliminate the exclusion in DSM-5? World Psychiatry 2012;11:11-5.

40. Wakefield JC, Schmitz MF, First MB et al. Should the bereavement exclusion for major depression be extended to other losses? Evidence from the National Comorbidity Survey. Arch Gen Psychiatry 2007;64:433-40.

41. Chouinard G, Chouinard VA, Corruble E. Beyond DSM-IV bereavement exclusion criterion E for major depressive disorder. Psychother Psychosom 2011;80:4-9.

42. Parker G. Classifying clinical depression: an operational proposal. Acta Psychiatr Scand 2011;123:314-6.

43. Broadhead WE, Blazer DG, George LK et al. Depression, disability days, and days lost from work in a prospective epidemiologic survey. JAMA 1990;264:2524-8.

44. Elkin I, Shea T, Watkins JT et al. National Institute of Mental Health Treatment of Depression Collaborative Research Program: general effectiveness of treatments. Arch Gen Psychiatry 1989;46:971-82.

45. Shorter E. The doctrine of the two depressions in historical perspective. Acta Psychiatr Scand 2007;115:5-13.

46. Wakefield JC, Schmitz MF, Baer JC. Did narrowing the major depression bereavement exclusion from DSM-III-R to DSM-IV increase validity? Evidence from the National Comorbidity Survey. J Nerv Ment Dis 2011;199:66-73.

47. Wakefield JC, Schmitz MF, Baer JC. Relation between duration and severity in bereavement-related depression. Acta Psychiatr Scand 2011;124:487-94.

48. Stata Corporation. Stata statistical software, Release 12 (Version 12). Texas: Stata Corporation, 2011.

49. Kendler KS, Myers J, Zisook S. Does bereavement-related major depression differ from major depression associated with other stressful life events? Am J Psychiatry 2008;165:1449-55.

50. Zisook S, Kendler KS. Is bereavement-related depression different than nonbereavement-related depression? Psychol Med 2007;37:779-94.

51. Narrow WE, Rae DS, Robins LN et al. Revised prevalence estimates of mental disorders in the United States: using a clinical significance criterion to reconcile 2 survey's estimates. Arch Gen Psychiatry 2002;59:115-23.

52. Beals J, Novins DK, Spicer P et al. Challenges in operationalizing the DSM-IV clinical significance criterion. Arch Gen Psychiatry 2004;61:1197-207.

53. Ustun B, Kennedy C. What is "functional impairment"? Disentangling disability from clinical significance. World Psychiatry 2009;8:82-5.

54. Wakefield JC. Disability and diagnosis: should role impairment be eliminated from DSM/ICD diagnostic criteria? World Psychiatry 2009;8:87-8.

55. Wakefield JC, Spitzer RL. Lowered estimates – but of what? Arch Gen Psychiatry 2002;59:129-30.

Suicidal ideation and reported suicide attempts in Greece during the economic crisis

Marina Economou[1,2], Michael Madianos[3], Lily Evangelia Peppou[1], Christos Theleritis[1], Athanasios Patelakis[1], Costas Stefanis[1]

[1]University Mental Health Research Institute (UMHRI); [2]First Department of Psychiatry, Medical School, University of Athens, Eginition Hospital; [3]Department of Mental Health and Behavioural Sciences, School of Health Sciences, University of Athens, Athens, Greece

The financial crisis in Greece is largely impinging on the health and mental health of the population, raising concerns about a potential rise in suicide rates. The aim of this study was to explore changes in suicidal ideation and reported suicide attempts between 2009 and 2011 in a representative sample of the population and in several population subgroups. The socio-economic predictors of suicidal ideation and suicide attempts in 2011 were also investigated. Two nationwide cross-sectional telephone surveys were conducted in 2009 and 2011 using the same methodology. A random and representative sample of 2192 and 2256 people, respectively, took part in the surveys. Between 2009 and 2011, there was a substantial increase in the prevalence of suicidal ideation and reported suicide attempts. People suffering from depression, men, married individuals, people experiencing financial strain, people with low interpersonal trust, and individuals with a history of suicide attempts were particularly vulnerable.

Suicide is a huge public health issue worldwide. Each year roughly one million people die by suicide around the world, making it one of the leading causes of death internationally (1,2). Congruent with this, the World Health Organization has encouraged collection of data on the prevalence and risk factors for suicide to inform the design of public health strategies and health care policies (3). Previous research has indicated that suicidal thoughts and attempts, hereafter referred to as "suicidality", should be conceptualized as predictors of more serious suicidal acts (4,5).

Suicide/suicidality has been found to bear a strong association with both psychiatric and socio-economic factors (6). The presence of a psychiatric disorder is among the most consistently identified risk factors for suicide/suicidality (7,8), with psychological autopsy studies revealing that more than 90% of suicide victims suffered from mental illness at the time of their death (9), most commonly from mood disorders (10). Conversely, growing evidence has documented a link between suicide/suicidality and socio-economic factors, such as unemployment, income, and family status (6,11,12). In line with all this, during periods of economic recession, with its ramifications including financial hardship, job loss, and elevated prevalence of depression (13–16), suicide becomes a nagging concern (17).

In 2008, as a consequence of the global financial crisis and the local unrelenting spending, the Greek population started facing a serious socio-economic turmoil. In 2010, a memorandum of economic and financial policies was signed in order to avert Greece's default. In 2011, the financial and socio-political climate became even worse: the unemployment rate reached 16.6% in May, the gross domestic product (GDP) further declined by 6.1%, and the debt grew from 105.4% of GDP in 2007 to 160.9% (18,19). The health and mental health

implications of the economic crisis in Greece have attracted increasing attention (13,20,21), with a strong emphasis on a potential rise in suicides/suicidality (21–24). This is of primary importance, given the country's low suicide rates recorded in previous studies (25–27).

In an endeavor to gauge the impact of the financial crisis on the mental health of the Greek population, two cross-sectional nationwide studies were conducted in 2009 and 2011 (before and after the introduction of the memorandum of economic and financial policies in the daily life of the population). The present study concentrates on suicidality findings. It reports on the prevalence of suicidal ideation and suicidal attempts in a representative sample of the Greek population and in various population subgroups between 2009 and 2011 and on predictors of suicidal ideation and suicidal attempts in 2011.

METHODS

The sampling frame of the study was the national phone-number databank, providing coverage of the vast majority of households in the country. A random sample of telephone numbers belonging to individuals were selected from the directory. Within each household, the person who had his or her birthday last was selected for an interview.

Telephonic interviews were conducted with adults aged between 18 and 69 years during the same time period (February to April) in 2009 and 2011. In 2009, of the 2667 calls made, there were 2192 successfully completed interviews (response rate=82.2%). One hundred eighty four people (6.9%) hung up immediately and 290 (10.9%) refused to be interviewed or did not complete the interview. In 2011, of the 2820 calls made, there were 2256

successfully completed interviews (response rate=80.5%). Two hundred and three people (7.2%) hung up immediately and 347 (12.3%) refused to be interviewed or did not complete the interview. No statistically significant differences were found between participants who could be interviewed and those who could not in terms of gender, age, and place of residence.

All interviews were carried out by well-trained graduates in social sciences, including psychology. The training included lectures, role playing, and pilot phone interviews which were taped and reexamined, for a total of 60 h.

The method of computer-assisted telephone interviewing was used (28), as it enables automatic control of questionnaire branching, on-line verification checks, and automatic scheduling of future call backs (e.g., if the call is not answered or the interview is not completed).

Information about the occurrence of a major depressive episode, suicidal ideation, and suicide attempts during the past month was collected using the relevant module of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) (29), which has been standardized in the Greek population and extensively used in clinical and epidemiologic studies (e.g., 30,31).

To confirm the reliability of the diagnosis, a random sample of the calls (10%) was rated by ten interviewers. The Fleiss' kappa was found to be 0.89 in 2009 and 0.90 in 2011. Furthermore, a random sample of participants (10%) agreed to be reinterviewed by a psychiatrist. The rate of diagnostic agreement was 87% in 2009 and 89% in 2011.

To assess participants' degree of financial strain, the Index of Personal Economic Distress (IPED) was used (13). This self-reported measure encompasses eight questions describing participants' difficulty in fulfilling daily financial demands of a household during the last six months. Responses are made on a three-point scale (never, 1; sometimes, 2; often, 3), while the composite score of the scale can range from 8 (no economic problems) to 24 (serious economic problems). A previous exploration of the psychometric properties of the scale identified 15 as the cut-off point producing the best results in terms of sensitivity and specificity (13). Congruent with this, participants scoring above this cut-off point were regarded as being in high financial distress. The scale displayed good internal consistency (Cronbach α=0.93).

Interpersonal trust was assessed by the relevant questions from the European Social Survey (32). Questions were rated on a scale from 0 to 10, with values close to 0 indicating low interpersonal trust. As the internal consistency of the scale was good (Cronbach α=0.77), a composite scale score was computed.

The socio-demographic characteristics of the respondents (gender, age, family status, place of residence, education, employment status) and their previous contacts with mental health professionals, use of psychiatric medication, and admissions to an inpatient service were recorded.

Table 1 Socio-demographic characteristics of the 2009 and 2011 samples compared to the 2001 population census

	2009		2011		2001
	n	%	n	%	%
Gender					
Male	1080	49.3	1090	48.3	46.8
Female	1112	50.7	1166	51.7	54.2
Age groups					
<24	210	9.6	223	9.9	9.2
25–34	411	18.7	426	18.9	19.8
35–44	325	14.8	367	16.3	16.2
45–54	452	20.6	425	18.8	19.4
55–64	384	17.5	368	16.3	16.0
>65	409	18.6	447	19.8	18.4
Marital status					
Single	486	22.2	455	20.2	21.0
Married	1487	67.8	1585	70.3	70.0
Widowed	123	5.9	138	6.1	7.1
Divorced	96	4.3	78	3.5	3.0
Education (years)					
<11	1077	49.1	1351	59.9	61.0
12	813	37.1	654	29.0	28.0
>13	302	13.8	251	11.1	11.0
Place of residence					
Athens greater area	808	36.9	847	37.5	39.3
Thessalonica and central Macedonia	275	12.5	388	17.2	15.0
Rest of country	1109	50.6	1021	45.3	45.7
Occupation					
Employed	1184	84.4	1214	85.5	94.0
Unemployed	218	15.6	206	14.5	6.0

The study received approval from the University Mental Health Research Institute Ethics Committee and was performed in accordance with the ethical standards delineated in the 1964 Declaration of Helsinki. Informed consent was obtained from all participants.

To explore differences between categorical variables, χ^2 test with Yates' correction was used. To identify predictors of suicidal ideation and recent suicide attempt, two logistic regression models were computed, with presence/absence of suicidal ideation/recent suicidal attempt as the dependent variable. All analyses were performed by SPSS v.17.

RESULTS

In Table 1, the socio-demographic characteristics of the 2009 and 2011 samples are presented, along with those of the Greek population according to the 2001

Table 2 Prevalence (%) of suicidal ideation in population subgroups in 2009 and 2011

	2009 (n=2192)	2011 (n=2256)	χ^2 (df=1)	p
Gender				
Male	4.4	7.1	6.41	0.011
Female	5.9	6.3	0.05	0.817
Age				
<24	13.9	4.9	15.83	<0.001
25–34	7.0	9.9	1.19	0.275
35–44	3.4	6.6	2.69	0.100
45–54	7.4	7.8	0.00	0.923
55–64	1.9	7.2	14.41	<0.001
>65	3.2	5.0	1.08	0.299
Marital status				
Unmarried	11.6	5.6	17.24	<0.001
Married	2.3	7.3	38.08	<0.001
Education				
<11	4.3	6.8	3.39	0.065
12	6.3	6.8	0.01	0.921
>13	6.0	6.6	0.17	0.680
Place of residence				
Rest of country	4.9	5.8	0.95	0.328
Athens	5.7	8.0	3.16	0.075
Employment status				
Unemployed	4.0	10.2	3.31	0.069
Economically active	5.2	7.2	3.05	0.080
Not economically active	5.3	4.9	0.06	0.796
Sought professional help				
No	4.5	4.7	0.05	0.810
Yes	8.3	17.3	13.36	<0.001
Used psychotropic medications				
No	5.2	5.9	0.86	0.353
Yes	4.5	22.7	11.10	<0.001
Major depression diagnosis				
No	2.5	2.3	0.15	0.69
Yes	54.9	56.0	0.00	0.94

census. The composition of the samples was congruent with the population census.

The proportion of respondents who reported suicidal ideation was 6.7% in 2011 versus 5.2% in 2009 ($\chi^2=3.92$, df=1, p=0.04). As shown in Table 2, a significant increase in the prevalence of suicidal ideation was observed in men (7.1% vs. 4.4%, $\chi^2=6.41$, df=1, p=0.011), but not in women (6.3% vs. 5.9%, $\chi^2=0.05$, df=1, p=0.817). The prevalence of suicidal ideation also increased among respondents aged 55–64 years (7.2% vs. 1.9%, $\chi^2=14.41$, df=1, p<0.001), while it decreased in those younger than 24 years (4.9% vs. 13.9%, $\chi^2=$

15.83, df=1, p<0.001). An increase was also observed among married individuals (7.3% vs. 2.3%, $\chi^2=38.08$, df=1, p<0.001), while the prevalence decreased in unmarried respondents (5.6% vs. 11.6%, $\chi^2=17.24$, p<0.001). Suicidal ideation was also increased between 2009 and 2011 among respondents who used psychotropic medications (22.7% vs. 4.5%, $\chi^2=11.10$, df=1, p<0.001) and those who had sought help from a mental health professional (17.3% vs. 8.3%, $\chi^2=13.36$, df=1, p<0.001).

The proportion of respondents who reported a recent suicide attempt was 1.5% in 2011 versus 1.1% in 2009 ($\chi^2=1.16$, df=1, p=0.28) (23). As shown in Table 3, a

Table 3 Prevalence (%) of reported suicide attempts in population subgroups in 2009 and 2011

	2009 (n=2192)	2011 (n=2256)	χ^2 (df=1)	p
Gender				
Male	0.4	2.0	11.12	<0.001
Female	1.8	1.0	1.93	0.165
Age				
<24	0.0	1.3	1.53	0.215
25–34	0.0	1.7	6.73	0.009
35–44	0.0	2.3	4.56	0.033
45–54	3.2	0.7	5.33	0.021
55–64	1.2	2.8	1.93	0.164
>65	1.4	0.6	0.55	0.457
Marital status				
Unmarried	2.8	1.4	3.14	0.076
Married	0.3	1.6	10.81	0.001
Education				
<11	2.1	2.9	0.39	0.530
12	0.3	1.4	1.25	0.262
>13	0.1	1.2	5.96	0.015
Place of residence				
Rest of country	1.3	1.8	0.74	0.388
Athens	0.9	1.1	0.02	0.874
Employment status				
Unemployed	0.0	4.4	4.12	0.042
Economically active	0.4	1.1	1.95	0.162
Not economically active	1.9	1.6	0.03	0.861
Sought professional help				
No	0.7	1.3	2.49	0.115
Yes	2.7	2.6	0.00	1.000
Used psychotropic medications				
No	1.1	1.3	0.36	0.546
Yes	1.1	5.2	1.26	0.260
Major depression diagnosis				
No	0.8	0.5	0.97	0.324
Yes	6.6	13.0	2.56	0.109

Table 4 Logistic regression results with suicidal ideation (presence/absence) as dependent variable

	OR	95% CI	p
Major depression diagnosis			
No[a]			
Yes	47.72	22.18–62.66	<0.001
Gender			
Female[a]			
Male	1.96	0.93–4.13	NS
Age (years)	0.96	0.72–1.29	NS
Education (years)	1.11	0.58–2.13	NS
Place of residence			
Athens[a]			
Rest of country	1.57	0.70–3.54	NS
Marital status			
Not married[a]			
Married	1.51	0.59–3.82	NS
Employment status			
Unemployed[a]			
Economically active	0.92	0.26–3.23	NS
Not economically active	0.71	0.15–3.25	NS
IPED (score)	1.07	1.07–1.40	<0.05
Previous suicide attempt			
No[a]			
Yes	6.89	1.66–8.60	<0.01
Interpersonal trust scale (score)	0.93	0.87–0.98	<0.001

[a]Reference category
IPED – Index of Personal Economic Distress

significant increase in the prevalence of a recent suicide attempt was observed in men (2.0% vs. 0.4%, $\chi^2=11.12$, df=1, p<0.001), but not in women (1.0% vs. 1.8%, $\chi^2=1.93$; df=1, p=0.165). The prevalence of a recent suicide attempt also increased among people aged 25–34 years (1.7% vs. 0.0%, $\chi^2=6.73$, df=1, p=0.009) and 35–44 years (2.3% vs. 0.0%, $\chi^2=4.56$, df=1, p=0.033), whereas it decreased in respondents aged 45–54 years (0.7% vs. 3.2%, $\chi^2=5.33$, df=1, p=0.021). An increase was also observed among married people (1.6% vs. 0.3%, $\chi^2=10.81$, df=1, p=0.001) and in those with the highest educational attainment (1.2% vs. 0.1%, $\chi^2=5.96$, df=1, p=0.015). No unemployed respondent reported a recent suicide attempt in 2009, while the proportion was 4.4% in 2011 ($\chi^2=4.12$, df=1, p=0.042).

As shown in Table 4, the significant predictors of suicidal ideation in 2011 were the presence of major depression during the previous month, financial hardship experienced, a previous history of suicide attempt, and a low interpersonal trust. People who fulfilled DSM-IV criteria for major depression were 48 times more likely to present suicidal ideation than people

without that diagnosis (OR=47.72, 95% CI=22.18–62.66, p<0.001). Respondents with a history of suicide attempt were seven times more likely to report suicidal ideation than people without such a history (OR=6.89, 95% CI=1.66–8.60, p<0.01).

As reported in Table 5, the significant predictors of a recent suicide attempt in 2011 were the presence of major depression during the previous month, financial hardship, a previous history of suicide attempt, being male, and being married. People who fulfilled DSM-IV criteria for major depression were 97 times more likely to report a suicide attempt during the previous month than people without that diagnosis (OR=97.39, 95% CI=71.06–134.45, p<0.01). Men were 12 times more likely to report a recent suicide attempt than women (OR=12.26, 95% CI=5.10–16.78, p<0.05), and married people were 53 times more likely to report it than unmarried ones (OR=53.29, 95% CI=33.29–64.40, p<0.05). People with a previous history of suicide attempt were 14 times more likely to report a recent suicide attempt than those without such a history (OR=14.41, 95% CI=1.79–15.95, p<0.01).

Table 5 Logistic regression results with reported suicide attempt (presence/absence) as dependent variable

	OR	95% CI	p
Major depression diagnosis			
No[a]			
Yes	97.39	71.06–134.45	<0.01
Gender			
Female[a]			
Male	12.26	5.10–16.78	<0.05
Age (years)	0.43	0.16–1.16	NS
Education (years)	2.88	0.53–5.69	NS
Place of residence			
Athens[a]			
Rest of country	1.89	0.21–6.67	NS
Marital status			
Unmarried[a]			
Married	53.29	33.29–64.40	<0.05
Employment status			
Unemployed[a]			
Economically active	0.40	0.02–6.74	NS
Not economically active	14.90	0.35–24.76	NS
IPED (score)	1.23	1.02–1.49	<0.05
Previous suicide attempt			
No[a]			
Yes	14.41	1.79–15.95	<0.01
Interpersonal trust scale (score)	1.03	0.87–1.22	NS

[a]Reference category
IPED – Index of Personal Economic Distress

DISCUSSION

This study provides evidence of a substantial increase in the prevalence of suicidal ideation and reported suicide attempts between 2009 and 2011 in a representative sample from the Greek population. People suffering from depression, men, married individuals, people experiencing financial strain, people with low interpersonal trust, and those with a previous history of suicide attempts were particularly vulnerable.

Previous research in the country has displayed important fluctuations in reported suicide ideation and suicide attempt rates across the years as well as a strong link between these rates and socio-economic factors (13,26). Specifically, in an epidemiologic study conducted in 1978, the 1-month prevalence of suicidal ideation was found to be 4.8%, whereas in 1984 it reached 10.9% (26). This substantial rise was attributed to the economic recession striking the country at the time, which peaked in 1984 and necessitated the application of economic measures during the period 1985–1986 (33). A similar trend was recorded for reported suicide attempts during the month prior to the interview, with a rate of 0.7% in 1978 and 2.6% in 1984. In the year 2008, prior to the outset of the economic crisis in Greece, the 1-month prevalence of suicidal ideation was 2.4% and that of suicide attempts was 0.6 (13). Both rates are substantially lower than those in 2009 and 2011.

In a similar vein, Zacharakis et al (25) explored suicide rates in Greece in the period 1980–1995 and found they were among the lowest in the world, possibly due to an array of social and cultural factors: the strong family ties, the support and help offered by social networks, and religious practices. Suicide rates remained low until 2009. According to the official data from the police registry, there were 507 completed suicides and serious suicide attempts in 2009, 622 in 2010, and 598 in 2011 (34).

These findings run in parallel with the gradual increase in the prevalence of major depression in Greece during the past few years (16), confirming that the economic crisis in Greece has impinged on the mental health of the population, similarly to what observed in other countries (14,35).

Our findings about the population subgroups most vulnerable to suicidality can be explained by the socio-economic variables that play a prominent role during recession, in particular unemployment, job insecurity, and loss of income (36–38). For example, people with high educational attainment might face difficulty in finding a job or might be experiencing a mismatch between their educational level and their salary. Men and married people are the breadwinners in the Greek family and may feel incapable of providing their families with the essentials due to unemployment or job insecurity. The age groups displaying the highest increase in suicidality in our study were those experiencing the largest changes in unemployment rates in recent years (39). Furthermore, people aged 55–64 years had to deal with early compulsory retirement in 2011. Research has shown that people who approach retirement at the time of an economic crisis are particularly susceptible to stress and uncertainty due to disruptions to their financial preparations (40) and reliance on fixed income resources (e.g., pensions), which are subject to market fluctuations (41).

The significant increase in the prevalence of suicidality among those who have sought professional help and those who take psychotropic medications is congruent with the increase in the use of mental health services and in the incidence of help-seeking for psychological problems during times of economic adversity (42).

In our study, the only significant protective factor with respect to suicidal ideation was interpersonal trust. This has been considered for long as a measure of social capital (43), which in turn has been linked to lower mortality rates (44), including deaths from suicide (45), lending in this way support to Durkheim's seminal work on social integration and suicide (46). Social capital in general and interpersonal trust in particular can protect citizens from developing suicidal ideation, even during periods of economic crisis. This supports recommendations to enhance social capital to mitigate the health and mental health effects of the recession (17,47).

Our data suggest that being male increases substantially the odds of attempting suicide. This can be explained by a variety of factors, such as job insecurity, loss of income, men's social roles being closely bound to employment conditions, and low availability of social support. For example, Moller-Leimkuhler (48) associated male vulnerability for suicide with the observation that men tend to be less socially integrated, have less social support, and are emotionally more isolated.

The increased risk for suicide attempts among married individuals can be interpreted in the light of the family stress model (49,50), which postulates that couples experiencing financial strain become angry, sad, and pessimistic about the future. The emotional distress leads the spouses to interact in a nonsupportive and irritable manner, resulting in marital conflict. Marital conflict in turn is associated with depression (49,50) and suicide (51). This model has received support in studies conducted in the United States (52), Czech Republic (53), Finland (54), Romania (55), and Korea (56).

Clinicians should be aware of the above risk factors. They should explore in depth whether men, people with major depression, those experiencing financial strain, individuals with previous suicide attempts, married individuals, and people with low levels of interpersonal trust display suicidal ideation or have attempted suicide. Clinicians should not ignore that about 90% of unplanned and 60% of planned first suicide attempts occur within 1 year of the onset of suicidal ideation (8). Within this context, early detection of mental health problems and depression in

particular is of primary importance (57). Programs specifically designed to train primary care physicians in the recognition and treatment of depression have reported reductions in suicide rates (58). The development of social protection programs such as labor force expansion programs, social support systems, and access to health care and health insurance might mitigate the negative mental health effects of economic crisis and unemployment (17,47).

The present study was not without its shortcomings. Due to the cross-sectional nature of the design, causal inferences cannot be drawn. In addition, reported suicidal ideation and suicide attempts can be susceptible to recall bias or to reluctance on the part of respondents to disclose such a sensitive information. Although the anonymity of telephone interviewing has been shown to facilitate the disclosure of this type of information (59,60), it cannot be excluded that the prevalence rates found in this study represent an underestimation of true rates.

In conclusion, our findings confirm that the influence of the recession on mental health in the Greek population is being pervasive and that systematic efforts should be undertaken to mitigate these effects. A strategic plan for treating major depression and preventing suicide should be implemented, if Greece is to retain its low position in suicide rates worldwide.

References

1. World Health Organization. Prevention of suicide: guidelines for the formulation and implementation of national strategies. Geneva: World Health Organization, 1996.
2. US Department of Health and Human Services. With understanding and improving health and objectives for improving health. Washington: US Department of Health and Human Services, 2000.
3. World Health Organization. Suicide prevention (SUPRE). Geneva: World Health Organization, 2007.
4. Brezo J, Paris J, Tremblay R et al. Personality traits as correlates of suicide attempts and suicidal ideation in young adults. Psychol Med 2006;36:180-206.
5. Suominen K, Isometsa E, Suokas J et al. Completed suicide after a suicide attempt: a 37-year follow-up study. Am J Psychiatry 2004;161:562-3.
6. Nock MK, Borges G, Bromet EJ et al. Suicide and suicidal behavior. Epidemiol Rev 2008;30:133-54.
7. Nock MK, Borges G, Bromet EJ et al. Cross-national prevalence and risk factors for suicidal ideation, plans, and attempts in the WHO World Mental Health Surveys. Br J Psychiatry 2008;192: 98-105.
8. Kessler RC, Borges G, Walters EE. Prevalence of and risk factors for lifetime suicide attempts in the National Comorbidity Survey. Arch Gen Psychiatry 1999;56:617-26.
9. Cavanagh JT, Carson AJ, Sharpe M et al. Psychological autopsy studies of suicide: a systematic review. Psychol Med 2003;33: 395-405.
10. Bostwick JM, Pankratz VS. Affective disorders and suicide risk: a reexamination. Am J Psychiatry 2000;157:1925-32.
11. Gunnell D, Lopatatzidis A, Dorling D et al. Suicide and unemployment in young people. Analysis of trends in England and Wales, 1921-1995. Br J Psychiatry 1999;175:263-70.
12. McMillan KA, Enns MW, Asmundson GJ et al. The association between income and distress, mental disorders, and suici-

13. Madianos M, Economou M, Alexiou T et al. Depression and economic hardship across Greece in 2008 and 2009: two cross-sectional surveys nationwide. Soc Psychiatry Psychiatr Epidemiol 2011;46:943-52.
14. Lee S, Guo WJ, Tsang A et al. Evidence for the 2008 economic crisis exacerbating depression in Hong Kong. J Affect Disord 2010;126:125-33.
15. Wang JL, Smailes E, Sareen J et al. The prevalence of mental disorders in the working population over the period of global economic crisis. Can J Psychiatry 2010;55:598-605.
16. Economou M, Madianos M, Peppou L et al. Major depression in the era of economic crisis: a replication of a cross-sectional study across Greece. J Affect Disord (in press).
17. World Health Organization. Impact of economic crises on mental health. Geneva: World Health Organization, 2011.
18. Eurostat. Euro area and EU27 government deficit at 6.0% and 6.4% of GDP respectively. Luxembourg: Eurostat, 2011.
19. Hellenic Statistical Authority. Unemployment rate at 16.6% in May 2011. Piraeus: Hellenic Statistical Authority, 2011.
20. Kentikelenis A, Papanicolas I. Economic crisis, austerity and the Greek public health system. Eur J Public Health 2012;22:4-5.
21. Kentikelenis A, Karanikolos M, Papanicolas I et al. Health effects of financial crisis: omens of a Greek tragedy. Lancet 2011; 378:1457-8.
22. Stuckler D, Basu S, Suhrcke M et al. Effects of the 2008 recession on health: a first look at European data. Lancet 2011;378: 124-5.
23. Economou M, Madianos M, Theleritis C et al. Increased suicidality amid economic crisis in Greece. Lancet 2011;378:1459.
24. Economou M, Madianos M, Peppou LE et al. Suicidality and the economic crisis in Greece. Lancet 2012;380:337.
25. Zacharakis CA, Madianos M, Papadimitriou GN et al. Suicide in Greece 1980-1995: patterns and social factors. Soc Psychiatry Psychiatr Epidemiol 1998;33:471-6.
26. Madianos M, Madianou-Gefou D, Stefanis CN. Changes in suicidal behavior among nation-wide general population samples across Greece. Eur Arch Psychiatry Clin Neurosci 1993;243:171-8.
27. World Health Organization. Suicide rates in Greece. www. who.int.
28. Ketola E, Klockars M. Computer-assisted telephone interview (CATI) in primary care. Fam Pract 1999;16:179-83.
29. First MB, Spitzer R, Gibbon M et al. Structured Clinical Interview for DSM-IV Axis I Disorders, patient edition. New York: Biometrics Research, New York State Psychiatric Institute, 1996.
30. Madianos M, Papaghelis M, Philippakis A. The reliability of SCID-I in Greece in clinical and general population. Psychiatriki 1997;8:101-8.
31. Madianos M, Economou M, Stefanis C. Long-term outcome of psychiatric disorders in the community: a 13 year follow up study in a nonclinical population. Compr Psychiatry 1998;39:47-56.
32. European Social Survey. Questionnaire. www.europeansocial survey.org.
33. Madianos M, Stefanis CN. Changes in the prevalence of symptoms of depression and depression across Greece. Soc Psychiatry Psychiatr Epidemiol 1992;27:211-9.
34. Ministry of Public Order & Citizen Protection. Table on suicides (completed and attempted) from 2009 until 10-12-2011. Athens: Police Headquarters, 2011.
35. Hong J, Knapp M, McGuire A. Income-related inequalities in the prevalence of depression and suicidal behavior: a 10-year trend following economic crisis. World Psychiatry 2011;10:40-4.
36. Gavrilova NS, Semyonova VG, Evdokushkina GN et al. The response of violent mortality to economic crisis in Russia. Popul Res Policy Rev 2000;19:397-419.

37. Gunnell D, Harbord R, Singleton N et al. Factors influencing the development and amelioration of suicidal thoughts in the general population. Cohort study. Br J Psychiatry 2004;185:385-93.
38. Chang SS, Gunnell D, Sterne JAC et al. Was the economic crisis 1997-1998 responsible for rising suicide rates in East/Southeast Asia? A time-trend analysis for Japan, Hong Kong, South Korea, Taiwan, Singapore and Thailand. Soc Sci Med 2009;68:1322-31.
39. Hellenic Statistical Authority. Research on Labour Force: May 2012. Piraeus: Hellenic Statistical Authority, 2012.
40. Kelly S. Don't stop thinking about tomorrow: the changing face of retirement – the past, the present, the future. Sydney: AMP. NATSEM, 2009.
41. Sargent-Cox K, Butterworth P, Anstey KJ. The global financial crisis and psychological health in a sample of Australian older adults: a longitudinal study. Soc Sci Med 2011;73:1105-12.
42. Catalano R. The health effects of economic insecurity. Am J Public Health 1991;81:1148-52.
43. Kawachi I. Social capital and community effects on population and individual health. Ann NY Acad Sci 1999;896:120-30.
44. Kawachi I, Kennedy BP, Lochner K et al. Social capital, income inequality and mortality. Am J Public Health 1997;87:1491-8.
45. Duberstein P, Conwell Y, Connor K et al. Poor social integration and suicide: fact or artifact? Psychol Med 2004;34:1331-7.
46. Durkheim E. Le suicide. Étude de sociologie. Paris: Les Presses universitaires de France, 1897.
47. Wahlbeck K, McDaid D. Actions to alleviate the mental health impact of the economic crisis. World Psychiatry 2012;11:139-45.
48. Moller-Leimkuhler A. The gender gap in suicide and premature death or: why are so men so vulnerable? Eur Arch Psychiatry Clin Neurosci 2003;253:1-8.
49. Conger RD, Elder GH, Lorenz FO et al. Linking economic hardship to marital quality and instability. J Marriage Fam 1990;52: 643-55.
50. Conger RD, Wallace LE, Sun Y et al. Economic pressure in African American families: a replication and extension of the family stress model. Dev Psychol 2002;38:179-93.
51. Vijayakumar L. Suicide prevention: the urgent need in developing countries. World Psychiatry 2004;3:158-9.
52. Vinokur AD, Price RH, Caplan RD. Hard times and hurtful partners: how financial strain affects depression and relationship satisfaction of unemployed persons and their spouses. J Pers Soc Psychol 1996;71:166-79.
53. Hraba J, Lorenz FO, Pechacova Z. Family stress during the Czech transformation. J Marriage Fam 2000;62:520-31.
54. Kinnunen U, Pulkkinen L. Linking economic stress to marital quality among Finnish marital couples: mediator effects. J Fam Issues 1998;19:705-24.
55. Robila M, Krishnakumar A. Effects of economic pressure on marital conflict in Romania. J Fam Psychol 2005;19:246-51.
56. Kwon HK, Rueter MA, Lee MS et al. Marital relationships following the Korean economic crisis: applying the family stress model. J Marriage Fam 2003;65:316-25.
57. Bebbington PE, Minot S, Cooper C et al. Suicidal ideation, self-harm and attempted suicide: results from the British psychiatric morbidity survey 2000. Eur Psychiatry 2010;25:427-31.
58. Mann JJ, Apter A, Bertolote J et al. Suicide prevention strategies: a systematic review. JAMA 2005;294:2064-74.
59. Evans E, Hawton K, Rodham K et al. The prevalence of suicidal phenomena in adolescents: a systematic review of population-based studies. Suicide Life Threat Behav 2005;35: 239-50.
60. Nock MK, Banaji MR. Prediction of suicide ideation and attempts among adolescents using a brief performance-based test. J Consult Clin Psychol 2007;75:707-15.

Supported employment: cost-effectiveness across six European sites

Martin Knapp[1,2], Anita Patel[2], Claire Curran[1], Eric Latimer[3], Jocelyn Catty[4], Thomas Becker[5], Robert E. Drake[6], Angelo Fioritti[7], Reinhold Kilian[5], Christoph Lauber[8], Wulf Rössler[9], Toma Tomov[10], Jooske van Busschbach[11], Adelina Comas-Herrera[1], Sarah White[4], Durk Wiersma[11], Tom Burns[12]

[1]Personal Social Services Research Unit, London School of Economics and Political Science, Houghton Street, London WC2A 2AE, UK; [2]Centre for the Economics of Mental and Physical Health, King's College London, Institute of Psychiatry, De Crespigny Park, London, SE5 8AF, UK; [3]Division of Social and Transcultural Psychiatry, Montreal, Quebec, H3A 1A1 Canada; [4]Division of Mental Health, St. George's University of London, London, UK; [5]Department of Psychiatry II, University of Ulm, BKH Günzburg, Germany; [6]New Hampshire-Dartmouth Psychiatric Research Centre, Lebanon, NH, USA; [7]Programma Salute Mentale, Azienda USL, Rimini, Italy; [8]Institute of Psychology, Health and Society, University of Liverpool, Liverpool, L69 3GL, UK; [9]Psychiatric University Hospital, Zürich, Switzerland; [10]Institute of Human Relations, Sofia, Bulgaria; [11]Psychiatry Department, University Hospital, Groningen, Netherlands; [12]University Department of Psychiatry, Warneford Hospital, Oxford, UK

A high proportion of people with severe mental health problems are unemployed but would like to work. Individual Placement and Support (IPS) offers a promising approach to establishing people in paid employment. In a randomized controlled trial across six European countries, we investigated the economic case for IPS for people with severe mental health problems compared to standard vocational rehabilitation. Individuals (n=312) were randomized to receive either IPS or standard vocational services and followed for 18 months. Service use and outcome data were collected. Cost-effectiveness analysis was conducted with two primary outcomes: additional days worked in competitive settings and additional percentage of individuals who worked at least 1 day. Analyses distinguished country effects. A partial cost-benefit analysis was also conducted. IPS produced better outcomes than alternative vocational services at lower cost overall to the health and social care systems. This pattern also held in disaggregated analyses for five of the six European sites. The inclusion of imputed values for missing cost data supported these findings. IPS would be viewed as more cost-effective than standard vocational services. Further analysis demonstrated cost-benefit arguments for IPS. Compared to standard vocational rehabilitation services, IPS is, therefore, probably cost-saving and almost certainly more cost-effective as a way to help people with severe mental health problems into competitive employment.

Key words: Supported employment, cost-effectiveness, severe mental illness, economics, work

People with severe mental illness face many challenges in securing paid work, and employment rates are low (1). Not surprisingly, many public and other bodies emphasize the need to target help on these individuals (2,3). As macroeconomic pressures mount and public budgets face substantial cuts, it becomes all the more pressing to know whether such help is cost-effective (do the outcomes justify the costs?) and to gauge its budget impact (what is the impact on overall expenditure?).

Individual Placement and Support (IPS) has emerged as an effective way to help many people with severe mental illness obtain competitive employment (4,5) and could potentially contribute to social and economic inclusion. A multisite randomized trial of IPS, conducted in six European cities (the EQOLISE study), was the first to examine directly the hypothesis that IPS would prove more effective than comparison services in Europe (6).

As in the United States (4), Canada (7), Australia (8), and Hong Kong (9), the study found that IPS participants were much more likely to work in competitive settings and worked more hours than individuals receiving comparison services. Recently, Bond et al (10) argued that the positive findings in support of IPS in the United States "may transport well into new settings as long as programs achieve high fidelity to the IPS model". However, what are the economic consequences? In this study, we examined the cost-effectiveness, budget impact, and overall economic impact of IPS, using data from the EQOLISE trial.

METHODS

Overall design of the EQOLISE study

In the EQOLISE trial, 312 individuals with severe mental illness (schizophrenia and schizophrenia-like disorders, bipolar disorder, or depression with psychotic features, using IDC-10 criteria) were randomly assigned to receive either IPS (n=156) or standard vocational services (n=156). The sample was drawn from six European cities: Groningen (Netherlands), London (UK), Rimini (Italy), Sofia (Bulgaria), Ulm-Günzburg (Germany), and Zurich (Switzerland). People who entered the trial had been ill and experiencing major difficulties accomplishing normal roles for at least 2 years and had not been employed for at least 1 year. They were followed for 18 months.

People assigned to the IPS group received IPS services with fidelity ratings ranging from good to fair (61 to 70 of 75, with a median of 65) (11). Comparison interventions, which were selected to represent the best

typical vocational rehabilitation service in each city, followed the train-and-place approach and consisted of day treatment or, in the case of Ulm, residential care. Randomization was at the individual participant level and stratified using the minimization technique by center diagnosis and work history (more or less than 1 year of employment in a previous job). Further details are given elsewhere (6,12).

Economic evaluation

The economic evaluation was carried out from the perspective of the health and social care system: the costs of mental and physical health care, social care (including care accommodation), and vocational rehabilitation services were considered. The number of days worked in competitive settings, and the percentage of sample members who worked at least 1 day, served as measures of effectiveness for the cost-effectiveness analysis.

In addition, we were interested to examine whether IPS was cost-saving compared to vocational rehabilitation services (in the sense that it cost less to run), and whether it was cost-beneficial (in the sense that the outcomes achieved by IPS when expressed in monetary terms exceeded the costs, compared to vocational rehabilitation).

Measures

Vocational staff in each service kept track of each individual's work experience on an ongoing basis, noting which individuals worked for at least 1 day in competitive settings, and alerting research staff to any jobs that clients might obtain. Research staff then contacted individuals and administered a questionnaire, at the start and after the end of each job, ascertaining hours and days worked.

Data on individual characteristics, outcomes, and use of services were collected at baseline, and 6, 12, and 18 months later. A tailored version of the Client Socio-demographic and Service Receipt Inventory – European Version (CSSRI-EU) (13) was administered at each of these assessment points to collect individual-level data on socio-demographics, usual living situation, employment, income, use of health and social care services, and medication use over the previous 6 months.

The costs of IPS and usual vocational services were calculated from information collected locally from these services in each site. To keep unit costs in line with costs estimated for other services, we applied UK unit costs to human resources. Other revenue and overhead costs were calculated on the basis of service-level data on the proportion of their total costs that were com-

prised of salary costs. We applied that same ratio to the salary costs we calculated for each service. Capital costs were excluded due to a lack of data across the six countries. Where relevant, costs were converted using purchasing power parities to 2003 prices (in British pounds). Total costs for each service were divided by the number of clients to derive average cost per client, adjusted to reflect an 18-month period. For sites with multiple IPS or other vocational services or sites that supplied data at multiple time points, we calculated costs per client for each service/time point and then took an average of these for each group.

Costs for other services were assigned by multiplying service use frequencies by unit costs. Unit costs for 2003 (when the trial began) were taken from the annual Personal Social Services Research Unit (PSSRU) volume for England (14). As other countries included in the study have no comparable sources of unit costs, and given the complications generated by using multiple cost bases, figures for England were used for all countries.

Analyses

Data were analyzed using SPSS for Windows Release 12.0.1 (15) and STATA 8.2 (16) and 10.1 (17) for Windows. Individuals were analyzed in the group to which they were randomized regardless of the type or level of input received from IPS or other vocational services. Analyses were conducted for all six centers together, on the grounds of statistical power, with subsequent examination of center-specific results.

Costs were compared at each assessment point and as totals over the whole 18-month period and are reported as mean values with standard deviations. Mean differences and 95% confidence intervals were obtained by nonparametric bootstrap regressions (1000 repetitions), which included baseline costs as a covariate.

Some values for 18-month cost data had to be imputed because 83 clients had missing cost data at one or more of the three follow-up points. Missing 18-month costs were estimated using the multiple imputation procedure in Stata 10.1, which estimated a predictive model for costs based on costs at each time point, age, gender, country, and randomization group. Budget impact was assessed by making comparisons of total costs over 18 months, both with and without imputation for missing values.

Incremental cost-effectiveness ratios were computed for each cost-outcome combination that showed both higher costs and better outcomes. These were calculated as the mean cost difference between the IPS and vocational services over the 18-month follow-up period divided by the mean difference between the groups in the outcome measure over that same period.

Table 1 Mean health and social care costs (£, 2003) for 6-month period at T0, T1, T2, and T3

| | Time | IPS | | | Vocational services | | | Difference between IPS and vocational services[a] | |
		N	Mean	SD	Valid N	Mean	SD	Mean	95% CI
Accommodation	0	156	822	4612	156	928	4801	−107	−1141, 946
	1	141	531	1952	130	391	1389	146	−222, 523
	2	133	499	1821	130	523	1977	−23	−479, 438
	3	132	536	1981	120	748	2165	−206	−715, 311
Inpatient services	0	156	6034	10,575	156	5007	10,044	1027	−1121, 3293
	1	141	1861	6830	130	4056	9737	−2580	−4335, −717
	2	133	2499	7951	130	3222	8490	−1253	−3126, 696
	3	132	3441	10,915	120	3475	9176	−606	−2837, 1640
Outpatient services	0	156	442	1415	156	269	1137	172	−119, 423
	1	141	296	1189	130	132	644	161	−40, 396
	2	133	107	467	130	105	637	3	−139, 128
	3	132	242	1314	120	41	183	199	18, 455
Community−based services	0	156	512	1531	156	480	1388	32	−273, 353
	1	141	605	1475	130	626	1484	1	−285, 286
	2	133	543	1546	130	544	1241	19	−274, 348
	3	132	911	3006	120	498	1626	417	−156, 1027
Community−based professions	0	156	977	1437	156	811	1182	166	−132, 467
	1	141	835	1233	130	1198	2960	−464	−1036, 13
	2	133	1073	4435	130	706	1172	355	−219, 1237
	3	132	834	1643	120	790	1352	−11	−377, 355
Medication	0	156	483	490	156	502	596	−19	−144, 103
	1	141	559	538	130	522	569	49	−50, 156
	2	133	520	613	128	522	604	22	−99, 155
	3	132	624	756	120	700	935	−62	−254, 117
Total (excl. intervention cost)	0	156	9269	10,980	156	7998	10,991	1271	−994, 3661
	1	141	4688	7236	130	6926	10,417	−2720	−4624, −813
	2	133	5241	9428	128	5694	9460	−960	−3228, 1443
	3	132	6589	12,560	120	6253	9905	−319	−2781, 2336

IPS – Individual Placement and Support
[a]Based on bootstrapped linear regression of group upon cost (1000 repetitions)
T1, T2, and T3 mean differences are adjusted for baseline estimate of relevant cost component

To assess the impact of sampling uncertainty on the probability that IPS is cost-effective given varying levels of willingness to pay (λ) for an additional unit of effectiveness (an additional day of work, or an additional 1% of study participants who worked for at least 1 day), cost-effectiveness acceptability curves based on the net benefit approach were constructed (18). These were based on the usual formula (net benefit=λ E – C), where E is effectiveness (additional day of work or additional 1% of clients who worked for at least 1 day), C is cost, and λ is the willingness to pay for one additional unit of effect.

A series of net-benefit values were calculated for each individual for a range of λ values between £0 and £1000 (in £200 increments). After calculating net benefit for each individual for each value of λ, coefficients of differences in net benefit between groups were obtained through a series of bootstrapped linear regressions (1000 repetitions) of group upon net benefit. The resulting coefficients were examined to calculate the proportion of times that the IPS group had a greater net benefit than the comparison services group for each value of λ. Finally, these proportions were plotted to generate cost-effectiveness acceptability curves based on pooled and site-specific perspectives. Imputed values were used for these calculations.

For the (partial) cost-benefit analysis, we calculated the monetary value of days employed minus total costs

Table 2 Costs of the IPS and vocational service interventions over 18 months, average per client (£, 2003)

	IPS	Vocational services
London	2086	3234
Ulm	1568	8586
Rimini	2467	9520
Zurich	1870	14,447
Groningen	1692	1385
Sofia	4757	1567

IPS – Individual Placement and Support

(intervention plus other services used) for the IPS and vocational rehabilitation groups, and then compared them by regressing net benefit (per individual) on randomization allocation, adjusting for baseline costs. We used bootstrap regression. The monetary value attached to each day of employment was based on the standard assumption in economic analyses that the gross wage paid is an estimate of the social value of what is produced. The average gross rate of pay for someone who was previously supported by welfare benefits because of sickness or disability was calculated from UK data on destinations of benefit leavers and the wages they earned in 2003 (19). This gives a gross average daily wage of £54.81, which was then applied to data collected in the trial on number of days worked. Note that this is a partial cost-benefit analysis, because we did not attach monetary values to any observed improvements in health or quality of life.

RESULTS

Sample

The characteristics of the sample members at baseline have been reported elsewhere: there were no differences between the IPS and control groups on any of the baseline variables measured, including age, gender, education, living situation, immigrant status, lifetime hospital admissions, distribution of diagnoses, or work history during the previous 5 years (6).

Outcomes

As reported previously (6), the EQOLISE trial found that IPS was more effective than vocational services for every vocational outcome studied: 85 (55%) of the individuals assigned to IPS worked for at least 1 day during the 18-month follow-up period compared with 43 (28%) individuals assigned to vocational services. Individuals assigned to vocational services were significantly more

Table 3 Intervention costs and total 18-month costs (£, 2003)

	IPS			Vocational services			Difference between IPS and vocational services[a]	
	N	Mean	SD	N	Mean	SD	Mean	95% CI
Intervention (IPS/vocational services)	156	2424	1110	156	6446	4816	−4022	−4791, −3239
Overall								
Excluding intervention cost								
Available cases	120	15,490	20,329	109	19,488	25,855	−5233	−10,855, 20
Imputed	156	16,453	22,514	156	18,999	23,541	−3845	−7854, 862
Including intervention cost								
Available cases	120	17,814	20,201	109	26,206	27,076	−9616	−15,544, −4262
Imputed	156	18,877	22,372	156	25,445	24,856	−7880	−12,249, −3151
Site−specific								
Including intervention cost and based on imputed data								
London	25	7414	5232	25	10,985	8929	−3769	−7654, −240
Ulm	26	18,442	17,832	26	33,414	24,275	−14,057	−24,875, −3468
Rimini	26	32,194	39,256	26	36,480	35,195	−10,261	−20,038, 601
Zurich	26	20,483	15,908	26	36,133	22,691	−17,944	−28,956, −8545
Groningen	26	22,469	23,388	26	22,209	24,912	233	−13,495, 14,171
Sofia	27	12,079	5870	27	13,359	9865	−2026	−6684, 2081

IPS – Individual Placement and Support
[a]Based on bootstrapped linear regression of group upon cost (1000 repetitions)
T1, T2, and T3 mean differences are adjusted for baseline estimate of relevant cost component
Eighty-three cases had missing cost data at one or more of the three time points

Table 4 Incremental cost-effectiveness ratios for IPS versus vocational services (based on total 18-month costs)
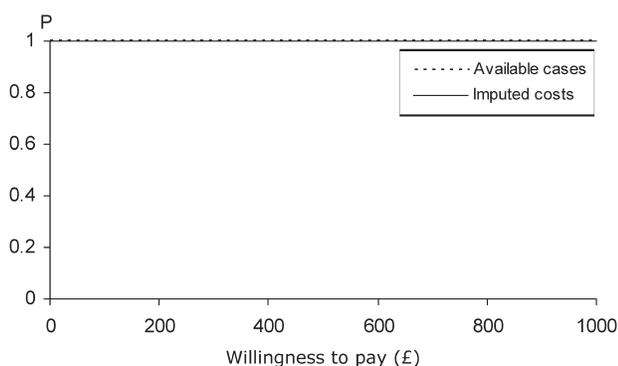
Cost perspective	Additional cost per additional 1% of people working at least 1 day	Additional cost per additional day worked
Overall–available cases	IPS dominates	IPS dominates
Overall–imputed costs	IPS dominates	IPS dominates
London–imputed costs	IPS dominates	IPS dominates
Ulm–imputed costs	IPS dominates	IPS dominates
Rimini–imputed costs	IPS dominates	IPS dominates
Zurich–imputed costs	IPS dominates	IPS dominates
Groningen–imputed costs	£233 / 7.7%=£30	£233 / 24.2 days=£10
Sofia–imputed costs	IPS dominates	IPS dominates

IPS – Individual Placement and Support

Figure 2 Probability that Individual Placement and Support is cost-effective compared with vocational services for a range of values of willingness to pay for an additional day of work
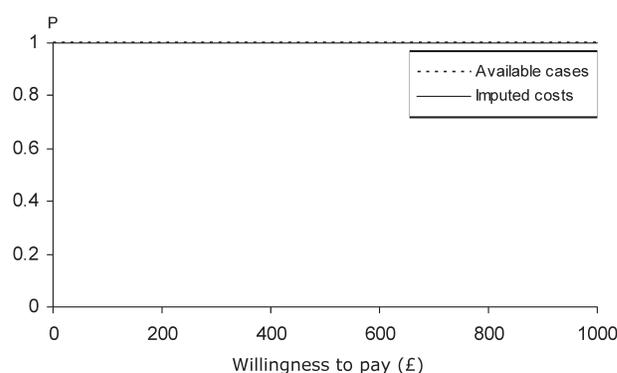
likely to drop out of the service (45%) and to be readmitted to hospital (31%) than people in the IPS arm of the trial (13% and 20%, respectively). The trial also found that context was important, with local unemployment rates explaining a substantial proportion of the observed variation in IPS effectiveness.

Costs

Inpatient costs for the IPS group, which were somewhat higher than those for the usual care group at baseline, declined much more than those for the usual care group over the first 6 months following randomization, so that adjusted inpatient costs over the first 6 months were significantly lower for IPS than for the usual care group (Table 1). However, the difference diminished over the subsequent 6 months, and these inpatient costs were virtually identical over the final 6 months. In contrast, outpatient service costs (adjusted for baseline outpatient costs) were greater for the IPS group over the final 6-month follow-up period, but the difference was small.

Total costs over the first 6 months were lower for the IPS group by more than £2,700, but differences over the two subsequent 6-month periods were not significant.

The cost of the IPS intervention itself varied threefold across sites (being highest in Sofia and lowest in Ulm), while the costs of comparison interventions varied more than 10-fold (being highest in Zurich and lowest in Groningen; Table 2). Looking only at intervention costs, IPS was more expensive than comparison services in two of the sites, less expensive in the four others.

Table 3 presents costs summed over 18 months, distinguishing between intervention and other costs (aggregated), with and without imputations for missing values. It also presents total costs (including imputations) by site. Averaged across sites, IPS services cost £4022 less than other vocational services. Total per person costs over 18 months (adjusted for baseline) were significantly lower – by about one-third – for the IPS group. Including imputations for missing values confirmed this. Total adjusted costs were lower for the IPS group at five out of six sites

Figure 1 Probability that Individual Placement and Support is cost-effective compared with vocational services for a range of values of willingness to pay for an additional 1% in people working at least 1 day
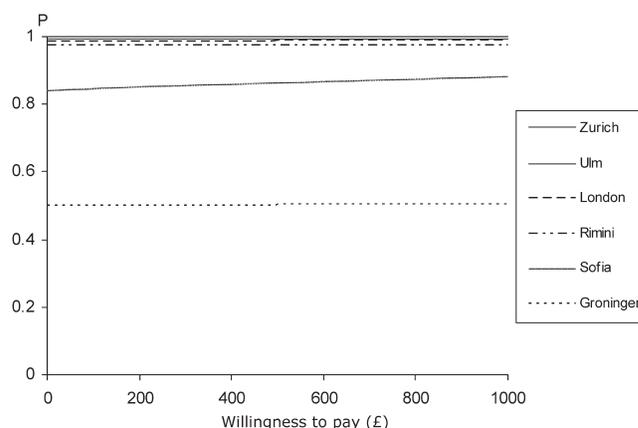

Figure 3 Probability (by site) that Individual Placement and Support is cost-effective compared with vocational services for a range of values of willingness to pay for an additional 1% in people working for at least 1 day

(the exception being Groningen), with differences for London, Ulm, and Zurich reaching statistical significance.

Cost-effectiveness

Incremental cost-effectiveness ratios were computed for each of the two outcomes in turn, first for the whole sample and then for each of the six sites (Table 4). At the five sites where overall costs were lower, IPS dominated the control condition: i.e., it was both more effective (on both outcome measures) and less costly. At the Groningen site, spending an additional £30 per person over 18 months by switching from usual vocational services to IPS resulted in an additional 1% of individuals working at least 1 day in a competitive setting; £10 per person "purchased" an additional day of work. It may be noted, however, that the difference of 24.2 days worked was large in relation to the difference of 7.7% in the proportion of people who worked at least 1 day, because one individual in the IPS service worked 456 days over the 18-month period. If this person was excluded from the analyses, the difference in days worked fell to 8.3; after this exclusion, £28 would be needed to achieve one additional day of work.

Cost-effectiveness acceptability curves illustrate the probability that IPS is cost-effective in comparison with vocational services as a function of the amount a decision maker is willing to pay for an additional 1% of clients working for at least 1 day over the 18-month period or for an additional day of work (Figures 1 and 2, respectively). This probability was nearly equal to 1 in each case, for willingness-to-pay thresholds ranging from 0 to £1000. Inclusion or noninclusion of imputed values for missing data made no material difference to the result.

Cost-effectiveness acceptability curves were plotted for each site for the outcome measuring "additional 1% of clients working" (Figure 3). With the smaller sample sizes involved, Zurich, Ulm, London, and Rimini showed the highest probabilities that IPS is cost-effective. Sofia followed closely. Groningen showed the lowest level, and IPS and vocational services would generally be interpreted from this evidence to be equivalent in that site. As a sensitivity analysis, the willingness to pay for an additional 1% of clients working at the Groningen site was increased to £5,000 and £10,000. The probability of cost-effectiveness still only reached 0.545 at the £10,000 threshold.

Cost benefit

The difference between the cost of the intervention and the value of employment achieved (days worked, valued at the expected gross wage in the UK for someone moving into employment following welfare benefits support because of sickness or disability) averaged −£9,440 for individuals in the IPS group and −£25,151 for individuals in the vocational rehabilitation group. These negative signs indicate that the costs of intervention and support exceeded the monetary value of the employment gained. To compare between the two groups, bootstrap regression (1000 replications) was used to adjust for baseline costs (to be consistent with our other analyses) and revealed a difference in net benefit of +£17,005 in favour of IPS. In other words, this (partial) cost-benefit analysis shows that IPS represents a more efficient use of resources than its comparator.

DISCUSSION

Employment is a major contributor to an individual's economic status, social position, and quality of life. Unfortunately, people with severe mental illness have high rates of unemployment. For example, a five-country European study found less than a quarter of people with schizophrenia were in paid employment, the proportion being as low as 5% in London (20). The economic and social impacts of employment difficulties are enormous. For individuals, it can mean long-term reliance on state welfare benefits, insecure low-paid work, and a disability trap that makes it hard to escape (21). For the broader society, the impacts are the risk of an almost permanently marginalized, socially excluded group of people (21), and high costs: productivity losses because of unemployment or absenteeism account for a large proportion of the overall cost of schizophrenia across many countries (22).

Public policies across much of the world emphasize the importance of promoting employment opportunities for disadvantaged groups, including people with chronic disabilities and health problems (2,3). Although that policy attention has tended to focus more on people with common mental disorders, various attempts have been made to improve access to employment for people with severe mental health problems. These include the development of sheltered work settings, clubhouse models and social firms, and, more recently, integration into competitive work settings without prior preparatory steps, following the IPS approach. IPS seeks to place people in open paid employment, providing them with intensive and ongoing support. The approach has an encouraging track record in a number of US sites, and is beginning to be explored elsewhere. This wider exploration is needed because, for example, European health systems, benefits systems and labour markets differ in important ways from those in the United States.

In this multicenter European trial of supported employment, IPS was found to dominate alternative vocational services against which it was matched, producing better outcomes in terms of both the proportion of people who worked for at least 1 day and the number of days they worked at lower cost overall to the government provider of

health and social care services. This pattern held at five of the six European centers, Groningen being the exception. With the inclusion of imputed values, the difference was maintained. An analysis of uncertainty using cost-effectiveness acceptability curves yields a consistent overall view of the findings in that, whether imputed values are used or not, IPS is almost certain to be viewed as more cost-effective than standard vocational services even if the decision maker is not willing to pay anything for an additional 1% of clients working at least 1 day or for an additional day of work. That IPS would yield better competitive employment outcomes than comparison vocational services in Europe should not be surprising, given that IPS has consistently done so almost everywhere it has been tested, whether in the United States, Canada, Australia, or Hong Kong (10). An exception is the Supported Work And Needs (SWAN) study (23), although concerns have been expressed about the fidelity of the IPS service delivered (24).

There are few cost-effectiveness results to frame the findings of the present study. Only three previous trials of IPS appear to have reported cost-effectiveness results, and cost-benefit results are even rarer. Comparing IPS with an enhanced vocational rehabilitation program in inner-city Washington, Dixon et al (25) estimated that IPS allowed clients to achieve additional hours of competitive work at an average cost of $13 per hour or $283 per additional week of competitive work (counting direct mental health costs). The SWAN trial found that, although the intervention cost only £296 per client, control group participants who were admitted to hospital had longer stays, so that total costs were £2176 higher on average for control group clients. The intervention was thus cost-effective (lower costs with similar effectiveness), but the saving in hospitalization seems unlikely to be attributable to the intervention, which had a very low intensity (23,26). Applying a cost-benefit framework to the New Hampshire trial of IPS, Clark et al (27) estimated a marginally higher benefit–cost ratio for IPS than for group skills training, from the perspectives of society as a whole (2.18 vs. 2.07) as well as from the perspective of government (1.74 vs. 1.39). Here both interventions were associated with significant, and nearly identical, reductions in costs of hospitalization.

The difference in the present study is partly attributable to IPS itself being less costly than comparison services: it cost less than comparison services in four sites. It is also attributable to lower inpatient costs – unlike the finding in the Washington trial. Among the five quasi-experimental studies that have looked for an association between hospital admissions (or hospital inpatient days) and being in IPS, three report no evidence of an association (28–30), whereas two others report fewer admissions for the IPS group (31–33). In one case, however, fewer admissions were found only among people with higher outpatient mental health service use (32).

There are a number of reasons why IPS might reduce hospital use. Vocational advisors may happen to observe,

for example, signs that their client is on the way to a crisis and alert his or her clinicians. Their relationship with a client may in and of itself have a therapeutic effect. Clients who do begin to work may experience an improvement in symptoms and self-esteem (34,35), which might in turn reduce hospitalizations. Studies that have considered the effects of working on overall treatment costs do suggest that, in clients who enter into work (which IPS facilitates but does not guarantee), there are reductions in treatment costs (36–38), and these are largely influenced by inpatient use.

In the present study, inpatient hospital use for the IPS group was reduced significantly only during the first 6 months; the difference essentially disappeared by the end of the follow-up period. Further analyses (not reported here) indicated considerable variability in the difference in inpatient costs between IPS and comparison groups across sites and over time. Indeed, both fixed effects and random effects regressions of inpatient costs over time, service and the interaction between the two, indicated an overall downward trend in hospitalization costs, but no difference in trend between IPS and usual services (p=0.34 and 0.44, respectively). The observed difference at 6 months could therefore be attributable to chance. In only one of the six sites (Groningen) did IPS generate numerically higher costs than the comparison intervention (but the difference was not significant). This was the site where IPS was implemented in the least effective way compared with usual services: it appears to represent an atypical experience.

Variations in vocational service costs across sites also bear comment. Not surprisingly, given the heterogeneity in traditional vocational services, the cost of comparison services varied widely across sites. The considerable (threefold) variation in costs of IPS services was more surprising, because the same unit costs were used to calculate those costs across sites, and because all sites achieved good or fair levels of fidelity to the IPS model (6). Differences in infrastructure may account for some of the variability in IPS intervention costs.

The cost-effectiveness analysis was conducted from the perspective of the health care system, with costs measuring only health and social care inputs. Although effectiveness was gauged in terms of employment gained, this is a valid aim for community mental health services. When we turned to the cost-benefit arguments, we attached an estimate of the societal value of the employment gained but we did not attempt to attach monetary values to any other clinical or quality of life gains. Even so, this partial analysis demonstrated the broader social value of the IPS approach.

Limited sample size for the cost analysis is a limitation of the study, although one that is difficult to avoid given the complexity (and cost) of conducting studies such as this. The use of UK unit costs for all study sites may also be viewed as a limitation of the study, but this could not be avoided given the

absence of country-specific information to compute valid and comparable unit costs in all sites. Moreover, using country-specific unit costs introduces further extraneous variation that would have to have been adjusted for in the analyses. In fact, unit costs for health services and social care are largely driven by local wage rates. Lower wage rates in some sites, such as Sofia, would tend to reduce all unit costs more or less proportionately, so that it is unlikely that the observed differences in cost between IPS and comparison services would alter very much in magnitude. Another limitation is that it was not possible to take into account changes in the cost of welfare benefits linked to unemployment benefits or changes in income tax contributions. For a cost-effectiveness analysis, these would be irrelevant as they are transfer payments, but they would be of interest to government that has to fund them.

When public bodies seek to introduce policies to improve employment rates among people with mental health needs, they do not tend to devote much attention to people with the most severe needs. This may be because of the comparatively small numbers of people involved, and perhaps because policy-makers do not believe much can be done at an affordable cost. However, this six-country European study paints a rosier picture.

This is not merely a case of helping people move from unemployment to employment, fundamentally important though that is, but of addressing needs of people facing long-term disadvantage. Employment is both a source of income and independence and a major contributor to social inclusion, self-determination, and recovery. IPS appears to provide an effective and cost-effective means of helping many people with a serious mental illness to come closer to achieving their employment goals.

References

1. Marwaha S, Johnson S, Bebbington P et al. Rates and correlates of employment in people with schizophrenia in the UK, France and Germany. Br J Psychiatry 2007;191:30-7.
2. Department of Health. No health without mental health: a cross-government mental health outcomes strategy for people of all ages. London: Department of Health, 2011.
3. European Commission. Improving the mental health of the population: towards a strategy on mental health for the European Union. Brussels: Commission of the European Union, 2005.
4. Bond GR, Drake RE, Becker DR. An update on randomised controlled trials of evidence-based supported employment. Psychiatr Rehabil J 2008;31:280-90.
5. Corrigan PW, Mueser KT, Bond GR et al. Principles and practice of psychiatric rehabilitation: an empirical approach. New York: Guilford, 2008.
6. Burns T, Catty J, Becker T et al. The effectiveness of supported employment for people with severe mental illness: a randomised controlled trial. Lancet 2007;370:1146-52.
7. Latimer E, Lecomte T, Becker D et al. Generalisability of the individual placement and support model of supported employment: results of a Canadian randomised controlled trial. Br J Psychiatry 2006;189:65-73.
8. Killackey E, Jackson HJ, McGorry PD. Vocational intervention in first-episode psychosis: individual placement and support v. treatment as usual. Br J Psychiatry 2008;193:114-20.
9. Wong K, Chiu R, Tang B et al. A randomised controlled trial of a supported employment program for persons with long-term mental illness in Hong Kong. Psychiatr Serv 2008;59:84-90.
10. Bond GR, Drake RE, Becker DR. Generalizability of the Individual Placement and Support (IPS) model of supported employment outside the US. World Psychiatry 2012;11:32-9.
11. Bond GR, Becker DR, Drake RE et al. A fidelity scale for the Individual Placement and Support model of supported employment. Rehabil Couns Bull 1997;40:265-84.
12. Catty J, Lissouba P, White S et al. Predictors of employment for people with severe mental illness: results of an international six-centre randomised controlled trial. Br J Psychiatry 2008;192:224-31.
13. Chisholm D, Knapp M, Knudsen HC et al. Client Socio-demographic and Service Receipt Inventory – EU version: development of an instrument for international research. Br J Psychiatry 2000;177(Suppl. 39):28-33.
14. Curtis L, Netten A. Unit Costs of Health and Social Care 2003. Canterbury: PSSRU, University of Kent, 2003.
15. SPSS Inc. SPSS for Windows Release 12.0.1. SPSS Inc, 1989-2001.
16. StataCorp LP. STATA 8.2 for Windows. StataCorp LP, 1985-2004.
17. StataCorp LP. STATA 10.1 for Windows. StataCorp LP, 1985-2009.
18. van Hout BA, Al MJ, Gordon GS et al. Costs, effects and C/E-ratios alongside a clinical trial. Health Econ 1994;3:309-19.
19. Bowling J, Coleman N, Wapshott J et al. Destinations of benefit leavers. London: Department for Work and Pensions, 2004.
20. Knapp M, Chisholm D, Leese M et al. Comparing patterns and costs of schizophrenia care in five European countries: the EPSILON study. Acta Psychiatr Scand 2002;105:42-54.
21. Drake RE, Bond G, Thornicroft G et al. Mental health disability: an international perspective. J Disability Policy Studies (in press).
22. Knapp M, Mangalore R, Simon J. The global costs of schizophrenia. Schizophr Bull 2004;30:279-93.
23. Howard L, Heslin M, Leese M et al. Supported employment: randomised controlled trial. Br J Psychiatry 2010;196:404-11.
24. Latimer E. An effective intervention delivered at sub-therapeutic dose becomes an ineffective intervention. Br J Psychiatry 2010; 196:341-2.
25. Dixon L, Hoch JS, Clark R et al. Cost-effectiveness of two vocational rehabilitation programs for persons with severe mental illness. Psychiatr Serv 2002;53:1118-24.
26. Heslin M, Howard L, Leese M et al. Randomised controlled trial of supported employment in England: 2 year follow-up of the Supported Work And Needs (SWAN) study. World Psychiatry 2011;10:132-7.
27. Clark RE, Xie H, Becker DR et al. Benefits and costs of supported employment from three perspectives. J Behav Health Serv Res 1998;25:22-34.

28. Bailey E, Ricketts S, Becker D et al. Do long-term day treatment clients benefit from supported employment? Psychiatr Rehab J 1998;22:24-9.

29. Becker DR, Bond GR, McCarthy D et al. Converting day treatment centers to supported employment programs in Rhode Island. Psychiatr Serv 2001;52:351-7.

30. Clark RE, Bush PW, Becker DR et al. A cost-effectiveness comparison of supported employment and rehabilitative day treatment. Admin Policy Ment Health 1996;24:63-77.

31. Drake RE, Becker DR, Biesanz JC et al. Rehabilitative day treatment vs. supported employment: I. Vocational outcomes. Commun Ment Health J 1994;30:519-31.

32. Drake R, Becker D, Biesanz JC et al. Day treatment vs supported employment for persons with severe mental illness: a replication study. Psychiatr Serv 1996;47:1125-7.

33. Henry AD, Lucca AM, Banks S et al. Inpatient hospitalisations and emergency service visits among participants in an Individual Placement and Support (IPS) model program. Ment Health Serv Res 2004;6:227-37.

34. Bond G, Resnick S, Drake R et al. Does competitive employment improve nonvocational outcomes for people with severe mental illness? J Consult Clin Psychol 2001;69:489-501.

35. Mueser KT, Becker DR, Torrey WC et al. Work and non-vocational domains of functioning in persons with severe mental illness: a longitudinal analysis. J Nerv Ment Dis 1997;185:419-26.

36. Bush P, Drake R, Xie H et al. The long term impact of employment on mental health service use and costs for persons with severe mental illness. Psychiatr Serv 2009;60:1024-31.

37. Perkins DV, Born DL, Raines JA et al. Program evaluation from an ecological perspective: supported employment services for persons with serious psychiatric disabilities. Psychiatr Rehabil J 2005;28:217-24.

38. Schneider J, Boyce M, Johnson R et al. Impact of supported employment on service costs and income of people with mental health needs. J Ment Health 2009;18:533-42.

"We lost all we had in a second": coping with grief and loss after a natural disaster

Samanthika Ekanayake[1], Martin Prince[2], Athula Sumathipala[3], Sisira Siribaddana[3], Craig Morgan[4]

[1]Health Systems and Health Equity Research Group, Centre for Addiction and Mental Health, Toronto, Canada; [2]Section of Epidemiology, Institute of Psychiatry, King's College, London, UK; [3]Institute for Research and Development, Colombo, Sri Lanka; [4]Section of Social Psychiatry, Institute of Psychiatry, King's College, London, UK

Natural disasters cause immense suffering among affected communities. Most occur in developing countries, which have fewer resources to respond to the resulting traumas and difficulties. As a consequence, most survivors have to rely on their own coping resources and draw from what support remains within family, social networks and the wider community to manage and deal with their losses and consequent emotional distress. Taking the 2004 Asian tsunami as an example, this article reports findings from a qualitative study designed to investigate how survivors responded in Sri Lanka, and the range of coping strategies adopted and resources mobilized. In-depth interviews were conducted with 38 survivors purposively sampled from the Matara district of southern Sri Lanka. Survivors' accounts emphasized the importance of extended supportive networks, religious faith and practices, and cultural traditions in facilitating recovery and sustaining emotional well-being. Government and external aid responses that promoted these, through contributing to the re-establishment of social, cultural, and economic life, were particularly valued by participants. Recourse to professional mental health care and Western psychological interventions was limited and survivors preferred to seek help from traditional and religious healers. Our findings tentatively suggest that long-term mental health following disaster may, in the first instance, be promoted by supporting the re-establishment of those naturally occurring resources through which communities traditionally respond to suffering.

Key words: Natural disaster, trauma, resilience and coping, emotional distress

Natural disasters are occurring globally at an increasing rate, especially due to the upward trend of hydro-meteorological disasters (1,2). Most of these natural disasters are unpredictable and rapid; consequently, often many thousands experience immense losses with considerable subsequent social and economic hardships. However, the World Health Organization has estimated only 5–10% of those in disaster affected communities develop serious emotional and mental health problems which need intervention; many more, it seems, have the resources to mitigate the impact of grief and trauma, perhaps drawing on internal resilience and external supportive resources (3). The majority of natural disasters occur in developing countries, where the availability of professional mental health services is limited. This poses important questions about how affected communities and individuals manage and cope with trauma and loss in the aftermath of disaster, and the appropriate place of mental health services, especially psychological interventions, in this.

Coping and resilience have been among the key interests of researchers who have studied disaster affected communities (4,5). The ability to cope and recover from loss is determined by a collection of factors (6) in pre-disaster, within-disaster, and post-disaster periods (7). Personal strength, religious belief and faith, and external support appear to play key roles in coping after a massive disaster or major life events (8,9). A large number of studies on coping have been conducted with hurricane Katrina survivors in the United States (10–13). A few studies are available with 2004 Asian tsunami affected populations (14,15), 2008 Chinese earthquake survivors (16), and survivors of 1998–2005 floods in Bangladesh (17). All suggest coping strategies mediate the impact of disaster.

Most of the studies to date have used quantitative methods, with standardized scales to assess coping. Only a small amount of research has used in-depth methods to explore in more detail how disaster survivors manage and cope (5). The only qualitative study with 2004 Asian tsunami survivors was conducted in Tamil Nadu, India (4). The authors identified a range of strategies used by survivors (e.g., accepting loss and tolerance of hardships, social gatherings to remember the dead, and organizing remarriages). The most common form of coping was spiritual (i.e., requiems, rituals, and spiritual help-seeking). However, our understanding of how survivors, particularly in non-Western settings, manage and mobilize resources to cope with disaster remains limited.

Using in-depth qualitative methods, we set out to investigate the coping strategies adopted by survivors of the 2004 Boxing Day tsunami in Sri Lanka to deal with a range of psychological and social problems.

METHODS

The setting of this study was the Matara district of southern Sri Lanka, where 19,744 families were affected, 2235 were displaced, and 1342 people died (18). We conducted field work from October 2007 to September 2008 in selected Grama Niladari divisions (the lowest administrative unit in Sri Lanka, usually comprising two to three villages). Ethical approvals were obtained from

the ethics committees of King's College London, UK and the Faculty of Medical Sciences, University of Sri Jayawardanepura, Sri Lanka.

This was primarily a qualitative study, based on individual in-depth interviews with survivors, with researcher observations and secondary official sources providing additional data. In addition, we used two screening tools – the PRIME-MD Patient Health Questionnaire (PHQ-9, 19) and the Composite International Diagnostic Interview (CIDI, 20), Section K – to provide standardized information on the current mental health status of participants.

Participants aged 18 years and older were purposively selected based on a predesigned sampling grid, which was developed to ensure that we included both men and women, different age groups, and people with a variety of experiences of the tsunami (i.e., family member dead or missing, or otherwise affected, displaced or resettled). To increase the heterogeneity of the sample, various religious, ethnic, and socioeconomic groups were also selected. To identify potential participants, a list of affected people was obtained from village level government officers. The final sample consequently consisted of 38 adult survivors from 12 Grama Niladari divisions from Matara and Weligama Divisional Secretariats, of whom 21 were women and 17 were men.

In-depth interviews with participants were based on a topic guide developed specifically for this project. The key topics included experiences on the day of the tsunami, the nature of any losses or consequences due to the event, responses and ways of coping, and the extent of available support (both informal and formal). Interviews were performed at participants' homes or at a place of their choice. All of them were conducted in Sinhalese by the first author (SE) and were recorded with the permission of the participants. Field notes were taken by a research assistant. Soon after completion, the interviews were transcribed verbatim into Sinhala and then translated into English. The first author translated all the interviews initially and then each of these translations was separately discussed against its Sinhala transcript with at least two people who have done extensive writing in both Sinhala and English, including the third author (AS). In addition, observation and secondary sources were used.

The interview transcripts were analyzed using thematic analysis, with some elements of grounded theory (i.e., use of constant comparison to move beyond identification of individual themes to explore connections between themes, as a basis for more theoretical formulations). The analysis was conducted in the following steps: detailed reading and annotating of transcripts to identify initial themes or codes; defining and redefining of codes; development of higher order, more abstract, categories; refinement of codes and categories, through comparing and contrasting; and development of an overarching thematic framework. Coding was jointly done by the first (SE) and senior author (CM) and codes were further discussed with the research team. When the coding frame was finalized, we assessed intercoder reliability for 20 transcripts and achieved more than 90% agreement. Data analysis was aided by NVivo7 (21).

RESULTS

The study group comprised 21 women and 17 men aged 18–65 years. Among the 38 participants, 36 were Sinhalese and two were Muslim. We could not find any Tamil participant from the study area. Most were Buddhist (34) and two each were Catholic and Muslim. Five participants sustained severe physical injuries during the tsunami and 11 experienced moderate injuries. The study group included 20 individuals who lost one or multiple family members, three of whom never recovered their relatives' bodies. To some degree almost everyone in the study group had been displaced and experienced loss of property, livelihood or household items.

Results from the PHQ and CIDI indicated that five participants were positive for post-traumatic stress disorder and somatoform disorders, and three were positive for major depression, using standard diagnostic criteria. Seven participants reported other depressive and/or anxiety symptoms without meeting criteria for disorder during the time of data collection.

Participants described a range of strategies that were adopted at different points following the tsunami to manage and respond to the immediate and lasting emotional trauma and practical devastation they and their communities experienced. These strategies are described in the following sections.

Resilience and faith

Many participants felt that the impetus to overcome distress should come from themselves and highlighted the importance of self-motivation and adopting a positive outlook. In this, emotional distress was conceived in terms of lack of stability and balance, with the consequent goal being to re-establish "emotional stability" or a "firm state of mind". The idea of hopefulness and motivation to overcome distress and traumatic memories was shared by most participants.

"I look at everything positively. I had that strength all the way and that is my nature. I don't have unattainable expectations in life and I don't despair on any circumstance".

Many, particularly women and older participants, talked about the importance and relevance of their faith

and religion, and sought to make sense of their losses through their religious faith and beliefs. For example, Buddhists often commented that the tsunami was an example of Buddha's preaching on tentativeness in life and nature.

"We lost all what we had in a second. Soon after the incident we were really hopeless and lost all our hopes for the future. Even now we don't go after sophisticated materials due to that experience. As it was shown in Buddha's preaching, now we understood the temporary nature of life".

The Catholic and Muslim participants were also convinced that God had helped them to escape from such a catastrophe ("it was God's will") and that God would protect them from any future disaster.

What is particularly noteworthy here is that these accounts all share an element of fatalism and ultimate trust in a higher authority that will provide protection against the unpredictable and uncontrollable. In this sense, the inner strength described by some individuals that enabled them to recover from the emotional trauma of the tsunami derives from faith and religious belief; this belief is reinforced as the events of the tsunami are made sense of by reference to (and therefore provide support for) the teachings of their faith.

There were nonetheless some for whom the tsunami did not make sense in relation to previously held beliefs. This was particularly common among those who had lost someone close. This may be a sign of hopelessness and anger that their faith had not spared them suffering and had not protected their family. One participant, for example, blamed God for being cruel to her and taking the life of her 6-year-old daughter.

"As stated by all the religions if there is any such supernatural power why couldn't they save the lives of those victims? If so what is the purpose in having a religion. Even though I am a Catholic my daughter went to temple every poya day [full moon day] with my neighbors. No religion was able to save her life".

Moreover, the data tentatively suggest that loss of faith was common among those with long-term mental health problems, and it may be that loss of faith following such trauma deprives individuals of a culturally rooted means of making sense of misfortune and in doing so compounds the loss and suffering.

Sharing pain

Many participants, especially women, talked of discussing their problems and pain with someone close or trustworthy. Talking to others, at one level, served the practical purposes of eliciting advice and providing company. But its value extended beyond this. For most participants, it was more importantly a way to "get pain out the chest", perhaps even a form of witnessing in which comfort was sought and gained through the process of sharing experiences of suffering.

"We were able to come to terms with the situation as all of us were together and there was a mutual exchange of ideas. Every one shared their experiences".

This extended beyond close friends and family. For example, talking to religious leaders, such as village Buddhist monks, priests of the churches, or *maulavi* (leaders) of mosques, was commonly mentioned, especially by older participants. Various meetings organized by village level relief organizations were also identified as common opportunities to share experiences. The use of the word "sharing" to describe these activities is notable, and at the risk of over-interpretation it hints at the importance of mutual exchange in the disclosure of distress, at the comfort that can derive from knowing others have had similar experiences. After all, everyone in the community, to some extent, was affected by the tsunami and could at some level relate to the distress caused.

Almost everyone in the study group talked about the significance of informal social resources such as interpersonal networks of family, friends, and neighbors in coping with the emotional and practical consequences of the tsunami. The sense of community solidarity and strong extended family structure in traditional Sri Lankan society was highlighted by many as underpinning this. Those with a family member who had died or was missing frequently described receiving considerable support from extended family, many of whom helped practically by assuming tasks and duties performed by the deceased.

"My father was the only income earner of the family... So we badly felt it. But it is a relief that our elder brother and uncles all help us. Our elder brother started working in my uncle's hotel after my father's death and he earns for the family. Also other members of my mother's family help us, they give us money. My brother takes us to night tuition classes as our farther did in the past".

This again extended beyond family and there were many accounts that suggested strong community cohesion after the tsunami. The support provided by religious leaders and nonaffected community members was greatly acknowledged. Most of the Buddhist temples, Catholic churches, and mosques organized memorial

services and other religious programs to help overcome ongoing fear and sadness in the affected communities.

Becoming engaged

Many participants commented that keeping busy was a successful way of dealing with stress. As one participant put it: "*When the mind is filled with active feelings, there is no room for the pain to become entrenched*". In line with this, many engaged in work and leisure activities and religious rituals as a way of providing relief from their troubles. In the early stages, much effort was directed toward helping others practically; many acknowledged that villages were more united than before the tsunami and helped each other with cleaning up, searching for missing people, and mourning. There were many sports activities, drawing sessions, and dramas organized for children. Many of these activities, then, fulfilled a dual purpose, both as practical necessities (cleaning up, earning income, and passing time) and psychological and emotional distraction. The community nature of many of the activities further suggests that they formed part of a wider communal response – a process of re-establishing social, cultural, and economic life following the devastation of the tsunami.

Participants' accounts suggested that engagement in religious activities increased notably during the early days after the tsunami. For example, Buddhists frequently talked about how they performed religious rituals such as *Bodhi Pooja* (bathing at the foot of the Bo tree with water and milk), lighting of oil lamps, and chanting of and listening to *Pirith* (Buddha's preaching) as a means to gain prosperity and avoid hardships or evil forces, while meditation (*Anapana sathi* by Buddhists) was also mentioned as a way of gaining relaxation of mind. Indeed, religious practices and rituals have been ubiquitous responses to the tsunami, providing both individual comfort and opportunities for community reintegration and regeneration.

Private grief, public mourning

Creating monuments in the place where a person is cremated is a common way of paying respect to the dead in the Sri Lankan culture. However, in the aftermath of the tsunami, this was unattainable, as many of the dead were missing and some were buried in mass burial sites. To deal with this – both the loss and the difficulties in fulfilling culturally sanctioned and expected rituals – individuals and families adopted a number of alternative strategies to demonstrate respect and deal with memories of loved ones. Collecting reminders, such as photographs and souvenirs, was common and enlarged photographs of the dead have been posted in the front area of many households. One participant showed letters written by his wife and mentioned that he still keeps them to remember the "good old days". Construction of monuments in the name of the dead was also common, and some participants mentioned that they spent the government's death payments solely on such activities.

"We tended to have stronger faith in our religious beliefs. So we performed lots of religious activities for our dead son. All the financial compensations we received on behalf of him were used for these religious activities and we didn't spend anything for our personal expenses".

These monuments transform private grief into a form of public mourning, in which respect and love for the dead is outwardly displayed. In effect, the loss and sorrow is shared with the community and in the process managed and, ultimately, healed.

Outside aid

The overwhelming majority of participants, in talking about how they managed and recovered following the tsunami, talked primarily about drawing from their own resources or from supports that predated the tsunami (i.e., family, friends, churches and temples). When participants talked about government responses and external aid, views were mixed. The supportive environment provided by the government and humanitarian agencies was cited by some participants as a major factor in the return to normalcy. Some cultural events such as New Year festivals and religious events such as food donations were organized to promote community participation and generate aid. This noted, many participants were still critical of what they saw as disorganized relief distribution together with unfair compensation and suggested this compounded suffering and caused additional burden.

In the aftermath of the tsunami, some outside organizations provided psychological interventions for affected people. Despite the fact that the number of mental health professionals in Sri Lanka is limited (the precise number is unknown), "counselors", "psychologists", and other mental health professionals were attached to various organizations. Interestingly, the narratives of participants indicated that the demand for such services was extremely low, as people were more interested (when seeking external help) in getting support from traditional healers or traditional physicians, which included astrologers, diviners, oracles, traditional doctors (especially Ayurvedic doctors), and other types. The most common activities reported were *thel methurum* (application of oils to body and head to remove evil spirits), *dehi kepili*

(use of spiritual powers to remove evil spirits), and performing services to Gods and Goddesses. Participants reported that getting support from traditional healers was commonly used to overcome sleeping problems, fearful dreams, and screaming during sleep or flashbacks.

DISCUSSION

Broadly, the strategies and supportive resources described in participants' accounts of the aftermath of the tsunami can be distinguished between those that were predominantly individual (or inward looking and private; i.e., inner-strength and positivity) and those that were interpersonal or social (or outward looking and public; i.e., emotional and practical support from family, friends and others in the community, including religious leaders and institutions). The form that each of these strategies took was colored by the predominant sociocentric and spiritual character of Sri Lankan society, such that apparently individual and private strategies for coping often became public expressions of loss, respect, and resilience (e.g., creating monuments to lost relatives). Similarly, individual responses were rooted in predominant beliefs about the origins of misfortune, and personal fortitude was often derived from fatalistic beliefs in the tentativeness of life and God's will. Therefore, coping responses were made possible and shaped by cultural and social norms and practices in Sri Lanka.

Participants in this study were sampled through a list given by village level government officers, which may have led to a selective group. This, though, was unavoidable. Accessing villages through government officers was essential to successfully conduct the data collection, as many were suspicious of unknown people collecting personal information. To minimize this potential problem, we tried to select participants from large lists provided by the government officers in accordance with our sampling grid. Some views may have been more prominent because of the sampling approach. For example, although the sample recruited in this study broadly reflected the religious composition of the Matara district and the Sri Lankan population, the narratives on religious coping strategies were dominated by the views of Buddhists (89% of the sample) compared with Muslims (8%) and Catholics (5%).

Many of the strategies identified in our study resemble problem-focused and emotion-focused coping mechanisms described by Lazarus and Folkman (22). There has been debate about which type of strategy (problem-focused or emotion-focused) is most effective (23). Our data cannot address this question. What the data do, however, is suggest that this kind of dichotomy is overly schematic and ignores the observation that individuals commonly use both kinds of strategies, together and at different points. What is more, perhaps the most striking finding from this study is the degree to which coping with (or responding to) the multiple traumas and problems created by the tsunami was social and public. For example, sharing experiences or feelings with others, as a kind of witnessing or collective consoling, was the most common response cited by the participants in our study. This has also been identified in previous studies conducted with disaster victims (4,5) and challenges the notion that coping is primarily individual.

Our findings further indicate that religion provided the primary framework for making sense of and responding to the tsunami, either as an active response (i.e., engaging in religious performances) or an emotional resource (i.e., faith). The role of religious or spiritual beliefs and how they contribute to recovery following natural disaster has not been much studied in the past (24), especially in South Asian cultures (25). The studies that have been conducted in Western countries with disaster survivors indicate that strong religious faith and beliefs are linked with lesser symptoms of emotional distress (11,13), whereas negative religious coping is often associated with greater distress. This was supported by our findings, in that those who talked of "loss of faith" appeared to suffer more long-term emotional distress. According to many participants, some of the religious practices contributed to relaxation and emotional well-being. The limited number of studies examining the impact of religious and traditional healing on coping has suggested positive effects, especially among non-Western communities (26). This indicates the importance of proper recognition and understanding of social and religious interpretations of coping, adaptive capacity, and resilience in different cultures.

Participants often emphasized the importance of cohesive communities and extended families as sources of help. The strength of social support in enabling individuals to cope with emotional distress consequent on trauma has been commonly identified in previous studies conducted in both Western and non-Western countries, and these have further highlighted the risk of long-lasting mental health problems in the absence of support (4,9). In line with previous findings, the results of this study indicate that a number of informal and formal sources, such as official organizations, mental health professionals, extended family members and friends, religious leaders, and school teachers, can provide psychological and practical support. In collectivist or sociocentric societies, emotional interdependence and protection are much encouraged (27). However, extreme natural disasters, such as the Asian tsunami, that affect entire communities can severely impact on the availability of otherwise naturally occurring supports, leaving individuals and communities struggling to cope – in the early days, desperately so.

Provision of professional psychological support for traumatized population has a long history, beginning at the very least with First World War veterans (28). The findings from this study suggest that availability of professional psychological support was minimal during the post-tsunami period in Sri Lanka. In a country with 21 million people and just 25 specialist psychiatrists (29) and no practicing clinical psychologists, the only available option for a majority of the survivors was relying on their own social, cultural, and religious supports. However, the question arises of whether such support is sufficient when whole communities are affected during a huge disaster like a tsunami or in situations where the degree of collective trauma engulfs the moral strength of families and communities, and if so what form this support should take. Many of the participants in our study neither placed much value on getting professional support nor did they conceive of the stress, grief, or worries as illnesses or conditions which needed professional help. Consulting a mental health professional is still uncommon among the majority of ordinary people in Sri Lanka, especially due to a high degree of stigma associated with being in contact with mental health services or to having someone who attends such services in the family.

There is only very limited research available on traditional healing practices which are used to treat those with mental health problems in Sri Lanka. Somasundaram et al (30) indicate that most of the traditional healers have received training and developed skills in some kind of healing practice. Many traditional healing practices in Sri Lanka are a combination of Ayurveda, Siddha, and local and spiritual practices. Usually members of the patient's family are present during the healing sessions – a marked contrast to Western psychotherapy in which families are usually excluded – which often comprise prayers, songs, and herbal massage. Usually Ayurvedic or local treatments include a lengthy conversation with the practitioner about patients' living circumstances or well-being along with a physical examination (31), and this provides opportunities for patients to discuss their social and emotional state in a less stigmatizing way.

There are numerous accounts of the support provided by religious organizations and leaders especially in offering shelter, collecting and distributing relief material, and implementing resettlement programs (25). The findings from this study extend this and suggest that religious leaders can play a significant role in providing emotional and psychological support.

Leaving aside the obvious need for immediate material and practical aid to assist those injured and displaced by disaster, the findings from this study suggest that long-term mental health may be promoted by supporting the re-establishment of those naturally occurring resources through which communities traditionally respond to suffering. In the Sri Lankan context, facilitating reunion of affected family members during the period of displacement and allowing them to live in close proximity in resettlement programs may have been a simple achievable step to ensuring access to informal support for those most affected. Along similar lines, rehabilitation efforts that seek to re-establish social and cultural customs and events may create opportunities for the kind of sharing and witnessing that, according to many participants in this study, can promote recovery. Conversely, relief efforts that are perceived to be unfair and corrupt can have the opposite effect of eroding the very cohesiveness of communities that could facilitate recovery – or at least this appears to be the case in Sri Lanka.

Concerning psychological interventions and mental health care, the data from this study suggest that these cannot simply be imported into settings unfamiliar with, and perhaps distrustful of, the individualized model of Western psychotherapy and psychiatry. Going further, the accounts of participants in this study support the Inter-Agency Standing Committee (IASC) guidelines on mental health and psychosocial support in emergency settings (32). It is imperative to consider and better understand local cultural beliefs about misfortune, mental health, and health before designing any mental health intervention. Further, where, as is the case in Sri Lanka, mental health services are underdeveloped, the provision of services in the aftermath of disaster may meet with limited uptake. The issue is, then, broader and concerns the development of mental health services outside disaster situations. Indeed, it is arguable that to focus on provision of mental health care during times of disaster (important and essential as this may be) misses the point that such provision, in countries like Sri Lanka, will inevitably falter while ever mental health care in general remains marginal. What this means is that, when disaster does strike, already devastated communities will have to draw from what limited resources remain to protect the mental health of those most affected.

References

1. Scheuren JM, le Polain O, Below R et al. Annual Disaster Statistical Review: the numbers and trends 2007. Brussels: Centre for

Research on the Epidemiology of Disasters and United Nations International Strategy for Disaster Reduction, 2008.

2. Moszynski P. Death toll climbs and healthcare needs escalate in Japan. BMJ 2011;342:d1859.

3. Ashraf A. Tsunami wreaks mental health havoc. Bull World Health Organ 2005;83:405-6.

4. Rajkumar AP, Premkumar TS, Tharyan P. Coping with the Asian tsunami: perspectives from Tamil Nadu, India on the determinants of resilience in the face of adversity. Soc Sci Med 2008; 67:844-53.

5. Ibañez GE, Buck CA, Khatchikian N et al. Qualitative analysis of coping strategies among Mexican disaster survivors. Anxiety Stress Coping 2004;17:69-85.

6. Gottlieb BH. Conceptual and measurement issues in the study of coping with chronic stress. In: Gottlieb BH (ed). Coping with chronic stress. New York: Plenum Press, 1997:3-42.

7. Eyre A. Literature and best practice review and assessment: identifying people's needs in major emergencies and best practice in humanitarian response. Report commissioned by the UK Department of Culture Media and Support, 2006.

8. Norris FH, Friedman M, Watson PJ. 60,000 disaster victims speak: Part II. Summary and implications of the disaster mental health research. Psychiatry 2002;65:240-60.

9. Somasundaram D. Collective trauma in northern Sri Lanka: a qualitative psychosocial-ecological study. Int J Ment Health Syst 2007;1:5.

10. Henderson T, Roberto KA, Kamo Y. Older adults' responses to Hurricane Katrina: daily hassles and coping strategies. J Appl Gerontol 2010;29:48-69.

11. Newton AT, McIntosh DN. Associations of general religiousness and specific religious beliefs with coping appraisals in response to Hurricanes Katrina and Rita. Mental Health Relig Cult 2009; 12:129-46.

12. Salloum A, Lewis ML. An exploratory study of African American parent-child coping strategies post-hurricane Katrina. Traumatology 2010;16:31-41.

13. Wadsworth ME, Santiago CD, Einhorn L. Coping with displacement from Hurricane Katrina: predictors of one-year post traumatic stress and depression symptom trajectories. Anxiety Stress Coping 2009;22:413-32.

14. Hollifield M, Hewage C, Gunawardena CN et al. Symptoms and coping in Sri Lanka 20–21 months after the 2004 tsunami. Br J Psychiatry 2011;192:39-44.

15. Leavy B, Slade M, Ranasinghe P. Casual thinking after a tsunami wave: Karma beliefs, pessimistic explanatory style and health among Sri Lankan survivors. J Relig Health 2009;48:38-45.

16. Yang J, Yang Y, Liu X et al. Self-efficacy, social support, and coping strategies of adolescent earthquake survivors in China. Soc Behav Pers 2010;38:1219-28.

17. Paul SK, Routray JK. Flood proneness and coping strategies: the experiences of two villages in Bangladesh. Disasters 2009;34:489-508.

18. National Disaster Management Centre. Tsunami situation report. Sri Lanka: National Disaster Management Centre, 2005.

19. Spitzer R, Kroenke K, Williams J. Validation and utility of a self-report version of PRIME-MD: the PHQ Primary Care Study. JAMA 1999;282:1737-44.

20. World Health Organization. Composite International Diagnostic Interview (CIDI). Geneva: World Health Organization, 1990.

21. QSR International Pty Ltd. NVivo qualitative data analysis software. Version 9. QSR International Pty Ltd, 2010.

22. Lazarus RS, Folkman S. Stress appraisal and coping. New York: Springer, 1984.

23. Hobfoll SE. The ecology of stress. New York: Hemisphere, 1988.

24. Davidson JRT, Connor KM, Lee LC. Beliefs in Karma and reincarnation among survivors of violent trauma: a community survey. Soc Psychiatry Psychiatr Epidemiol 2005;40:120-5.

25. de Silva P. The Tsunami and its aftermath in Sri Lanka; explorations of a Buddhist perspective. Int Rev Psychiatry 2006;18: 281-7.

26. Somasundaram D. Using cultural relaxation methods in post-trauma care among refugees in Australia. Int J Cult Ment Health 2010;3:16-24.

27. Bhugra D, van Ommeren M. Mental health, psychosocial support and the tsunami. Int Rev Psychiatry 2006;18:213-6.

28. Alexander DA. Early mental health intervention after disasters. Adv Psychiatr Treat 2005;11:12-8.

29. Siriwardhna C, Sumathipala A, Siribaddana S et al. Reducing the scarcity in mental health research from low and middle income countries: a success story from Sri Lanka. Int Rev Psychiatry 2011;23:77-83.

30. Somasundaram D, Sivayokan S, deJong J et al. Mental health in the Tamil community. Jaffna: Transcultural Psychosocial Organization, 2000.

31. Grønseth AS. Experiences of tensions in re-orienting relves: Tamil refugees in northern Norway seeking medical advice. Anthropol Med 2006;13:77-98.

32. Inter-Agency Standing Committee. IASC guidelines on mental health and psychosocial support in emergency settings. Geneva: Inter-Agency Standing Committee, 2007.

Is the concept of "dimension" applicable to psychiatric objects?

GERMAN E. BERRIOS[1], IVANA S. MARKOVÁ[2]

[1]Chair of Epistemology of Psychiatry, Robinson College, University of Cambridge, Cambridge, CB3 9AN; [2]Hull York Medical School, University of Hull, Hull, HU6 7RX, UK

Much has been written about the application of the concept of "dimension" to psychiatry. For example, it constituted one of the early desiderata for the construction of DSM-5, and as late as in 2006 great interest was being expressed in the "dimensional aspects of psychiatric diagnosis" and their "clinical and scientific feasibility" (1). Although this enthusiasm seems to have somewhat waned, confusion remains in various quarters as to the relevance of the concept of dimension to the psychiatric disciplines.

WHAT ARE DIMENSIONS?

There are at least three meanings to "dimension". The central and original one concerns the act of measuring ("a mode of linear measurement in a particular direction") and the magnitudes thereby obtained. By derivation, features of objects susceptible to measurement started to be called dimensions ("the three dimensions of a triangle, a multidimensional space"). Lastly, and by analogical usage, any components of any object or situation became their "dimensions" ("his acting added a new dimension to the play") (2). The act of "measuring" remains the definitory and operational meaning of "dimension". Since "dimensions" have been imported into psychiatry with the explicit purpose of making it more scientific (3), it has to be concluded that it is this central meaning and not the derivative and metaphorical usages that scientific psychiatrists are after.

OF WHAT OBJECTS CAN DIMENSIONS BE JUDICIOUSLY PREDICATED?

The world is populated by all manner of objects, some of which become, in the fullness of time, "epistemic things", that is, objects of science (4,5). In general, objects may exist in space, time, and combinations thereof. Much debate exists as to what constitutes an object. For the practical purposes of this article, object can be defined as "a thing or being of which one thinks or has cognition, as correlative to the thinking or knowing subject; something external, or regarded as external, to the mind" (2).

Objects thus defined have been classified into physical or natural (dogs, houses, rivers, clouds, murmurs, brains, atoms, etc.) and abstract or ideal (virtues, intentions, thoughts, beauty, gods, numbers, etc.). Natural objects do exist in space and time and this attribute makes them targets of measure-

ment. Dogs, tables or flowers are sufficiently stable in the space-time frame to be subject to dimensionalizing, that is, for measuring their spatial features with a standard yardstick and calling them "dimensions" (6). Conversely, abstract objects seem elusive to this type of manipulation (7,8). Indeed, many will feel that they are not susceptible to dimensionalizing at all. When confronted with a keen scientist developing a scale to "measure", say, the four cardinal virtues (temperance, prudence, courage and justice), most sensible people would agree that the concept of "measure" is being used in a metaphorical way, and that what the scientist is doing is undertaking a subjective form of "grading" and using numbers in a nonarithmetic way as labels for the levels of his scale.

Dogs, tables and virtues are not the only objects inhabiting the world. There are also complaints, moans, pains, afflictions, and mental symptoms; some of these are relevant to psychiatry. What sorts of objects are they? Some believe that the objects of psychiatry are like dogs or tables, that is, physical objects (some call them natural kinds) (9). Others may believe that they are ideal objects, like virtues or hopes. So far no *experimentum crucis* has been conceived that might "scientifically" help us to decide. In other words, the reasons for choosing whether psychiatric objects are natural or abstract are not scientific; hence, they are likely to be economic, social, ethical, or aesthetic.

In this article, we put forward the view that, in addition to natural and abstract objects, there are hybrid things in the world with shared features from both, and psychiatric objects are one of the best examples of that type. In principle, all mental symptoms are combinations of physical and ideal attributes; in practice, it is likely that the proportion of each will vary from symptom to symptom. Given that the meaning of all mental symptoms depends on their hybrid nature, reducing mental symptoms to either their physical (brain correlates) or ideal component (meaning) would impede understanding (10,11).

CAN PSYCHIATRIC (HYBRID) OBJECTS BE DIMENSIONALIZED?

Whether or not the concept of dimension applies to an object depends on its ontological structure, that is, the way in which it is made and framed in space and time. Because hybrid objects are *sui generis* in this regard, it is the duty of psychiatrists to determine whether they can be dimensionalized at all. As far as we know, such a task

has not yet been undertaken and yet the concept of dimension has been happily imported into psychiatry.

Why this haste is an important question. One explanation may be that such importation is encouraged by the (rife) belief that mental symptoms are physical objects (natural kinds) and hence that measuring their brain correlates is tantamount to measuring the symptoms in their totality (12). Another explanation may be that, under the influence of the old psychometry, it is believed that (even if they are hybrid objects) mental symptoms can be measured (captured) by "good quality" psychometry followed by sophisticated statistical analysis that might be able to convert numeric labels of intervals into real arithmetic "dimensions" (13). Whatever the explanation, we argue that the hybrid structure of mental symptoms needs to be fully understood before trying to dimensionalize them.

It has been claimed above that mental symptoms (e.g., hallucinations, delusions, obsessions, depressive mood) are hybrid objects, and that in each case the proportional contribution of physical and semantic attributes is likely to vary. To this, we could add that it is also likely that such proportions are liable to change for each symptom as it moves from the acute to the chronic state (i.e., hallucinations and delusions in the earlier episodes of psychosis may be structurally different from the "same" phenomena during the chronic stages) (14).

In practice, this would mean that the application of the same type of psychometric instrument to all mental symptoms, or to the same symptom during the early and chronic states of the disorder, may be unhelpful. This is because the reliability and validity of a scale depend on the stability in time and space of the object it purports to measure. In this sense, scales rely on the fact that the measurable "dimensions" or "components" of the object in question maintain their magnitude and combinatorial proportionality. If these were to change, then the scale would be useless at the second time round.

It must also be stated that, against renewed hopes that stable brain correlates will soon be discovered for each mental symptom, this has not happened so far and most (if not all) of the diagnostic scales in use obtain their information from verbal accounts given by patients, relatives and observers (15). By definition, this information is not susceptible to dimensionalizing. While there is nothing wrong with using subjective information to gain an idea of mental symptoms' severity or change, it would be wrong to call the number labels that we attach to levels of a scale dimensionalizing. The correct name for this act is grading.

THE CONCEPT OF GRADING

In the case of natural objects, dimensions are brought into being by the very act of measuring their spatial attributes. In the case of abstract objects, "dimensions" are used metaphorically as such objects are not framed in space.

Indeed, abstract objects are but constructs, bundles of qualities brought together within a given narrative (e.g., temperance, prudence, courage or justice). For example, moral narratives help to organize and categorize the actions of men and decide who possesses "more" of a particular virtue. To achieve these comparisons, throughout history, forms of evaluation have been developed to decide, for example, whether a given action is "unjust, partially just or just". How is this grading undertaken? On what is the subcategorization of its intervals based? Are its foundations stable enough to stand intrapersonal or transpersonal comparability? (16).

In this regard, the first point to make is that grading is carried out by means of categories that are external and reside not in the object itself (i.e., they are not internal to it) but in the eye of the evaluator. We have seen above that abstract objects (like virtues) are bundles of qualities brought together under a name. Grading consists in stretching each of these qualities along a score range marked by grading labels in which typically the higher scores are not mathematical multiples of the lower ones. Hence, they cannot really be considered as dimensions but only as grades.

Differentiating grading from measuring is essential. They are different mental operations and belong to different realms of knowledge. Grading is a form of evaluation and hence it accepts predicates such as fair, just, regular, consistent, benevolent, and so forth. Adjectives such as exact, reliable, valid, sensitive, specific, true, and the like cannot apply to it. Grading is always in the eye of the beholder, and the fact that some evaluators may be consistent in their evaluations (i.e., in attaching the same grading label to the same value or proportion) does not make grading into a form of measurement. Psychiatrists may want to use numbers as grading labels, but what they cannot do is perform mathematical operations on them. The point here is not simply one of semantics or misnomering. Believing that mental symptoms and disorders can be truly dimensionalized (i.e., measured) carries the erroneous implication that psychiatric objects are natural kinds. This implication has had (and has) as a consequence the undertaking of expensive and unproductive empirical research, and prevents the development of more useful approaches to psychiatry and its objects.

We argue that, in the current knowledge, psychiatric scales are no more than grading labels, and that the "dimensions" they purport to "measure" are no more than qualities stretched out along arbitrary ranges.

Given that: a) mental symptoms are hybrid objects, that is, composites of physical and abstract attributes, b) each mental symptom has a different structure when compared with another mental symptom and when compared with itself in the life history of a patient, and c) the physical components of mental symptoms are mostly unknown, it can be concluded that all we have

left to do is grade (evaluate) the subjective complaint as carried or communicated by the patient or the relative or as based on clinical observation. Evaluations are not related to measurement or dimensions; they are graded labels applied to the varied densities in which qualities may present themselves.

CONCLUSIONS

Dimensioning is a form of measurement that can only be applied to attributes of objects existing in space or time, that is, natural objects. Abstract objects can be evaluated, not measured. Hybrid objects (like mental symptoms) possess attributes from both types of objects and ideally they should be susceptible to measurement and evaluation. In practice, this is not the case, as the structure and components of mental symptoms remain mostly unknown. Little of substance is known about their natural properties, and, at any rate, their definition in most cases depends on their meaning, that is, the symbolic position they occupy in a given intersubjective context.

This implies that mental symptoms can only be evaluated (not measured). Evaluations are forms of grading by means of which qualities are stretched out and given grading labels capturing subqualities such as intensity, severity, duration, persistence, and so forth. As they are, grading labels can, in fact, be useful in the description and management of mental symptoms. However, they are not quantifiable. Neither the number-labels attached to them can be treated as real quantities nor can the evaluated qualities be called "dimensions". The same constraints apply to "mental disorders". The fact that throughout history mental symptoms have been made to cluster up in particular ways does not make the resulting clusters less qualitative. Like the mental symptoms that constitute them, mental disorders can only be graded or evaluated.

References

1. Helzer JE, Kraemer HC, Krueger RF et al (eds). Dimensional aspects in diagnostic classification. Arlington: American Psychiatric Association, 2008.
2. Oxford English Dictionary. Oxford: Oxford University Press, 2nd ed., 2009.
3. Cuthbert BN. Dimensional models of psychopathology: research agenda and clinical utility. J Abnorm Psychol 2005;114:565-9.
4. Rheinberger H-J. Towards a history of epistemic things. Stanford: Stanford University Press, 1997.
5. Daston L (ed). Biography of scientific objects. Chicago: University of Chicago Press, 2000.
6. Grossman R. Meinong's doctrine of the Aussersein of the pure object. Noûs 1974;8:67-82.
7. Lowe EJ. The metaphysics of abstract objects. J Philos 1995; 92:509-24.
8. Hale B. Abstract objects. Oxford: Basil Blackwell, 1987.
9. Wilkerson TE. Natural kinds. Aldershot: Avebury Press, 1995.
10. Berrios GE. Psychiatry and its objects. Revista de Psiquiatría y Salud Mental 2011;4:179-82.
11. Marková IS, Berrios GE. The epistemology of psychiatry. Psychopathology 2012;45:220-7.
12. Kendler KS, Zachar P, Craver C. What kinds of things are psychiatric disorders? Psychol Med 2011;41:1143-50.
13. Eysenck HJ. Classification and the problem of diagnosis. In: Eysenck HJ (ed). Handbook of abnormal psychology. London: Pitman, 1960:1-31.
14. Marková IS, Berrios GE. Mental symptoms: are they similar phenomena? The problem of symptom heterogeneity. Psychopathology 1995;28:147-57.
15. Marková IS, Berrios GE. Epistemology of mental symptoms. Psychopathology 2009;42:343-9.
16. Urmson JO. On grading. Mind 1950;59:145-69.

When children with autism become adults

FRED R. VOLKMAR, JULIE M. WOLF

Child Study Center, Yale University, New Haven, CT 06520, USA

Autism and related conditions have been described since the 1940s, but official recognition did not come until 1980, with the publication of the DSM-III. Early confusion centered on the validity of the condition, that is, whether it could be considered distinct from childhood schizophrenia. This confusion was clarified with work on clinical phenomenology and genetics of the two conditions. Specifically, differences in clinical features were identified, with autism being marked by profound social difficulties and very early onset relative to schizophrenia. It also became clear that autism was a strongly genetic disorder, distinct from schizophrenia.

Autism was associated, at least initially, with a rather poor outcome, with the earliest studies suggesting that about two-third of individuals, as adults, required institutional care (1,2). Early treatment approaches were centered on psychotherapy (often of parent and child), but gradually shifted as work indicated that structured behavioral and educational approaches were associated with better outcome, as was earlier diagnosis and intervention. It has become increasingly clear that greater public awareness, earlier intervention, and more effective management have had a major impact on the outcome of the condition (1).

OUTCOME IN AUTISM

There is some evidence that the core symptoms of autism abate to some degree in adolescence and young adulthood (3), with improvements in communication skills most common. Social impairments and repetitive behaviors tend to persist into adulthood. Further, not all individuals show improvements, and it is rare that individuals show gains to the extent that they no longer meet diagnostic criteria for autism spectrum disorders (ASD) (1-3).

With regard to neuropsychological functioning, intelligence quotient (IQ) is generally found to be stable over time. Adults with autism commonly have a range of challenges with regard to neuropsychological functions, including difficulties with social cognition, memory, executive functioning, and motor coordination, which can impact their ability to navigate the complexities of adult independence (4).

In terms of functional and daily living outcomes, early studies of individuals born in the 1970s and earlier consistently reported poor to very poor outcomes for the majority of subjects in adulthood (3,5,6). A minority of adults were found to live independently, with most remaining dependent on families. Likewise, a minority was employed or had attended college. The percentage of adults with autism who were found to have favorable outcomes ranged from 15 to 44% across studies (3,5). Early communication skills and level of cognitive functioning were found to be the strongest predictors of outcome, with those individuals with an IQ above 70 having the greatest likelihood of living independently. Some have also suggested that outcome is contingent on the amount of perceived social support available to the individual (7).

More recent studies have suggested a less bleak picture. Farley et al (8) found that outcomes had improved over earlier estimates, with half of their sample reporting "good" to "very good" overall outcomes. Likewise, Eaves and Ho (9) found about half of their sample to have "fair" to "good" outcomes, with the other half rated "poor" (but none rated "very poor"). The authors attribute this improvement to progress in early detection and intervention in recent years. However, with 50% of individuals still achieving poor outcomes, it is clear that additional supports and interventions are needed to further improve quality of life for adults with autism.

Outcomes with regard to social functioning suggest that difficulties making and maintaining friendships persist into adulthood. Twenty-five percent or less of adults with ASD were found to have true friendships (6,10). Predictors of participation in social activities in adulthood include greater independence in activities of daily living, better socialization skills, and greater number of services received. Peer victimization may remain a concern for young adults with ASD as well (11). Although sexual functioning develops normally, the social aspects of navigating sexual relationships and understanding appropriate sexual behavior present a challenge for many young adults. Impairments in social cognition and the tendency for rigid behaviors or obsessional interests can also result in legal challenges (e.g., failure to defer to authority figures, inappropriate online behaviors, stalking). Explicit education for young adults around these issues is important to ensure they understand the impact of their actions. Educating first responders is also critical for ensuring that these behaviors are not misunderstood.

The recognition of autism and severe social vulnerabilities in more cognitively able individuals was reflected in the inclusion of Asperger's disorder in DSM-IV and ICD-10. Both for Asperger's disorder and higher functioning autism, more and more individuals are able to seek higher educational and vocational training, although social and learning supports are often significant needs. Countries

vary considerably in the degree to which a disability like autism is eligible for special accommodations.

ISSUES OF DIAGNOSIS AND COMORBIDITY

Very early approaches to diagnosis centered on autism in young children. Over time, the need for a more developmental and life-span approach was recognized, for example, in DSM-III-R and particularly in ICD-10 and DSM-IV. The recognition of the broader range of the autism phenotype and of higher intellectually functioning individuals with severe and impairing social difficulties (e.g., those with Asperger's disorder) has raised important diagnostic issues that remain the topic of considerable debate and are reflected in the currently proposed DSM-5 approach. It remains to be seen how the latter will impact diagnostic practice.

Having a chronic condition like autism may predispose to other difficulties (11). The currently available literature does suggest increased rates of several clinical problems in adulthood, including anxiety and depression (both of which may respond to different treatment modalities). The limited treatment literature available specific to this age group and indeed the overall lack of research in adolescents and adults with autism remain significant obstacles for our understanding the best approaches to intervention.

CONCLUSIONS

The outcome in autism appears to have markedly improved over the past several decades – presumably reflecting a number of factors including earlier intervention and improved treatments. At the same time, many adults, even while living independently, need some supports, and a lack of treatment research and available services limits our knowledge of this population.

Often significant gaps exist between cognitive abilities and the capacity to generalize these to "real world" settings – this gap between intellectual function and adaptive skills can be quite significant and may be one of the factors that serve to predispose cases to other mental health problems. Many individuals are now attending college or vocational school, but can have persistent problems with social isolation and communication, which may also increase risk for anxiety and depression.

Research and service provision in this area remain extremely high priorities (11).

References

1. Volkmar F, Wiesner L. A practical guide to autism. Hoboken: Wiley, 2009.
2. Howlin P, Goode S, Hutton J et al. Adult outcome for children with autism. J Child Psychol Psychiatry Allied Discipl 2004; 45:212-29.
3. Seltzer MM, Shattuck P, Abbeduto L et al. Trajectory of development in adolescents and adults with autism. Ment Retard Dev Disabil Res Rev 2004;10:234-47.
4. Brereton AV, Tonge BJ. Autism and related disorders in adults. Curr Opin Psychiatry 2002;15:483-7.
5. Howlin P. Outcome in adult life for more able individuals with autism or Asperger syndrome. Autism 2000;4:63-83.
6. Renty JO, Roeyers H. Quality of life in high-functioning adults with autism spectrum disorder: the predictive value of disability and support characteristics. Autism 2006;10:511-24.
7. Farley MA, McMahon WM, Fombonne E et al. Twenty-year outcome for individuals with autism and average or near-average cognitive abilities. Autism Res 2009;2:109-18.
8. Eaves LC. Young adult outcome of autism spectrum disorders. J Autism Dev Disord 2008;38:739.
9. Orsmond G, Krauss M, Seltzer M. Peer relationships and social and recreational activities among adolescents and adults with autism. J Autism Dev Disord 2004;34:245-56.
10. Shtayermman O. Peer victimization in adolescents and young adults diagnosed with Asperger's syndrome: a link to depressive symptomatology, anxiety symptomatology and suicidal ideation. Issues Compr Pediatr Nurs 2007;30:87-107.
11. Rutter M. Child psychiatric diagnosis and classification: concepts, findings, challenges and potential. J Child Psychol Psychiatry Allied Discipl 2011;52:647-60.

Autism as the clinical core marker in schizophrenia

In his paper published in the June 2012 issue of *World Psychiatry*, J. Parnas focuses on the fundamental disturbance in patients before the onset of schizophrenic illness (1). He refers to the ICD-8, in which a personality disturbance was considered to be the clinical core marker of schizophrenia. In the ICD-9, this concept was retained including a disturbance of the will (inertia and negativism).

In his editorial in the same issue (2), M. Maj calls for follow-up studies on Parnas' Gestalt approach in order to capture the basis of schizophrenia. This dialog immediately brought to my mind the work of another Danish psychiatrist, E. Dein (1922–1975).

In the 1950s, Dein performed follow-up studies in patients within the spectrum of schizophrenia (3). He defined autism as "a permanent and socially perceptible deviation as to the interpersonal relations of the individual, characterized by a pervasive tendency to keep an abnormal distance to other people, to adopt a passive attitude and to be emotionally indifferent" (4). He found that 109 of 142 patients with chronic schizophrenia had autism before the onset of their illness, compared to only one of 52 patients with episodic schizophreniform (probably schizoaffective) states (3). The results obtained by Dein confirmed Kraepelin's observation that autism is a clinical core marker of schizophrenia. The Gestalt approach proposed by J. Parnas is a major contribution in this respect.

During our validation study on depression and mania scales (5), Dein told me that the most severe case of contact disturbance he had ever observed was in a patient with manic-depressive illness. In that illness, however, the disturbance does not appear outside acute episodes, in contrast to schizophrenia, where it represents a personality trait (4).

In his editorial, M. Maj also makes some remarks on the practical problems of Parnas' model. Among these is that the core Gestalt is possibly very reliable in the hands of super experts, but dangerously volatile in ordinary clinical practice (2). In this respect, Dein's words (4) echo Maj's concerns: "It is true that the ability of psychiatrists to perceive subtle shades in contact varies considerably, and perhaps a natural wish to establish contact may lure many psychiatrists into overestimating their rapport with the patient. It is at any rate inexpedient if a criterion so decisive for the diagnosis as autism must depend too much on the individuality of the psychiatrist, and it really may become a problem if the amount of his experience and competence comes to play a dominant role in clinical discussion".

Dein also states that "when evaluating possible autism... one should... try to close one's eyes to other symptoms, as for instance disturbances of thinking, transitivistic phenomena, delusions or hallucinations; these symptoms are sometimes so suggestive of the diagnosis of schizophrenia that one may be prejudiced in favour of the presence of autism" (4). When making an attempt to close the eyes of psychiatrists of the DSM-IV era to Schneider's first-rank symptoms in order to focus on the key feature of schizophrenia, Maj (6) has previously concluded that the concept of autism, as revisited by Parnas (7), deserves attention. We believe that this element should be explicitly introduced in the current debate in the journal.

Per Bech
Psychiatric Research Unit, Mental Health Centre North Zealand, University of Copenhagen, Dyrehavevej 48, DK-3400 Hillerød, Denmark

References

1. Parnas J. The core Gestalt of schizophrenia. World Psychiatry 2012;11:67-9.
2. Maj M. The self and schizophrenia: some open issues. World Psychiatry 2012;11:65-6.
3. Dein E. Personlighedstyper ved skizofreni og skizofreniforme psykoser. Nordic J Psychiatry 1964;18:88-105.
4. Dein E. On the concept of autism. Acta Psychiatr Scand 1966;191:124-35.
5. Bech P. Clinical psychometrics. Oxford: Wiley Blackwell, 2012.
6. Maj M. Critique of the DSM-IV operational diagnostic criteria for schizophrenia. Br J Psychiatry 1998;172:458-60.
7. Parnas J, Bovet P. Autism in schizophrenia revisited. Compr Psychiatry 1991;32:7-21.

Transition from psychiatric training to independent practice: a survey on the situation of early career psychiatrists in 35 countries

The transition from psychiatric training to independent practice is a crucial step in the professional development of every psychiatrist. For many psychiatrists, this phase also determines their choice of subspecialization and therefore has an important impact both on individual career paths as well as on the availability of psychiatrists in the various areas of our specialty.

Despite its importance, this period of professional development is relatively understudied (1). In accordance with the WPA aim to "promote the professional development of early career psychiatrists worldwide" (2), the members of the WPA Early Career Psychiatrists Council (ECPC), Europe I area, conducted a survey to investigate this period in more detail (3).

A 27-item online questionnaire was developed covering: a) factors influencing choice of psychiatric subspecialty and work setting, b) job availability, and c) availability of training and mentoring opportunities specific to this phase. All 60 Council members were asked to participate in the survey as experts of the situation in their countries. Forty Council members representing 35 countries from all five continents participated in the survey, resulting in a response rate of 66%.

Career choice was more often reported to be strongly or very strongly influenced by personal variables, such as salary expectations (30/40), personal interests (29/40), and compatibility with private life (27/40), than by external factors such as societal expectations (13/40) and the political environment (10/40). Strikingly, gender inequalities were reported to have a strong influence on career choice in three countries. Furthermore, six of 40 respondents stated that in their countries the location of practice was decided "by somebody else" rather than the psychiatrists themselves. In 12 countries, higher salaries and access to subspecialty training are used as incentives to recruit psychiatrists into underserved regions.

The risk for unemployment immediately after psychiatric specialization was judged either unlikely or very unlikely by almost all experts (37/40). However, more than half (21/40) stated it was difficult or very difficult to get the desired job. Positions in university hospitals and private psychiatric practices were most frequently cited as attractive career options. Of these, the availability of job opportunities in university hospitals was reported to be "quite bad" or "bad" in 21 of 40 countries. In addition, 14 of 40 representatives reported that it was not possible in their countries to immediately establish oneself as an independently practicing psychiatrist after completion of training. Limitations fixed by governments regarding the number of available positions and periods of mandatory service in the public sector – in one country up to nine years – were mentioned as restricting factors.

Although the transition between psychiatric training and independent practice is associated with high levels of stress, anxiety, and difficulties with patient care amongst young psychiatrists (4), the survey highlighted a lack of specific support during this phase. Specific training opportunities to develop "real world" psychiatric skills, for example, in leadership or management, were reported to exist only by eight of 40 respondents. While 19 representatives stated that in their country a mentor was available during psychiatric training, that number dropped to 11 for the first years of working as a specialist.

In our view, the transition from psychiatric training to independent practice should be recognized as a complex task and a crucial step toward mastery of our profession. It deserves both self-reflection by the young psychiatrist and support by psychiatric professional associations. Specific training opportunities for this transition period should be created. Furthermore, we believe that establishing incentives may be a more suitable approach than imposing restrictions in order to sustainably attract young psychiatrists to work in underserved regions of a country or neglected fields of psychiatry (5). While the latter approach may function in the short run, it can be a strain on the individual psychiatrist and a danger to recruitment in the long-term.

Florian Riese[1], Clare Oakley[2], Marie Bendix[3], Piirika Piir[4], Andrea Fiorillo[5]
[1]Psychiatric University Hospital, Zurich, Switzerland;
[2]Institute of Psychiatry, King's College, London, UK;
[3]Psychiatric Clinic Southwest, Karolinska University Hospital Huddinge, Stockholm, Sweden;
[4]Psychiatry Clinic, University of Tartu, Estonia;
[5]Department of Psychiatry, University of Naples SUN, Naples, Italy

References

1. Riese F, Salvi V, O'Leary P et al. Not quite there yet? The transition from psychiatric training to practice as a psychiatric specialist.

In: Fiorillo A, Calliess I, Sass H (eds). How to succeed in psychiatry – a guide to training and practice. Oxford: Wiley-Blackwell, 2012:147-60.

2. Maj M. Report on the implementation of the WPA Action Plan 2008-2011. World Psychiatry 2011;10:161-4.

3. Fiorillo A, Brambhatt P, Elkholy H et al. Activities of the WPA Early Career Psychiatrists Council: the Action Plan is in progress. World Psychiatry 2011;10:159.

4. Looney JG, Harding RK, Blotcky MJ et al. Psychiatrists' transition from training to career: stress and mastery. Am J Psychiatry 1980;137:32-6.

5. Gunther OH, Kurstein B, Riedel-Heller SG et al. The role of monetary and nonmonetary incentives on the choice of practice establishment: a stated preference study of young physicians in Germany. Health Serv Res 2010;45:212-29.

Mobile telepsychiatry in India

It was interesting to read B. Grady's article (1) in the October 2012 issue of *World Psychiatry* and draw parallels to our own experience in delivering telepsychiatry services to rural parts of Tamil Nadu in India.

Schizophrenia Research Foundation (SCARF) began experimenting with telepsychiatry in 2005 as part of its psychosocial intervention program for Tsunami victims and has since developed it into a full-fledged component of its community outreach program. The services were initially delivered through Integrated Services Digital Network (ISDN) lines and subsequently, with the availability of increased bandwidth, we shifted over to broadband. In 2010, with support from the Tata Education Trust, we expanded our service to cover the district of Pudukottai and pioneered the delivery of telepsychiatry services on a mobile platform.

The mobile service covers 156 villages with a population of about 300,000. At present the service focuses only on those with serious mental disorders and we estimate that about 1,000 people will avail of the service over three years.

SCARF's mobile telepsychiatry service is provided on a bus that has been custom-built to contain a consultation room and a pharmacy. In the consultation room, communication takes place between a psychiatrist based in Chennai and the patient and caregivers through flat screen TVs and state-of-the-art high-resolution cameras using a wireless Internet connection.

After a teleconsultation, a prescription is advised by the psychiatrist to the telepsychiatry clinic facilitator in the bus and dispensed by the on-board pharmacy. A follow-up appointment date is also given for review. The medication is provided free of cost. This is an essential component of the program considering the patients' financial limitations and also the fact that psychiatric drugs are rarely stocked in rural pharmacies.

Each patient receives a patient-held record designed to facilitate continuity of care and information sharing between health care professionals. It details their diagnosis, prescription, and any relevant investigations that the patient must get done independently, such as an EEG or blood tests.

Apart from teleconsultations, the package provides psychosocial interventions including psychoeducation for caregivers delivered by community health workers. There is also an emphasis on getting jobs for patients through existing schemes of government and in the voluntary sector. The health workers are supervised through videoconferencing and periodic onsite visits.

Improving awareness about mental illness is another important element of the package. Poor understanding of the illness delays early identification and treatment and thereby adds to the stigma experienced. The villagers themselves are targeted by awareness campaigns, which include street plays, the distribution of posters and pamphlets, and the screening of films. These are broadcast on a TV screen fitted to the rear of the bus, and were created specifically to educate people about the signs and symptoms of psychotic disorders. The film also explains the process of telepsychiatry and the objective of the program.

While the program has been by and large successful in demonstrating the feasibility of delivering a mobile telepsychiatry service, a major challenge has been managing an increasing patient load, as well as bringing into sharp focus Grady's view (1) that telepsychiatry redistributes resources rather than creating additional capacity.

Overall, our experience has been extremely positive and it encourages us to broaden the reach of the program. Integrating this strategy into the district mental health program could prove to be hugely beneficial especially in reaching out to remote areas. With the recent advances in telecommunication facilities in India, this is the ideal time to exploit the immense potential of telepsychiatry.

Rangaswamy Thara,
John Sujit
Schizophrenia Research Foundation, Chennai, India

Reference

1. Grady B. Promises and limitations of telepsychiatry in rural adult mental health care. World Psychiatry 2012;11:199-201.

The characteristics, content, performance, and impact of the WPA website (www.wpanet.org)

LEVENT KÜEY

WPA Secretary General and Website Editor

The WPA has been improving its organizational image and identity in line with its Action Plan of 2008–2011 and that of 2011–2014. The official website of the WPA, along with its other media channels (WPA News, the official WPA newsletter, and the WPA E-Bulletin) has played an important role in this process.

Since 2008, WPA media channels were reconstructed and improved to be in concert both in their contents and aesthetics. The general perspective has been to reach elegancy in simplicity. WPA media channels were enriched to raise their efficiency, both at inter-organizational and intra-organizational levels, and to mirror the voice of all WPA components. Requests for contributions were sent out regularly and any submitted item was published after an edition and approval process. The WPA Secretary General, as the editor of these media channels, monitored this route with the collaboration of the WPA Executive Committee members and the WPA Secretariat staff. This article focuses on the main characteristics, content, performance and impact of the WPA website.

The renewal of the website and the transfer of the webmaster and the server, aiming to improve the global impact of the WPA website by revising its content, infrastructure, visual design, and process of maintenance, started by early 2009 and were finalized in a year. A totally reconstructed new website was published on April 12, 2010, after comprehensive discussions in the WPA Executive Committee (2008–2011), and with the IT staff responsible for the technical administration of the website, aiming to enhance its readability, usability, and impact.

CHARACTERISTICS

The WPA website was reconstructed and redesigned, aiming to be easy to browse for the users, easy to manage for the webmasters and editor, and with a higher quality content and aesthetics based on a well-developed infrastructure.

This website is open to all visitors without any password filtering area, which contributes to the organizational transparency and accessibility of the work of the WPA in general. This also gives the opportunity both to mental health professionals and nonprofessionals to reach the educational material in various languages.

The home page has been shortened and packed, hence made suitable for a user's eye to track. The content of the home page is now more systematic and categorized. All of these changes make it easier for the user to follow the content and give priority according to his/her own needs.

A lateral navigation and menu system was developed, so that users can browse easier. For navigation aid, a "breadcrumb trail system", which gives users a way to keep track of their locations within programs or documents, was designed and applied, so that users can know how they get there while browsing.

The infrastructure was categorized and the database was dynamically coded to manage articles, meetings, files, or any item used in the site. All of the pages were coded systematically and are processed in hyper text markup language (HTML) format automatically.

A web content management system which supports categorization and management of the content and makes it possible to create a sitemap, was built. All of the pages and contents are dynamically shown in their own templates. All the visuals and templates are relatively easy to modify. A text editor, "WYSIWYG" (What You See is What You Get), was used on the administration area to decrease the amount of typos and similar mistakes and make it easier to add images, links, .pdf, and/or.doc files to a page.

The interactive materials on the website were renamed and uploaded according to their locations in the main navigation. This new system of documents provides content usability in the website and also in the administration area. Uploading process of new materials was systematically designed, also giving the opportunity of hosting interactive applications, such as video recordings, password-protected areas (if needed), registration for applications, and so forth. Accordingly, a module that supports videos and presentations for the section of "WPA E-Learning" was designed and added.

CONTENT

The content of the website reflects and reconstructs the activities of all the WPA components. It currently hosts more than 3500 items, covering news on all areas of activities of the WPA and its components. All issues of the WPA official journal, *World Psychiatry*, continuing educational material for psychiatrists, educational material for the general public on mental health issues, material describing successful experiences in the mental health field, and information on WPA Secretariat activities, meetings, publications, and scientific sections are published. Furthermore, information on the structure and work of the WPA Committees and the WPA normative instruments are posted.

A highly visited section of the website hosts *World Psychiatry*. Psychiatrists across the world can read and freely download the journal not only in English but also in various languages; either full issues or individual papers or abstracts are available in Spanish, Chinese, Russian, French, Arabic, Turkish, Japanese, Polish, Romanian, and Italian.

A WPA E-Learning Programme was developed and implemented in 2010. This program covers videos and slide sets of prominent scientific lectures and presentations from the WPA Congresses, starting with a selection from the WPA International Congress 2009, Florence. Currently, 33 lectures in

video and synchronized .ppt format are up-loaded. Furthermore, an online public education program, aiming to provide high-quality and reliable scientific information on mental health and psychiatry to nonprofessionals, is also hosted in the website.

Other WPA educational products that are available on and freely downloadable from the website include three sets of slides based on a series of WPA books dealing with the recognition, epidemiology, pathogenesis, cultural aspects, medical costs, and management of the comorbidity of depression with diabetes, heart disease, and cancer (1-3). In addition to their English versions, these slide sets are available in up to 17 languages (English, French, Portuguese, Spanish, Italian, Estonian, Croatian, German, Swedish, Azeri, Bosnian, Romanian, Czech, Russian, Indonesian, Bangla, and Japanese). An educational module and two sets of slides on physical illness in patients with severe mental disorders, developed by the WPA (4,5), are also posted on the website.

Moreover, the WPA website hosts some essential documents for the benefit of improving the ethical and scientific quality standards of our profession: the Madrid Declaration on Ethical Standards (as amended by the General Assembly of the WPA in Buenos Aires on September 21, 2011); the WPA template for undergraduate and postgraduate education in psychiatry and mental health; recommendations for relationships of psychiatrists, health care organizations working in the psychiatric field, and psychiatric associations with the pharmaceutical industry (6); recommendations on best practices in working with service users and family carers (7); and WPA guidance papers on implementation of community mental health care (8), how to combat stigmatization of psychiatry and psychiatrists (9), mental health care in migrants (10), and promotion of mental health in children of persons with severe mental disorders (11) are examples of such documents.

PERFORMANCE

The performance of the website, which shows a wide acceptance across the world, is followed closely with peri-odic analysis. Here, we review the performance of the website since its publication, covering the period between April 12, 2010 and October 12, 2012.

As shown in Table 1, during these 2.5 years, more than 200,000 people have visited the WPA website, making more than 300,000 visits. These visits came from 209 countries/territories, practically covering all parts of the world. If we note that the number of countries in which WPA has national member societies is 117, this means that the WPA is even reaching over its organizational limits by its website.

The website is continuously visited by new people, with the proportion of new visitors being 69%. This is also reflected in the fact that 68% of the visitors of the website reach it via "search engines", while 17% via "referring sites", and 15% using "direct traffic".

IMPACT

One of the widely used criteria to measure the impact of a website is "page rank check", a free service provided by Google. The page rank value indicates the importance of a particular website/page. Being an objective measure of its citation importance, it also corresponds well with people's subjective idea of importance. Currently, the page rank of the WPA website is 7/10 (that is, the page rank value is 7 from 10 possible points), which reflects a high impact compared to many other similar websites.

As the editor and the WPA Secretary General, I have been facilitating this process with the cooperation of the WPA Executive Committee members of 2008–2011 and of 2011–2014 and our past and current secretariat staff, Anna Engström, Francesca Sotgiu, and Pamela Atiase, and our IT staff at the Istanbul-based agency Saglik Bahcesi. Their enthusiasm, support, and skillful efforts are highly appreciated.

In fact, a website is a highly flexible living platform, a work in progress where we can explore further improvements continuously. I hope the users of the WPA website will continue to help us in improving its quality by sending their contributions and comments.

Table 1 WPA website statistics (April 12, 2010 to October 12, 2012)

Visitors	215,836
Visits	313,916
Countries/territories	209
Visits per day (average)	348
Pages per visit (average)	3.36
Page titles visited (at least once)	3134
Page titles viewed	1,054,151

References

1. Katon W, Maj M, Sartorius N. Depression and diabetes. Chichester: Wiley, 2010.
2. Glassman A, Maj M, Sartorius N. Depression and heart disease. Chichester: Wiley, 2011.
3. Kissane DW, Maj M, Sartorius N. Depression and cancer. Chichester: Wiley, 2011.
4. De Hert M, Correll CU, Bobes J et al. Physical illness in patients with severe mental disorders. I. Prevalence, impact of medications and disparities in health care. World Psychiatry 2011;10:52-77.
5. De Hert M, Cohen D, Bobes J et al. Physical illness in patients with severe mental disorders. II. Barriers to care, monitoring and treatment guidelines, plus recommendations at the system and individual level. World Psychiatry 2011; 10:138-51.
6. Appelbaum P, Arboleda-Florez J, Javed A et al. WPA recommendations for relationships of psychiatrists, health care organizations working in the psychiatric field and psychiatric associations with the pharmaceutical industry. World Psychiatry 2011; 10:155-8.
7. Wallcraft J, Amering M, Freidin J et al. Partnerships for better mental health worldwide: WPA recommendations on best practices in working with service users and family carers. World Psychiatry 2011;10:229-37.
8. Thornicroft G, Alem A, Dos Santos RA et al. WPA guidance on steps, obstacles and mistakes to avoid in the implementation of community mental health care. World Psychiatry 2010;9:67-77.
9. Sartorius N, Gaebel W, Cleveland HR et al. WPA guidance on how to combat stigmatization of psychiatry and psychiatrists. World Psychiatry 2010;9: 131-44.
10. Bhugra D, Gupta S, Bhui K et al. WPA guidance on mental health and mental health care in migrants. World Psychiatry 2011;10:2-10.
11. Brockington I, Chandra P, Dubowitz H et al. WPA guidance on the protection and promotion of mental health in children of persons with severe mental disorders. World Psychiatry 2011;10:93-102.

WPA partnership with the World Health Organization in the development of the ICD-11 chapter on mental disorders

Paola Bucci

Department of Psychiatry, University of Naples SUN, Naples, Italy

The development of the 11th edition of the International Classification of Diseases (ICD) is ongoing, and the diagnostic system is expected to be published in the year 2015. The WPA is supporting the World Health Organization (WHO) in the production of the chapter on mental disorders.

The WPA Past-President, M. Maj, is a member of the International Advisory Group for the ICD-10 revision and the chairperson of the Working Group on Mood and Anxiety Disorders. Several WPA officers or experts have been appointed as chairpersons or members of ICD-11 Working Groups. The chairpersons include W. Gaebel (Working Group on Psychotic Disorders), P. Tyrer (Working Group on Personality Disorders), L. Salvador-Carulla (Working Group on Intellectual Disabilities), O. Gureje (Working Group on Somatic Distress and Dissociative Disorders), and D. Stein (Working Group on Obsessive-Compulsive and Related Disorders).

The WPA Member Societies participated in the WPA-WHO Global Survey of Psychiatrists' Attitudes Towards Mental Disorders Classification (1). This survey collected information from 4887 psychiatrists concerning their regular use of a formal classification system, the classification system most used, and the number of diagnostic categories desired, and their views on the most important purposes of classification, the use of strict criteria versus flexible guidance, how to incorporate severity and functional status in a classification of mental disorders, the utility of a dimensional component, the cultural applicability of current classifications, and the ease of use and the goodness of fit of ICD-10 diagnostic categories. The results of this survey are being used to improve the clinical utility of the ICD-11 chapter on mental disorders.

Several WPA Member Societies and experts are being or will be involved in ICD-11 field trials and in the various translations/adaptations of the diagnostic system. The WPA is actively contributing to the process of harmonization between the ICD-11 and the DSM-5.

Within the frame of the 15th World Congress of Psychiatry, held in Buenos Aires, Argentina in September 2011, several sessions related to the ICD-11 development took place, including a Special Symposium entitled "Developing the ICD-11 classification of mental disorders: field studies and global perspectives", with the participation of G. Reed, the WHO officer in charge of the process.

World Psychiatry is one of the main channels through which the international psychiatric community is being updated about the ICD-11 development. A special article authored by the ICD-11 International Advisory Group, summarizing the philosophy of the entire process, has been published in the journal (2), as well as the first report of the Working Group on Intellectual Disabilities (3) and a review of evidence and proposals for the ICD-11 classification of feeding and eating disorders (4). Several papers produced by the Working Group on Mood and Anxiety Disorders have been collected in a special supplement to the journal (5). Many relevant contributions have appeared in recent issues of the journal (6–25). All the above articles are available on the WPA website (www.wpanet.org).

References

1. Reed GM, Mendonça Correia J, Esparza P et al. The WPA-WHO Global Survey of Psychiatrists' Attitudes Towards Mental Disorders Classification. World Psychiatry 2011;10:118-31.
2. International Advisory Group for the Revision of ICD-10 Mental and Behavioural Disorders. A conceptual framework for the revision of the ICD-10 classification of mental and behavioural disorders. World Psychiatry 2011;10:86-92.
3. Salvador-Carulla L, Reed GM, Vaez-Azizi LM at al. Intellectual developmental disorders: towards a new name, definition and framework for "mental retardation/intellectual disability" in ICD-11. World Psychiatry 2011;10:175-80.
4. Uher R, Rutter M. Classification of feeding and eating disorders: review of evidence and proposals for ICD-11. World Psychiatry 2012;11:80-92.
5. Maj M, Reed GM (eds). The ICD-11 classification of mood and anxiety disorders: background and options. World Psychiatry 2012;11 (Suppl.1).
6. Maj M. Psychiatric diagnosis: pros and cons of prototypes vs. operational criteria. World Psychiatry 2011;10:81-3.
7. Widiger TA. Personality and psychopathology. World Psychiatry 2011;10:103-6.
8. Strakowski SM, Fleck DE, Maj M. Broadening the diagnosis of bipolar disorder: benefits vs. risks. World Psychiatry 2011; 10:181-6.
9. Goldberg D. The heterogeneity of "major depression". World Psychiatry 2011; 10:226-8.
10. Casey P, Bailey S. Adjustment disorders: the state of the art. World Psychiatry 2011;10:11-8.
11. Owen MJ. Is there a schizophrenia to diagnose? World Psychiatry 2011; 10:34-5.
12. Millon T. Further thoughts on the relation of personality and psychopathology. World Psychiatry 2011;10:107-8.
13. Links PS. Personality and psychopathology: the dangers of premature closure. World Psychiatry 2011;10:109-10.
14. Torgersen S. Personality may be psychopathology, and vice versa. World Psychiatry 2011;10:112-3.
15. Zimmerman M. Broadening the concept of bipolar disorder: what should be done in the face of uncertainty? World Psychiatry 2011;10:188-9.
16. Frank E. Bipolar spectrum: has its time come? World Psychiatry 2011; 10:193-4.
17. Carlson GA. Broadening bipolar disorder – by design or by accident? World Psychiatry 2011;10:195-6.

18. Maj M. Bereavement-related depression in the DSM-5 and ICD-11. World Psychiatry 2012;11:1-2.
19. Wakefield JC, First MB. Validity of the bereavement exclusion to major depression: does the empirical evidence support the proposal to eliminate the exclusion in DSM-5? World Psychiatry 2012;11:3-10.
20. Westen D. Prototype diagnosis of psychiatric syndromes. World Psychiatry 2012;11: 16-21.
21. Parnas J. The core Gestalt of schizophrenia. World Psychiatry 2012;11:67-9.
22. Carlson GA. Differential diagnosis of bipolar disorder in children and adolescents. World Psychiatry 2012;11:46-52.
23. Westen D, Malone JC, DeFife JA. An empirically derived approach to the classification and diagnosis of mood disorders. World Psychiatry 2012;11:172-80.
24. First MB. A practical prototypic system for psychiatric diagnosis: the ICD-11 Clinical Descriptions and Diagnostic Guidelines. World Psychiatry 2012;11: 24-5.
25. Jablensky A. Prototypes, syndromes and dimensions of psychopathology: an open agenda for research. World Psychiatry 2012;11:22-3.

Notice No responsibility is assumed by the Publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made.