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EDITORIAL

The dialogal basis of our profession: Psychiatry *with* the Person

Juan E. Mezzich

President, World Psychiatric Association

A recent historical opportunity to engage critical user groups – groups that traditionally protested outside on the street and which for the first time joined a WPA conference in Dresden, Germany – encourages us to reflect on the dialogal basis of our profession. To this effect, we briefly step into historical aspirations and recent public health and clinical statements arguing for a personalized and interactive approach in medicine at large and psychiatry in particular. And highlight the WPA Institutional Program on Psychiatry for the Person, especially psychiatry *with* the person, as a paradigmatic response to these challenges.

HISTORICAL AND CONTEMPORARY PERSPECTIVES

Ayurvedic and Chinese medical traditions, ancient and still practiced, with sound philosophical, experiential and experimental bases, focus on the patient's health rather than only on disease. Both of them articulate a comprehensive and harmonious framework of health and life and promote a highly personalized approach for the treatment of specific diseases and the enhancement of quality of life (1). Likewise, Hippocratic medicine emphasizes the wholeness of health and the value of engaging the patient as a full human being (2).

It happens that also recent major public health studies and statements are recommending a protagonic role for patients in the reorganization of health care services. The U.S. Presidential Commission on Mental Health Report (3), after a lengthy study documenting the inadequate state of mental health care in the United States, prescribed a number of necessary steps to be taken, including the development of a consumer-centered recovery-oriented mental health system. The WHO European Ministerial Conference on Mental Health in Helsinki (4) recommended, *inter alia*, to recognize the experience and knowledge of service users and carers and to empower them in the development of integrated health services.

In the clinical field, there is increasing recognition of the crucial role of a collaborative clinician-patient relationship. For example, Tasman (5) has cogently pointed out that this relationship must start from the first encounter and represents the fundamental matrix for the whole of care. It must ensure empathic listening, comprehensive diagnosis beyond symptom checklists, appreciation for symbolic meaning, broad treatment techniques and effective therapeutic partnership instead of narrow and reductionistic approaches. Likewise, Alanen et al (6), through a well-known Finnish integrated model for need-adapted assessment and treatment, emphasizes the active engagement of the patient as an expert of his/her own life situation within the context of family and community.

WPA RESPONSE

Of relevance to these developments, the WPA published in 2003 the International Guidelines for Diagnostic Assessment (IGDA), at the core of which is a diagnostic model articulating standardized multiaxial and idiographic personalized components. The latter proposes the interaction among clinicians, the patient and the family to formulate together a joint statement on contextualized clinical problems, the patient's positive health, and expectations on health restoration and promotion (7). This diagnostic model is being applied in different countries, as illustrated by the Latin American Guide for Psychiatric Diagnosis (8), and is one of the starting points for the emerging development of a person-centered integrative diagnostic model (9).

Even more specifically, in response to the ancient and contemporary perspectives outlined above and consistent with its constitutional purposes, the WPA adopted at its 2005 General Assembly in Cairo a Strategic Plan that included as one of its broad goals to strengthen WPA relations with patient/user organizations. It also established the Institutional Program on Psychiatry for the Person, which aims to promote a psychiatry of the person, by the person, for the person and, last but not least, with the person. This program, through its conceptual, clinical diagnosis, clinical care and public health components, represents a paradigmatic shift from a disease-oriented to a person-centered perspective (encompassing both ill and positive aspects of health) in psychiatry in particular and medicine at large. It is already attaining significant achievements and attracting wide attention throughout WPA and other major international medical and health organizations (see 10 for a general program outline).

The fourth programmatic objective, psychiatry *with* the person, is in fact the focus of this editorial. In reflection of this, a fundamental feature of the Institutional Program is the affirmation of the personhood of the patient and the commitment to work in respectful and collaborative partnership with the person who consults. This includes, first, work with individuals which highlights the ethical underpinnings of this effort. It also encompasses work with patient groups including those critical of psychiatry.

AN OPENING AT A DRESDEN CONFERENCE

It is on the above grounds that the WPA Thematic Conference held in Dresden on June 6-9, 2007 represents a crucial new opening for dialogue. The conference had an intriguing and sensitive overall topic, "Coercive Treatment in Psychiatry: A Comprehensive Review". John Monahan, Scientific Committee chair, anticipated on his invitation letter that "while the fissures in this area run so deep and are so long-standing that achieving consensus is unlikely, our aspiration is that this historic meeting will sharpen moral issues, clarify political viewpoints, identify evidence-based practices, and share cutting-edge data on one of the most contested topics of our time". In fact, as reported by Thomas Kallert, Organizing Committee chair, the Conference succeeded in attracting participants from 36 different countries, with virtually all world experts on this field attending and speaking at it, all leading to an absolutely top scientific program. But there was additionally a surprising event that marked the Conference indelibly. Most of the user groups critical of psychiatry (but not all), which traditionally would be expected to protest outside, decided to come in and engage with us in a discussion of serious concerns. This opening had a crucial value for WPA as this substantially broadened our range of patients/users interlocutors which also encompass groups (including self-help groups) with which psychiatric organizations have been interacting for a long time.

In a historic encounter on June 6, requested formally and with the endorsement of World Health Organization by Mind Freedom International and other European and World networks of current and past users of psychiatric services (European Network of ex-Users and Survivors of Psychiatry, ENUSP; World Network of Users and Survivors of Psychiatry, WNUSP), the president and other top leaders of WPA met with four representatives of the user organizations. The encounter originally scheduled to last one hour, spontaneously extended to three. A range of issues were discussed and possibilities for continuing the dialogue in congresses and other settings were explored. David Oaks, Director of Mind Freedom International, describing the encounter, stated: "This conversation was different than usual. Yes, once more, the proof will be in the results. But all involved felt they were heard and respected in this discussion" (11).

During the following day, the WPA Executive Committee suspended temporarily its official meeting in order to attend the keynote lecture by Ms. Dorothea Buck on "70 Years of Coercion in German Psychiatric Institutions, Experienced and Witnessed". On the basis of her personal history she challenged a psychiatry that neglects communication with patients and demanded a paradigm shift based on the wealth of patients' experiences. After her lecture, the WPA president presented a thank you speech for Ms. Buck's articulate and moving lecture. At an immediately ensuing press and news conference, representatives of the WPA, Council of Europe, and user organizations sitting at the main table held a lively exchange of questions, answers and comments with press representatives and the general audience. The issues experienced globally by service users, the patterns and diversity of their organizations, and prospective opportunities for continuing the Dresden dialogue and for user participation in activities of the WPA and their national member societies were broadly discussed.

CONCLUDING REMARKS

We have briefly outlined and discussed the dialogal bases of our profession with an emphasis on the objective to promote a psychiatry *with* the person. Reference has been made to historical and contemporary perspectives in the health field and WPA's response to them. A renewed commitment to the clinician-patient relationship appears crucial as well as building an effective dialogue with patient and user groups (as well as trialogues including families) respecting the diversity of their perspectives. Let's take advantage of the Dresden opening to find creative paths to work together for the fulfillment of psychiatry's and medicine's helping soul and the advancement of health in individuals and communities.

References

1. Patwardhan B, Warude D, Pushpangadan P et al. Ayurveda and traditional Chinese medicine: a comparative overview. Evidence-based Complementary and Alternative Medicine 2005;2:465-73.
2. Jouanna J. Hippocrates. Baltimore: Johns Hopkins University Press, 1999.
3. US Presidential Commission on Mental Health. Achieving the promise: transforming mental health care in America. Final report. Rockville: US Department of Health and Human Services, 2003.
4. World Health Organization European Ministerial Conference on Mental Health. Mental health action plan for Europe. Facing the challenges, building solutions. Copenhagen: World Health Organization European Office, 2005.
5. Tasman A. Presidential address: the doctor-patient relationship. Am J Psychiatry 2000;157:1763-8.
6. Alanen YO, Lehtinen V, Lehtinen K et al. The Finnish integrated model for early treatment of schizophrenia and related psychoses. In: Martindale B, Bateman A, Crowe M et al (eds). Psychosis. Psychological approaches and their effectiveness. London: Gaskell, 2000:235-65.
7. Mezzich JE, Berganza CE, von Cranach M et al. Essentials of the WPA International Guidelines for Diagnostic Assessment (IGDA). Br J Psychiatry 2003; Suppl. 45.
8. Asociación Psiquiátrica de América Latina. Guía Latinoamericana de Diagnóstico Psiquiátrico (GLADP). Guadalajara: Asociación Psiquiátrica de América Latina, 2004.
9. Mezzich JE, Salloum IM. Towards innovative international classification and diagnostic systems: ICD-11 and person-centered integrative diagnosis. Acta Psychiatr Scand 2007;116:1-5.
10. Mezzich JE. Psychiatry for the Person: articulating medicine´s science and humanism. World Psychiatry 2007;6:1-3.
11. Oaks D. World Psychiatric Association meets with psychiatric survivor movement groups. www.mindfreedom.org.

Management of persons with co-occurring severe mental illness and substance use disorder: program implications

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Adults with severe mental illness have extraordinarily high rates of co-occurring substance use disorders, typically around 50% or more, which adversely affect their current adjustment, course, and outcome. Separate and parallel mental health and substance abuse treatment systems do not offer interventions that are accessible, integrated, and tailored for the presence of co-occurrence. Recent integrated interventions for this population have the specific goal of ameliorating substance use disorder and the general goal of improving adjustment and quality of life. The authors overview the current research and offer guidelines related to mission and philosophy, leadership, comprehensive reorganization, training, specific programs, and quality improvement.

Key words: Dual diagnosis, severe mental illness, substance use disorder, integrated interventions

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The ubiquitous interconnections and adverse interactions between mental illnesses and substance use disorders have been documented for over 25 years (1,2). The large population of persons with co-occurring disorders is enormously heterogeneous in regard to type and severity of mental illness and substance use disorder, psychosocial skills and supports, and many other factors (3,4).

Providing services for persons with co-occurring disorders presents a dilemma. In the traditional system of parallel substance abuse and mental health services, few clients are able to access needed treatments for both disorders, and the services are rarely tailored to address the common interactive elements of co-occurrence (5). Therefore, clinicians and researchers have developed a number of strategies that combine, or integrate, mental health and substance abuse interventions. Recent reviews have identified dozens of controlled studies examining a range of psychosocial interventions (6-8) or pharmacological interventions (9) for these people. In addition, the National Evidence-Based Practices Project studied in detail the process of implementation of services for people with co-occurring disorders across several treatment settings (10). Only a few years ago, clinical guidelines called for integrating mental health and substance abuse interventions generically, without specific guidelines for clinical subgroups (11). In this article, we overview recent research and consider the implications for programs providing services to adult clients who have severe mental illness and substance use disorder.

RESEARCH ON CO-OCCURRING SEVERE MENTAL ILLNESS AND SUBSTANCE USE DISORDER

Definitions

"Severe mental illness" is a widely used expression that includes diagnosis, disability, and duration (12,13). In the U.S., most public mental health programs require these criteria for admission, which closely parallel Social Security Administration criteria for disability payments and public insurance (14). Diagnosis encompasses major mental disorders, such as schizophrenia, severe bipolar disorder, and severe depression. Disability indicates serious inability to meet adult role requirements, such as functioning in work, relationships, and self-care. Duration usually entails at least two years of disability. Major mental disorders and substance use disorders are usually defined according to the standard nomenclature of the Diagnostic and Statistical Manual (15). Substance use disorders include abuse or dependence on alcohol or other psychoactive drugs, including prescribed medications used in greater amounts than indicated (and usually excluding nicotine use disorder). Several terms, including dual diagnosis, dual disorders, and co-occurring disorders, are widely used to describe clients who have co-occurring severe mental illness and substance use disorder. In this article, we use these three terms interchangeably.

Interventions for mental illness and substance use disorder include treatments and rehabilitation. Treatments are medications or psychosocial strategies aimed at controlling or eliminating the symptoms or causes of illness or disorder; rehabilitation interventions are intended to improve skills and supports to enable persons to overcome the disabilities associated with illness or disorder. Treatment and rehabilitation overlap considerably.

Recovery has become a dominant concept in the health care system, but has not been consistently defined. It refers to a process of overcoming illness, rather than merely controlling symptoms, and moving beyond illness to pursue a satisfying and meaningful life (16-19). The term recovery is variously used for inspiration, advocacy, service development, policy, and other purposes. It often implies func-

tional outcomes, such as personally meaningful activities and relationships, but also refers to an individual's process of building hope and autonomy.

Prevalence

All mental illnesses, including mood, anxiety, personality, and schizophrenia-spectrum disorders, are associated with an increase in co-occurring substance use disorder compared to the general population (20-22). Furthermore, individuals with the most severe psychiatric disorders tend to have the highest rates of co-occurring substance use disorders. For example, in the largest general population survey of comorbidity conducted to date, the rate of lifetime alcohol or drug use disorder in the general population was approximately 17%, compared to 47% for people with schizophrenia, 56% for people with bipolar disorder, and about 30% for people with another mood disorder or an anxiety disorder (21). These prevalence rates are consistent with many other surveys of people with schizophrenia or bipolar disorder, which indicate lifetime prevalence rates for substance use disorders of about 50% (23-25) and rates for current or recent substance disorder in the range of 25-35% (26-28).

Demographic, family history, and personality characteristics of individuals prone to substance use disorders are similar in persons with severe mental illness and in the general population. Male sex, younger age, lower levels of education, and single marital status are all related to higher vulnerability to substance use disorders, with race/ethnicity often related to the type of substance misused but not the overall prevalence rate (24). Family history of substance use disorder is related to substance use disorder in persons with severe mental illness (29,30), as well as history of conduct disorder and adult antisocial personality disorder (31,32). Individuals with severe mental illness living in urban vs. rural areas do not tend to differ in overall rates of substance use disorder, although the types of substances may vary as a function of their market availability (33). Setting is also related to prevalence (34): individuals with severe mental illness receiving emergency or acute care treatment, as well those who are homeless (35,36) or incarcerated (33,37), have increased rates of substance use disorder.

Psychosocial interventions

Many recent reviews have addressed the rapid development of psychosocial interventions for people with dual diagnosis (6-8,38). The most recent systematic review identified 45 independent controlled clinical trials (7). Despite methodological problems, these studies show the following: a) there is inconsistent evidence to support any individual psychotherapy intervention; b) peer-oriented group interventions directed by a professional leader, despite heterogeneity of clinical models, are consistently effective in helping clients to reduce substance use and to improve other outcomes; c) contingency management also appears to be effective in reducing substance use and improving other outcomes, but has been less thoroughly studied and rarely used in routine programs; d) long-term (one year or more) residential interventions, again despite heterogeneity of models, are effective in reducing substance use and improving other outcomes for clients who have failed to respond to outpatient interventions and for those who are homeless; e) intensive case management, including assertive community treatment, consistently improves residential stability and community tenure, but does not consistently impact substance use; and f) several promising interventions, including family psychoeducation, intensive outpatient programs, self-help programs, and jail diversion and release programs, have received minimal research attention but warrant further study.

Pharmacological interventions

Pharmacological management of both the psychiatric and the substance use disorder is an important foundation of the treatment of clients with co-occurring severe mental illness and substance use disorder. In all of the above psychosocial studies, clients in psychosocial treatment research also received medication management, which was rarely accounted for in analyses. Research on the effects of medications themselves, however, is in its infancy. Thus far research suggests two main points. First, medications shown to be effective for the treatment of alcohol disorders in the general population, such as disulfuram and naltrexone, are probably effective also in clients with serious mental illness (9,39). Second, some medications that treat the mental illness may lead to reduction in the severity of the substance use disorder. Antidepressants appear to reduce not only symptoms of depression but also alcohol use in clients with major depression and alcohol disorder (40). Mood stabilizers are active not only on mania but also on alcohol use in clients with bipolar disorder and comorbid alcohol dependence (41,42). Typical antipsychotics improve the symptoms of schizophrenia but have little effect on co-occurring substance use. Most of the newer (atypical) antipsychotics are equally effective as the typical antipsychotics in improving schizophrenia symptoms and may offer some benefit in reducing craving or substance use, but research is preliminary (43). Clozapine is clearly the most powerful drug in treating schizophrenia symptoms and, at least in quasi-experimental studies, appears to be at the same time the most effective antipsychotic medication in relation to substance use.

Implementation of dual diagnosis programs

Experience with demonstration projects (44) as well as the recent National Evidence-Based Practices Project (10,45)

identify several factors that are critical for successful implementation and maintenance of dual diagnosis programs. These include clear guidelines regarding mission and philosophy, active leadership, comprehensive reorganization, longitudinal training and supervision, and quality improvement.

Course, outcomes, and recovery

As has been clear for many years, the natural course of severe mental illness for most people trends toward improvement, remission of symptoms, and recovery of functioning and quality of life over time, provided the affected individual does not suffer early mortality related to the illness (46). The same is true for individuals with alcohol use disorders (47). For individuals with co-occurring disorders, there has been little longitudinal evidence, though 3-year follow-ups do indicate steady improvements (48-50). Our recent 10-year prospective follow-up shows that steady movement toward recovery is the modal path (51). In this study, dual diagnosis clients themselves identified recovery outcomes and cutoffs: living independently, working in a competitive job, having regular contact with friends who were not substance users, expressing positive quality of life, actively managing substance use disorder, and controlling psychiatric symptoms. The major findings were the following: a) clients improved on all of these outcomes steadily over 10 years, b) the six domains were minimally related to one another, and c) the timing and sequence of movement toward recovery varied widely across clients. In other words, some became employed first, while others made progress in other domains first. We interpreted these findings to mean that recovery is expectable and normative, and that recovery occurs in individual patterns, domains, and rates. We also found that early mortality was common among those who did not attain remission of their substance use disorders (51).

PROGRAM IMPLICATIONS

Mission and philosophy

The clearest implication of the research on prevalence is that all programs for people with severe mental disorders should be considered dual diagnosis programs. Clients with co-occurring disorders are the norm rather than the exception. Every mental health clinician and every mental health program should embrace this reality and adopt reasonable modifications. Specialty teams will simply not suffice, because many clients will be left undiagnosed, untreated, and without needed supports for recovery. Further, many programmatic elements will not be tailored for the needs of dually disordered clients.

Longitudinal research shows that recovery is not only possible but appears to be the modal process for people with dual diagnoses. Nevertheless, many clients, families, and clinicians experience severe short-term problems and, for understandable reasons, manifest discouragement, hopelessness, and despair. They often have little or no information regarding the availability of effective treatments and the possibilities for long-term recovery. These findings imply an ethical imperative to provide education and hope. Hope is an essential aspect of the process of recovery (52-54). Accordingly, hopefulness and a realistic expectation of dual recovery inform the philosophy of dual diagnosis treatment. All clients can be seen as having potential to recover, and all clinicians can be helpful by conveying a realistic message of optimism regarding long-term recovery.

Leadership

The change from a single diagnosis to a dual diagnosis orientation requires many people to modify their attitudes, knowledge, and behaviors. This will not occur quickly. Above all it necessitates leadership. Based on the National Evidence-Based Practices Project (10) and other experiences (44,55), we recommend that leadership be construed in tiers of responsibility. At the ground level, all clinicians, clients and families have roles to play. They need to believe in dual recovery, become educated about their respective roles, and develop the skills and supports to facilitate recovery. They also need to be empowered to help plan and direct the changes. At the level of program managers, supervisors and trainers, leadership involves carefully planning to modify many programs and to facilitate learning for all staff. At the level of director and governance, leaders need to articulate vision, values and commitment. They also need to direct the strategy to insure that organizational structures (e.g., medical records) and finances support the changes.

Comprehensive reorganization

Dual diagnosis typically ramifies into many areas of one's life, and research shows that recovery encompasses different pathways, domains, styles, preferences and timing from one individual to the next. An individualized approach to intervention needs to address several areas of recovery, offer education and intervention choices, and be based on shared decision-making (56). This level of individualization will permit each client to pursue a path that he or she believes in.

Further, all programs need to be modified to insure that they are optimally helpful for clients with dual disorders. For example, medication management needs to avoid dangerous interactions and potentially addictive medications, such as benzodiazepines (57). Supported employment services need to focus on jobs and supports that enhance abstinence (58). Skills training needs to address managing drug purveyors as well as making friends (59).

Training

Training should address the generic needs of all staff as well as the needs of those who are specialists. Because of the high prevalence of substance use disorders in people with severe mental illness, all clinicians need basic training in working with dually diagnosed individuals (60). This includes information about the interactions between substance use and psychiatric illness, clues and instruments for recognizing and assessing substance use problems, an understanding of the concepts of stages of change (61) and stages of treatment (62), treatment planning skills, strategies for engaging clients in treatment and enhancing their motivation for sobriety, and the principles of collaborating with family members and other significant persons in treatment (59). In addition, clinicians who specialize in the treatment of persons with a dual disorder need to develop additional expertise in specific therapeutic modalities, including individual cognitive-behavioral therapy, group-based motivational and skills training approaches, family therapy, as well as skills for addressing common problem areas such as housing instability, legal problems, health problems, and trauma/victimization (59,63,64).

Special programs: group counseling and housing

Peer-oriented groups are the centerpiece of dual diagnosis treatment. The evidence shows that groups are the most effective first-line intervention to help people recover from co-occurring substance use disorder. The groups can be organized in different ways, using different models, meeting at different intensities, and for clients at different stages of recovery. There is as yet no evidence that one type of group is more effective than another; the key is steady attendance for several months, probably at least a year. Therefore, we recommend offering several options so that clients can find a group in which they feel comfortable.

Long-term residential treatment is the only established intervention for clients who do not respond to outpatient integrated treatments. As with group interventions, effective residential treatment programs vary considerably. The common elements of effective programs include flexible entry and discharge, integrated treatment for mental health and substance problems, a focus on employment and other aspects of rehabilitation, graduated approaches to lapses or relapses, and expected tenure of one year or more (65).

Of course, not all clients want or qualify for long-term residential treatment, and programs probably need a variety of other housing approaches (66). For example, a "housing first" approach helps many clients to escape from homelessness and to become motivated for further goals (67). There is also some evidence for a continuum approach to housing (68). Because housing is a primary goal for many clients and the evidence for specific approaches is not strong, providing multiple options makes sense here also.

Quality improvement

Another critical element of organization is quality improvement. This can take many forms, but most current approaches involve system engineering, data-based supervision, computerized medical records, electronic decision support systems, fidelity reviews, and intensive review of individual clients who are not making progress (69). A full discussion of quality improvement mechanisms is beyond the scope of this paper, but commitment to quality improvement is essential for successful program implementation.

CONCLUSIONS

As the literature on dual diagnosis continues to develop rapidly, programmatic implications for treating clients with co-occurring disorders become more specific. This paper overviews several steps that all mental health leaders should consider, including efforts to reconfigure mental health programs into dual recovery programs. We strongly urge further research with greater standardization and methodological rigor to move this field ahead (70).

References

1. Caton CLM. The new chronic patient and the system of community care. Hosp Commun Psychiatry 1981;32:475-8.
2. Pepper B, Krishner MC, Ryglewicz H. The young adult chronic patient: overview of a population. Hosp Commun Psychiatry 1981; 32:463-9.
3. Lehman AF. Heterogeneity of person and place: assessing co-occurring addictive and mental disorders. Am J Orthopsychiatry 1996; 66:32-41.
4. Ridgely MS, Osher FC, Goldman H et al. Chronic mentally ill young adults with substance abuse problems: a review of research, treatment, and training issues. Baltimore: University of Maryland, 1987.
5. Polcin DL. Issues in the treatment of dual diagnosis clients who have chronic mental illness. Prof Psychol Res Pract 1992;23:30-7.
6. Brunette MF, Mueser KT, Drake RE. A review of research on residential programs for people with severe mental illness and co-occurring substance use disorders. Drug Alcohol Rev 2004;23:471-81.
7. Drake RE, O'Neal E, Wallach MA. A systematic review of research on interventions for people with co-occurring severe mental and substance use disorders. J Subst Abuse Treat (in press).
8. Mueser KT, Drake RE, Sigmon SC et al. Psychosocial interventions for adults with severe mental illnesses and co-occurring substance use disorders: a review of specific interventions. J Dual Diagnosis 2005;1:57-82.
9. Petrakis IL, Nich C, Ralevski E. Psychotic spectrum disorders and alcohol abuse: a review of pharmacotherapeutic strategies and a report on the effectiveness of naltrexone and disulfiram. Schizophr Bull 2006;32:644-54.
10. McHugo GM, Drake RE, Whitley R et al. Fidelity outcomes in the national implementing evidence-based practices project. Psychiatr Serv (in press).
11. United States Department of Health and Human Services. Assessment and treatment of patients with coexisting mental illness and alcohol and other drug abuse. Rockville: United States Department of Health and Human Services, 1994.

12. Ruggeri M, Leese M, Thornicroft G et al. Definition and prevalence of severe and persistent mental illness. Br J Psychiatry 2000; 176:149-55.
13. Schinnar A, Rothbard A, Kanter R et al. An empirical literature review of definitions of severe and persistent mental illness. Am J Psychiatry 1990;147:1602-8.
14. Stabo JD, McGerary M, Barnes DK. Improving the social security disability decision process: final report. Washington: National Academies Press, 2006.
15. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington: American Psychiatric Association, 1994.
16. Bellack AS. Scientific and consumer models of recovery in schizophrenia: concordance, contrasts, and implications. Schizophr Bull 2006;32:432-42.
17. Deegan PE. Recovery: the lived experience of rehabilitation. Psychosoc Rehabil J 1988;11:11-9.
18. Ralph RO. Review of recovery literature: a synthesis of a sample of recovery literature 2000. Alexander: National Technical Assistance Center for State Mental Health Planning, 2000.
19. New Freedom Commission on Mental Health. Achieving the promise: transforming mental health care in America. Washington: Department of Health and Human Services, 2003.
20. Kessler RC, Crum RM, Warner LA et al. Lifetime co-occurrence of DSM-III-R alcohol abuse and dependence with other psychiatric disorders in the National Comorbidity Survey. Arch Gen Psychiatry 1997;54:313-21.
21. Regier DA, Farmer ME, Rae DS et al. Comorbidity of mental disorders with alcohol and other drug abuse. JAMA 1990;264:2511-8.
22. Teeson M, Halal W, Lynskey M et al. Alcohol and drug use disorders in Australia: implications of the National Survey of Mental Health and Wellbeing. Aust N Zeal J Psychiatry 2000;34:206-13.
23. Cuffel BJ. Comorbid substance use disorder: prevalence patterns of use, and course. In: Drake RE, Mueser KT (eds). Dual diagnosis of major mental illness and substance disorder, Vol. II. San Francisco: Jossey-Bass, 1996:93-105.
24. Kavanagh DJ, Waghorn G, Jenner L et al. Demographic and clinical correlates of comorbid substance use disorders in psychosis: multivariate analyses from an epidemiological sample. Schizophr Res 2004;66:115-24.
25. Mueser KT, Yarnold PR, Levinson DF et al. Prevalence of substance abuse in schizophrenia: demographic and clinical correlates. Schizophr Bull 1990;16:31-56.
26. Graham HL, Maslin J, Copello A et al. Drug and alcohol problems amongst individuals with severe mental health problems in an inner city area of the UK. Soc Psychiatry Psychiatr Epidemiol 2001; 36:448-55.
27. Mueser KT, Bennett M, Kushner MG. Epidemiology of substance abuse among persons with chronic mental disorders. In: Lehman AF, Dixon L (eds). Double jeopardy: chronic mental illness and substance abuse. New York: Harwood Academic Publishers, 1995: 9-25.
28. Rosenberg SD, Drake RE, Wolford GL et al. The Dartmouth Assessment of Lifestyle Instrument (DALI): a substance use disorder screen for people with severe mental illness. Am J Psychiatry 1998;155:232-8.
29. Gershon ES, DeLisi LE, Hamaovit J et al. A controlled family study of chronic psychoses: schizophrenia and schizoaffective disorder. Arch Gen Psychiatry 1988;45:328-36.
30. Strakowski SM, DelBello MP. The co-occurrence of bipolar and substance use disorders. Clin Psychol Rev 2000;29:191-206.
31. Hodgins S, Tiihonen J, Ross D. The consequences of conduct disorder for males who develop schizophrenia: associations with criminality aggressive behavior, substance use, and psychiatric services. Schizophr Res 2005;78:323-35.
32. Mueser KT, Rosenberg SD, Drake RE et al. Conduct disorder, antisocial personality disorder, and substance use disorders in schizophrenia and major affective disorders. J Stud Alcohol 1999;60:278-84.
33. Mueser KT, Essock SM, Drake RE et al. Rural and urban differences in patients with a dual diagnosis. Schizophr Res 2001;48:93-107.
34. Galanter M, Castaneda R. Substance abuse among general psychiatric patients: place of presentation, diagnosis, and treatment. Am J Drug Alcohol Abuse 1988;14:211-35.
35. Caton CLM, Shrout PE, Eagle PF et al. Risk factors for homelessness among schizophrenic men: a case-control study. Am J Publ Health 1994;84:265-70.
36. Caton CLM, Shrout PE, Dominguez B et al. Risk factors for homelessness among women with schizophrenia. Am J Publ Health 1995; 85:1153-6.
37. Peters RH, Greenbaum PE, Edens JF et al. Prevalence of DSM-IV substance abuse and dependence disorders among prison inmates. Am J Drug Alcohol Abuse 1998;24:573-87.
38. Drake RE, Mueser KT, Brunette M et al. A review of treatments for people with severe mental illness and co-occurring substance use disorder. Psychiatr Rehabil J 2004;27:360-74.
39. Petrakis IL, O'Malley SS, Rounsville B et al. Naltrexone augmentation of neuroleptic treatment in alcohol abusing patients with schizophrenia. Psychopharmacology 2004;172:291-7.
40. Nunes EV, Levin FR. Treatment of depression in patients with alcohol or other drug dependence: a meta-analysis. JAMA 2004; 291:1887-96.
41. Brady KT, Sonnes AR, Ballenger JC. Valproate in the treatment of acute bipolar affective episodes complicated by substance abuse: a pilot study. J Clin Psychol 1995;56:118-21.
42. Salloum IM, Cornelius JR, Daley DC et al. Efficacy of valproate maintenance in patients with bipolar disorder and alcoholism: a double-blind placebo-controlled study. Arch Gen Psychiatry 2005; 62:37-45.
43. Green AI, Noordsy DL, Brunette MF et al. Substance abuse and schizophrenia: pharmacotherapeutic intervention. J Subst Abuse Treat (in press).
44. Torrey WC, Drake RE, Dixon L et al. Implementing evidence-based treatment for persons with severe mental illnesses. Psychiatr Serv 2001;52:45-50.
45. Torrey WC, Lynde DW, Gorman P. Promoting the implementation of practices that are supported by research: the national implementing evidence-based practice project. Child Adolesc Psychiatr Clin N Am 2005;14:297-306.
46. McGlashan TH. A selective review of recent North American long-term follow-up studies of schizophrenia. Schizophr Bull 1988; 14:515-42.
47. Vaillant GE. Natural history of alcoholism revisited. Cambridge: Harvard University Press, 1995.
48. Drake RE, McHugo GJ, Xie H et al. Three-year outcomes of long-term patients with co-occurring bipolar and substance use disorder. Biol Psychiatry 2004;56:749-56.
49. Essock S, Mueser KT, Drake RE et al. Comparison of ACT and standard case management for delivering integrated treatment for co-occurring disorders. Psychiatr Serv 2006;57:185-96.
50. Xie H, McHugo GJ, Helmstetter BS et al. Three-year recovery outcomes for long-term patients with co-occurring schizophrenic and substance use disorders. Schizophr Res 2005;75:337-48.
51. Drake RE, McHugo GJ, Xie H et al. Ten-year recovery outcomes for clients with co-occurring schizophrenia and substance use disorders. Schizophr Bull 2006;32:464-73.
52. Corrigan PW, Salzer M, Ralph RO et al. Examining the factor structure of the recovery assessment scale. Schizophr Bull 2004;30: 1035-41.
53. Deegan P. Recovery and the conspiracy of hope. Presented at the Sixth Annual Mental Health Conference of Australia and New Zealand, Brisbane, September 1996.
54. Roe D, Chopra M, Rudnick A. Persons with psychosis as active agents interacting with their disorder. Psychiatr Rehabil J 2004; 28:122-8.

55. Rapp CA, Gosche RJ. The strengths model: case management with people with psychiatric disabilities. New York: Oxford University Press, 2006.
56. Adams JR, Drake RE. Shared decision making and evidence-based practice. Commun Ment Health J 2006;42:87-105.
57. Brunette M, Noordsy DL, Xie H et al. Benzodiazepine use and abuse among patients with severe mental illness and co-occurring substance use disorders. Psychiatr Serv 2003;54:1395-401.
58. Becker DR, Drake RE, Naughton W. Supported employment for people with co-occurring disorders. Psychiatr Rehabil J 2005;28:332-8.
59. Mueser KT, Noordsy DL, Drake RE et al. Integrated treatment for dual disorders: a guide to effective practice. New York: Guilford, 2003.
60. Maslin J, Graham HL, Cawley M et al. Combined severe mental health and substance use problems: what are the training and support needs of staff working with this client group? J Ment Health 2001;10:131-40.
61. Prochaska JO, Diclemente CC. The transtheoretical approach: crossing the traditional boundaries of therapy. Homewood: Dow-Jones/Irwin, 1984.
62. Osher FC, Kofoed LL. Treatment of patients with psychiatric and psychoactive substance use disorders. Hosp Commun Psychiatry 1989;40:1025-30.
63. Centre for Addiction and Mental Health. Best practices: concurrent mental health and substance use disorders. Ottawa: Health Canada, 2001.
64. Graham HL, Copello A, Birchwood MJ et al. Cognitive-behavioural integrated treatment (C-BIT): a treatment manual for substance misuse in people with severe mental health problems. Chichester: Wiley, 2004.
65. Brunette MB, Noordsy DL, Buckley P et al. Pharmacologic treatments for co-occurring substance use disorders in patients with schizophrenia: a research review. J Dual Diagnosis 2005;1:41-55.
66. Osher FC, Dixon LB. Housing for persons with co-occurring mental and addictive disorders. In: Drake RE, Mueser KT (eds). Dual diagnosis of major mental illness and substance abuse 2: Recent research and clinical implications. New directions for mental health services. San Francisco: Jossey-Bass, 1996:53-64.
67. Tsemberis S, Gulcur L, Nakae M. Housing first, consumer choice, and harm reduction for homeless individuals with a dual diagnosis. Am J Publ Health 2004;94:651-6.
68. McHugo GJ, Bebout RR, Harris M et al. A randomized controlled trial of integrated versus parallel housing services for homeless adults with severe mental illness. Schizophr Bull 2004;30:969-82.
69. Hermann RC. Improving mental healthcare: a guide to measurement-based quality improvement. Washington: American Psychiatric Publishing, 2005.
70. McHugo GJ, Drake RE, Brunette MF et al. Enhancing validity in co-occurring disorders treatment research. Schizophr Bull 2006; 32:655-65.

SPECIAL ARTICLE

Child murder by mothers: patterns and prevention

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The tragedy of maternal filicide, or child murder by mothers, has occurred throughout history and throughout the world. This review of the research literature sought to identify common predictors in the general population as well as in correctional and psychiatric samples. Further research is needed to improve identification of children and mothers at risk. Infanticide laws are discussed. Suggestions for prevention are made based on the current literature and the authors' experiences.

Key words: Filicide, infanticide, child homicide

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When a young child is murdered, the most frequent perpetrator is a victim's parent or stepparent (1). Rates of infanticide parallel suicide rates rather than murder rates (2). The risk of being a homicide victim is highest during the first year of life (3-5). Though the US has the highest rates of child homicide (8.0/100,000 for infants, 2.5/100,000 for preschool-age children, and 1.5/100,000 for school-age children), the problem of child homicide transcends national boundaries (6). These rates of child murder are probably underestimates, due to inaccurate coroner rulings and some bodies never being discovered (4,7,8).

Maternal filicide is defined as child murder by the mother. *Infanticide* is child murder in the first year of life. The term *neonaticide* was coined by Resnick (9) to describe murder of an infant within the first 24 hours of life. Almost all neonaticides are committed by mothers. Neonaticidal mothers are often young, unmarried women with unwanted pregnancies who receive no prenatal care. For a detailed analysis of the neonaticide literature and a discussion of neonaticide prevention, the reader is referred to our recent review (10).

Resnick's review of the world psychiatric literature on maternal filicide (11) found filicidal mothers to have frequent depression, psychosis, prior mental health treatment, and suicidal thoughts. Maternal filicide perpetrators have five major motives: a) in an *altruistic filicide*, a mother kills her child out of love; she believes death to be in the child's best interest (for example, a suicidal mother may not wish to leave her motherless child to face an intolerable world; or a psychotic mother may believe that she is saving her child from a fate worse than death); b) in an *acutely psychotic filicide,* a psychotic or delirious mother kills her child without any comprehensible motive (for example, a mother may follow command hallucinations to kill); c) when *fatal maltreatment* filicide occurs, death is usually not the anticipated outcome; it results from cumulative child abuse, neglect, or Munchausen syndrome by proxy; d) in an *unwanted child filicide,* a mother thinks of her child as a hindrance; e) the most rare, *spouse revenge filicide* occurs when a mother kills her child specifically to emotionally harm that child's father.

In developing countries, the preference for male infants may lead to sex-selective killings (12,13). Cultural and legal differences across countries may affect research findings. For example, one country's correctional sample may be similar to another country's psychiatric sample, depending on the laws and attitudes toward prosecution.

The purposes of this paper are to summarize recent research findings about maternal filicide, and to consider potential strategies for prevention. The authors completed database searches for peer-reviewed articles in English regarding maternal filicide over the past quarter century. Studies were separated by population type, as in our previous analysis (14), because studies in the general population differ from those in psychiatric or correctional populations. Maternal filicide-suicide (a mother kills both her child and herself) was considered independently.

MATERNAL FILICIDE RESEARCH FINDINGS

Countries represented in the English literature filicide search were Australia, Austria, Brazil, Canada, Finland, France, Hong Kong, Japan, Ireland, New Zealand, Sweden, Turkey, the United Kingdom, and the United States. In addition to studies of mothers who have committed filicide (3,4,15-55), several studies have investigated the prevalence of filicidal thoughts in various populations.

Infanticide

An American macro-level study of *infanticide* (victims in the first year of life) found increased rates with economic stress (24). Although England and Wales have Infanticide Acts, and Scotland does not, the countries experience similar rates of infanticide (3,38). Maternal infanticide studies in the general population (20,38,44,45) found a predominance of unemployed mothers in their early 20s. Many cases occurred in the context of child abuse (4), though some mothers had associated suicide attempts. Often they expe-

rienced psychiatric disorders (36 to 72%) (44,45). In Japan, the infant victims frequently had physical anomalies.

General population studies of maternal filicide

The mothers were often poor, socially isolated, full-time caregivers, who were victims of domestic violence or had other relationship problems. Disadvantaged socioeconomic backgrounds and primary responsibility for the children were common. Persistent crying or child factors were sometimes precipitants for the filicide. Some mothers had previously abused the child, while others were mentally ill and devoted to their child (41). Neglectful or abusive mothers were often substance abusers. Many of the perpetrators had psychosis, depression, or suicidality (15,16,18,20,28,40-43,45,48,51,52).

Correctional samples of maternal filicide

In the correctional population, filicidal mothers were frequently unmarried, unemployed abuse victims, who had limited education and social support (29-33,46-47,53,54). Some had decreased intellect, and a few considered the child victim to be abnormal. Several correctional studies noted frequent depression, psychosis, substance abuse, suicidality, and prior mental health care (33,46,47,53,54). Multiple stressors (economic, social, abuse history, partner relationship problems), primary caregiver status, and difficulty caring for the child were frequent.

Psychiatric samples of maternal filicide

The filicidal mothers in psychiatric samples had frequently experienced psychosis, depression, suicidality, and prior mental health care (18,19,22,25-27,34-37,39,49,50,55). Their mean age was in their late 20s (18,19,22,25,34-36). Some were diagnosed with personality disorders and some had low intelligence. Significant life stresses were often noted. Our recent study of mothers found not guilty by reason of insanity in two U.S. states found that the perpetrators were often depressed and frequently experienced auditory hallucinations, some of a command type. Over one third of the homicides occurred during pregnancy or the postpartum year. Almost all the mothers had *altruistic* or *acutely psychotic* motives (22). A small New Zealand study that interviewed the mothers after their filicides found that psychotic mothers who had committed filicide often killed suddenly without much planning, whereas depressed mothers had contemplated killing their children for days to weeks prior to their crimes (49).

Maternal filicide-suicide

A significant proportion (16-29%) of filicides end in completed suicide by the mother (56). Many other mothers make non-fatal suicide attempts in association with their filicides. When mothers of young children commit suicide, about 5% also kill at least one of their children (57,58).

Filicide-suicides have much in common with filicides committed by severely mentally ill mothers (15). Most frequently, these mothers have altruistic motives (15,23). Similar to results of other studies (15,20,48), our recent American study found that maternal filicide-suicide perpetrators killed older children more often than infants (mean age of children killed was 6 years old). The mothers often had evidence of depression or psychosis (23). These mothers often take the lives of all their young children.

Prevalence of filicidal thoughts

A relatively high incidence of filicidal thoughts has been found in mentally ill women. Jennings et al's (59) study of depressed mothers with children under age 3 found that 41% had thoughts of harming a child, compared with 7% of mothers in the control group. A pediatric study of mothers in the general population found that 70% of mothers with colicky infants experienced explicit aggressive thoughts toward their infants, and over a quarter (26%) of them had infanticidal thoughts during colic episodes (60). An Indian study (61) of hospitalized severely mentally ill postpartum women found that 43% had infanticidal ideation. Thirty-six percent of these women engaged in some type of infanticidal behavior. Their behavior was associated with negative maternal reaction to separation, psychotic beliefs about the infant, and female sex of the infant.

Our recent survey of psychiatrists at two American academic institutions found that many psychiatrists do not specifically ask their patients who are mothers about thoughts of harming their children, but rather they inquire generally about homicidal thoughts (62). The surveyed psychiatrists frequently underestimated the prevalence of depressed mothers who have thoughts of harming their children.

INFANTICIDE LAWS

Infanticide laws often reduce the penalty for mothers who kill their children up to one year of age, based on the principle that a woman who commits infanticide does so because "the balance of her mind is disturbed by reason of her not having fully recovered from the effect of giving birth to the child" (41). The British Infanticide Act of 1922 (amended in 1938) allows mothers to be charged with manslaughter rather than murder if they are suffering from a mental disturbance. The law was originally based on the outdated concept of lactational insanity, but the public's desire to excuse sympathetic women caused reluctance to alter the law after lactational insanity was discredited. Women convicted of infanticide often receive probation and referral to mental health treatment rather than incarceration (41).

Approximately two dozen countries currently have infanticide laws (Australia, Austria, Brazil, Canada, Colombia, Finland, Germany, Greece, Hong Kong, India, Italy, Japan, Korea, New Zealand, Norway, Philippines, Sweden, Switzerland, Turkey and the United Kingdom (12,19,21,41,63). The majority of nations that have infanticide laws have followed the British precedent and decrease the penalty for mothers killing children under one year old. However, the legal definition of infanticide varies among countries. The murder of children up to age ten is included in New Zealand (21).

In practice, however, women convicted of infanticide in England sometimes do not have significant mental illness as technically required by the law (64). Opponents of infanticide laws point out that fathers are granted far less leniency. A father who is equally psychotically depressed as a mother, who kills his 10-month-old child in an altruistic psychotic belief with an associated suicide attempt, should not be treated differently than a similarly situated mother. Some feminists criticize the infanticide laws for "pathologizing childbirth". They believe that making this exception for women denies them the same capacity for self-governance attributed to men (65). Furthermore, it is illogical that a mother who in the throes of postpartum psychosis killed her newborn and her two-year-old should be charged with infanticide/manslaughter for the homicide of the newborn and murder for the homicide of the two-year-old. If the U.S. had an infanticide law, Andrea Yates would not have qualified, because in addition to her infant she killed her four older children. An acutely psychotic mother who killed her 13 month old child would not qualify for the infanticide law in England though a mother who battered her 11 month old child might.

SUGGESTIONS FOR PREVENTION

Psychiatrists should assess filicide risk in a systematic way, as they do for suicide. First they must entertain the possibility of maternal filicide. Psychiatrists should intervene to prevent potential filicides in which maternal mental illness plays a role. Mothers who have altruistic or acutely psychotic motives for filicide may be psychotic, depressed, manic, or delirious. Some mothers who come to psychiatric attention because of severe mental illnesses, personality disorders, or substance use disorders may be abusing or neglecting their children. Psychiatrists may ask about child-rearing practices, parenting problems, and feelings of being overwhelmed. Strategies for prevention must be tailored to the different motivations of mothers who commit filicide.

Depressed mothers who have the potential to kill in extended suicides should be identified early. Mothers contemplating suicide should be asked directly about the fate of their children if they were to take their own life. Some will say their husband is quite able to look after them and others will volunteer that they would take their children to heaven with them. Thoughts or fears of harming their children should be queried. Threats must be taken seriously. A lesser threshold for hospitalization should be considered for mentally ill mothers of young children due to the possibility of multiple deaths from a filicide-suicide. Factors which potentially merit psychiatric hospitalization include maternal fears of harming their child, delusions of their child's suffering, improbable concerns about their child's health, and hostility toward a despised partner's favorite child (66).

Psychotic mothers who fear that their children may suffer a fate worse than death due to persecutory delusions should either be hospitalized or separated from their children. These mothers may be reluctant to share their delusional ideas. Delusions may sometimes be elicited through a sympathetic exploration of their concerns for the safety of their children. In some cases, the only evidence of concern is frequent checking by the mother on the health and safety of her children. Though psychotic mothers may have less warning about filicide, psychiatrists can ask about hallucinations or delusional thoughts regarding the children. Among Indian mothers with postpartum severe mental illness, a recent study found that mothers with delusions about their infant engaged in more abuse (67).

Early screening and identification of mental illness both antenatally and postnatally is important. The Edinburgh Postnatal Depression Scale (68,69) is a validated tool that can be easily administered both in pregnancy and the postpartum. Up to 4% of mothers with untreated postpartum psychosis will commit infanticide (70). Because hospital length of stay after delivery is shorter now, many cases of postpartum psychosis could be undetected in the community. Therefore, community education is important. Support services for mothers and accessible psychiatric services for at-risk populations are needed.

More filicides occur due to fatal maltreatment than because of maternal psychiatric illness. Many cases of fatal maltreatment filicide never come to psychiatric attention. Mothers may kill their children who fail to respond to demands such as to stop crying (15). Mothers who batter their children to death are likely to have abused their children more than once before (15,25). Early intervention to protect these children is more likely to fall to child protective agencies than to psychiatrists. All 50 states in the U.S. have mandatory reporting laws for professionals who suspect child abuse. Parenting classes, emotional support, and emergency numbers to call when mothers are overwhelmed can be helpful in preventing fatal maltreatment filicides. Maternal substance abuse must also be treated. Child protective agencies must remove children who are at risk of serious abuse. Mothers who are diagnosed with Munchausen syndrome should be evaluated to see if they have engaged in Munchausen syndrome by proxy behaviors. Child protective agencies should be receptive to accepting children into their care who are unwanted, even if no abuse or neglect has yet occurred.

Spouse revenge filicide is difficult to prevent, because there is usually little warning. This behavior most often oc-

curs after learning of spousal infidelity or in the course of child custody disputes. Sometimes a mother is so convinced that her child will be sexually abused if permanent custody is awarded to her ex-husband that she decides the child is better off in heaven. Evaluators of child custody disputes should be alert for this potential.

Children under age 5 may have limited contacts outside of their household and have difficulty speaking out to others, while older children often attend school and can thus reveal child abuse. In the U.S., child homicide rates peak in winter for young children under age 2, and in the summer for older children (ages 5-14) (71). Infant and child factors such as colic (60) or autism (72) may increase risk. This suggests a potential role for pediatricians in prevention as well.

CONCLUSIONS

A mother's motive for filicide may be *altruistic*, *acutely psychotic*, or due to *fatal maltreatment, unwanted child,* or *spouse revenge*. In addition, many mothers who do not attempt filicide experience thoughts of harming their child. Maternal filicide motives provide a framework for approaching filicide prevention. Suicidality, psychosis and depression elevate risk, as does a history of child abuse. Mentally ill filicidal mothers have very different risk profiles than mothers who fatally batter their children. Prevention is difficult, because many risk factors, such as maternal depression and social disadvantage, are common among non-filicidal mothers.

References

1. Bureau of Justice Statistics, US Department of Justice. Homicide trends in the United States: infanticide. www.ojp.usdoj.gov/bjs/homicide/children.htm.
2. Lester D. Murdering babies: a cross-national study. Soc Psychiatry Psychiatr Epidemiol 1991;26:83-8.
3. Marks MN, Kumar R. Infanticide in England and Wales. Med Sci Law 1993;33:329-39.
4. Brookman F, Nolan J. The dark figure of infanticide in England and Wales: complexities of diagnosis. J Interpers Violence 2006; 21:869-89.
5. Zawitz MW, Strom KJ. Firearm injury and death from crime, 1993-97. US Department of Justice, Bureau of Justice Statistics, 2000.
6. Finkelhor D. The homicides of children and youth: a developmental perspective. In: Kantor GK, Jasinski JL (eds). Out of the darkness: contemporary perspectives on family violence. Thousand Oaks: Sage, 1997:17-34.
7. Emery JL. Child abuse, sudden infant death syndrome, and unexpected infant death. Am J Dis Child 1993;147:1097-100.
8. Ewigman B, Kivlahan C, Land G. The Missouri child fatality study: underreporting of maltreatment fatalities among children younger than five years of age. Pediatrics 1993;91:330-7.
9. Resnick PJ. Murder of the newborn: a psychiatric review of neonaticide. Am J Psychiatry 1970;126:58-64.
10. Friedman SH, Resnick PJ. Neonaticide: phenomenology and prevention. Int J Law Psychiatry (in press).
11. Resnick PJ. Child murder by parents: a psychiatric review of filicide. Am J Psychiatry 1969;126:73-82.
12. Hesketh T, Xing ZW. Abnormal sex ratios in human populations: causes and consequences. Proc Natl Acad Sci USA 2006;103: 13271-5.
13. Wu Z, Viisainen K, Hemminki E. Determinants of high sex ratio among newborns: a cohort study from rural Anhui province, China. Reproductive Health Matters 2006;14:172-80.
14. Friedman SH, Horwitz SM, Resnick PJ. Child murder by mothers: a critical analysis of the current state of knowledge and a research agenda. Am J Psychiatry 2005;162:1578-87.
15. Alder C, Polk K. Child victims of homicide. Cambridge: Cambridge University Press, 2001.
16. Alder CM, Baker J. Maternal filicide: more than one story to be told. Women and Criminal Justice 1997;9:15-39.
17. Bourget D, Gagne P. Maternal filicide in Quebec. J Am Acad Psychiatry Law 2002;30:345-51.
18. Bourget D, Bradford JM. Homicidal parents. Can J Psychiatry 1990;35:233-8.
19. Cheung PTK. Maternal filicide in Hong Kong, 1971-1985. Med Sci Law 1986;26:185-92.
20. Daly M, Wilson M. Killing children: parental homicide in the modern west. In: Daly M, Wilson M (eds). Homicide. New York: Aldine de Gruyter, 1988:61-93.
21. Dean PJ. Child homicide and infanticide in New Zealand. Int J Law Psychiatry 2004;27:339-48.
22. Friedman SH, Hrouda DR, Holden CE et al. Child murder committed by severely mentally ill mothers: an examination of mothers found not guilty by reason of insanity. J Forensic Sci 2005; 50:1466-71.
23. Friedman SH, Hrouda DR, Holden CE et al. Filicide-suicide: common factors among parents who kill their children and themselves. J Am Acad Psychiatry Law 2005;33:496-504.
24. Gauthier DK, Chaudoir NK, Forsyth CJ. A sociological analysis of maternal infanticide in the United States 1984-1996. Deviant Behavior 2003;24:393-405.
25. Haapasalo J, Petaja S. Mothers who killed or attempted to kill their child: life circumstances, childhood abuse, and types of killing. Violence Vict 1999;14:219-39.
26. Holden CE, Burland AS, Lemmen CA. Insanity and filicide: women who murder their children. New Directions for Mental Health Services 1996;69:25-34.
27. Husain A, Daniel A. A comparative study of filicidal and abusive mothers. Can J Psychiatry 1984;29:596-8.
28. Karakus M, Ince H, Ince N et al. Filicide cases in Turkey, 1995-2000. Croat Med J 2003;44:592-5.
29. Korbin JE. Incarcerated mothers' perceptions and interpretations of their fatally maltreated children. Child Abuse Negl 1987;11: 397-407.
30. Korbin JE. Childhood histories of women imprisoned for fatal child maltreatment. Child Abuse Negl 1986;10:331-8.
31. Korbin JE. Fatal maltreatment by mothers: a proposed framework. Child Abuse Negl 1989;13:481-9.
32. Korbin JE. 'Good mothers', 'babykillers', and fatal child maltreatment. In: Scheper-Hughes N, Sargent C (eds). Small wars: the cultural politics of childhood. Berkeley: University of California Press, 1998:253-76.
33. Laporte L, Poulin B, Marleau J et al. Filicidal women: jail or psychiatric ward? Can J Psychiatry 2003;48:94-109.
34. Lewis CF, Baranoski MV, Buchanan JA et al. Factors associated with weapon use in maternal filicide. J Forensic Sci 1998;43:613-8.
35. Lewis CF, Bunce SC. Filicidal mothers and the impact of psychosis on maternal filicide. J Am Acad Psychiatry Law 2003;31:459-70.
36. McKee GR, Shea SJ, Mogy RB et al. MMPI-2 profiles of filicidal, mariticidal, and homicidal women. J Clin Psychol 2001;57:367-74.
37. McGrath PG. Maternal filicide in Broadmoor Hospital. J Forensic Psychiatry 1992;3:271-97.
38. Marks MN, Kumar R. Infanticide in Scotland. Med Sci Law 1996;

36:201-4.
39. Meszaros K, Fisher-Danzinger D. Extended suicide attempt: psychopathology, personality and risk factors. Psychopathology 2000; 33:5-10.
40. Meyer CL, Oberman M. Mothers who kill their children: understanding the acts of moms from Susan Smith to the "Prom Mom". New York: New York University Press, 2001.
41. Oberman M. Mothers who kill: coming to terms with modern American infanticide. American Criminal Law Review 1996;34:2-109.
42. Pritchard C, Bagley C. Suicide and murder in child murderers and child sexual abusers. J Forensic Psychiatry 2001;12:269-86.
43. Rouge-Maillart C, Jousset N et al. Women who kill their children. Am J Forensic Med Pathol 2005;26:320-6.
44. Sakuta T, Saito S. A socio-medical study on 71 cases of infanticide in Japan. Keio J Med 1981;30:155-68.
45. Silverman RA, Kennedy LW. Women who kill their children. Violence Vict 1988;3:113-27.
46. Smithey M. Maternal infanticide and modern motherhood. Criminal Justice 2001;13:65-83.
47. Smithey M. Infant homicide at the hands of mothers: toward a sociological perspective. Deviant Behavior 1997;18:255-72.
48. Somander LK, Rammer LM. Intra- and extra-familial child homicide in Sweden 1971-1980. Child Abuse Negl 1991;15:45-55.
49. Stanton J, Simpson A, Wouldes T. A qualitative study of filicide by mentally ill mothers. Child Abuse Negl 2000;24:1451-60.
50. Stone MH, Steinmeyer E, Dreher J et al. Infanticide in female forensic patients: the view from the evolutionary standpoint. J Psychiatr Pract 2005;11:35-45.
51. Vanamo T, Kauppo A, Karkola K et al. Intra-familial child homicide in Finland 1970-1994: incidence, causes of death and demographic characteristics. Forensic Sci Int 2001;117:199-204.
52. Wallace A. Homicide: the social reality. Sydney: New South Wales Bureau of Crime Statistics and Research, 1986.
53. Weisheit RA. When mothers kill their children. Soc Sci J 1986; 23:439-48.
54. Wilczynski A. Child homicide. London: Oxford University Press/ Greenwich Medical Media, 1997.
55. Xie L, Yamagami A. How much of child murder in Japan is caused by mentally disordered mothers? Intern Med J 1995;2:309-13.
56. Nock MK, Marzuk PM. Murder-suicide: phenomenology and clinical implications. In: Jacobs DG (ed). Guide to suicide assessment and intervention. San Francisco: Jossey-Bass, 1999:188-209.
57. Appleby L. Suicidal behaviour in childbearing women. Int Rev Psychiatry 1996;8:107-15.
58. Schalekamp R.J. Maternal filicide-suicide from a suicide perspective: assessing ideation. www.filicide-suicide.com/summary-dissertation.pdf.
59. Jennings KD, Ross S, Popper S et al. Thoughts of harming infants in depressed and nondepressed mothers. J Affect Disord 1999;54: 21-8.
60. Levitzky S, Cooper R. Infant colic syndrome: maternal fantasies of aggression and infanticide. Clin Pediatr 2000;39:395-400.
61. Chandra PS, Venkatasubramanian G, Thomas T. Infanticidal ideas and infanticidal behavior in Indian women with severe postpartum psychiatric disorders. J Nerv Ment Dis 2002;190:457-61.
62. Friedman SH, Sorrentino RM, Stankowski JE et al. Mothers with thoughts of murder: psychiatric patterns of inquiry. Presented at the American Psychiatric Association Annual Meeting, Toronto, May 2006.
63. Mendlowicz MV, Rapaport MH, Mecler K et al. A case-control study on the socio-demographic characteristics of 53 neonaticidal mothers. Int J Law Psychiatry 1998;21:209-19.
64. d'Orban PT. Women who kill their children. Br J Psychiatry 1979;134:560-71.
65. Coughlin A. Excusing women. 82 Cal L Rev 1994;1:6.
66. Guileyardo JM, Prahlow JA, Barnard JJ. Familial filicide and filicide classification. Am J Forensic Med Pathol 1999;20:286-92.
67. Chandra PS, Bhargavaraman RP, Raghunandan VN et al. Delusions related to infant and their association with mother-infant interactions in postpartum psychotic disorders. Arch Women Ment Health 2006;9:285-8.
68. Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression: development of the 10-item Edinburgh Postnatal Depression Scale. Br J Psychiatry 1987;150:782-6.
69. Ryan D, Milis L, Misri N. Depression during pregnancy. Can Fam Physician 2005;51:1087-93.
70. Altshuler LL, Hendrick V, Cohen LS. Course of mood and anxiety disorders during pregnancy and the postpartum period. J Clin Psychiatry 1998;59(Suppl. 2):29-33.
71. McCleary R, Chew KSY. Winter is the infanticide season. Homicide Studies 2002;6:228-39.
72. Palermo MT. Preventing filicide in families with autistic children. Int J Offender Ther Comp Criminol 2003;47:47-57.

Diagnosis and management of binge eating disorder

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This paper addresses current issues regarding the diagnosis and management of binge eating disorder (BED). Controversies in diagnosis include the lack of empirically validated criteria, the lack of a universally recognized operational definition of a "binge episode", and the lack of age-appropriate assessment instruments in light of growing reports of BED among children and adolescents. For adults with BED, several pharmacological and behavioral treatments have shown promise in reducing binge frequency and related psychological symptoms of disordered eating (i.e., disinhibition, hunger, depressed mood). Second-generation antidepressants and cognitive behavioral therapy are among the most widely studied treatments. However, no behavioral interventions have demonstrated efficacy with respect to weight loss (which is a critical concern for many BED sufferers who are overweight). Furthermore, randomized controlled trials for BED have been plagued by high drop out and placebo response rates, as well as by insufficient follow-up after active treatment ends to determine long-term outcomes. Therefore, the long-term utility of the various intervention strategies studied thus far remains unclear. More research is needed on innovative medications and behavioral treatments that explore novel modalities to reduce the subjectively reinforcing properties of binge eating. In addition, expanded use of information technologies may be particularly instrumental in the treatment of patients who experience marked shame, denial, and interpersonal deficits, or who face limited access to specialty care. Ultimately, examining BED within the broader context of the current obesity epidemic will be an important area of study.

Key words: Binge eating disorder, diagnostic criteria, antidepressants, behavioral therapy, information technologies

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The symptom of binge eating was first identified by Stunkard in 1959 (1). However, the syndrome of binge eating disorder (BED) has not yet achieved official diagnostic recognition and remains a syndrome in need of further study in the DSM-IV-TR (2).

DIAGNOSIS OF BED: CONTROVERSIES AND EVOLVING ASSESSMENT STRATEGIES

The diagnostic criteria for BED are listed in Table 1. Similar to bulimia nervosa (BN), the definition of a binge eating episode requires the consumption of an unusually large amount of food coupled with a sense of feeling out of control. Also as in BN, the frequency criterion is twice per week, although this criterion is not well supported by the literature for BN and has not been validated for BED (3,4). Where BN and BED diverge is that individuals with BED do not regularly engage in compensatory behaviors (i.e., purging, laxative abuse, excessive exercise), although the precise boundary between BED and non-purging BN is far from clear. In addition, to meet criteria for BED, the binge episodes are associated with at least three of the following criteria: a) eating more rapidly than normal; b) eating when not physically hungry; c) eating until uncomfortably full; d) eating alone because of shame; and e) feeling disgusted with oneself, depressed or guilty after overeating (2). Finally, the individual experiences marked distress regarding binge eating. Although some of these criteria date back to the DSM-III criteria for bulimia, none have been empirically validated for BED.

Given the concern with proliferation of categories in the DSM, experts have proposed guidelines to consider before adding a syndrome to the DSM. Blashfield et al (5) proposed five taxonomic guidelines: a) there should be sufficient journal and empirical articles published on the proposed syndrome within the last 10 years; b) explicit diagnostic criteria should have been proposed in the literature and measurement procedures exist for assessing the syndrome; c) at least two empirical studies (by independent research groups) demonstrate good inter-rater reliability; d) the category represents a syndrome of frequently co-occurring symptoms; and e) at least two independent, empirical studies demonstrate that the proposed category can be differentiated from other categories with which it may be con-

Table 1 DSM-IV-TR diagnostic criteria for binge eating disorder

A. Recurrent episodes of binge eating
An episode of binge eating is characterized by both of the following:
1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances
2. The sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. Binge-eating episodes are associated with three (or more) of the following:
1. Eating much more rapidly than normal
2. Eating until feeling uncomfortably full
3. Eating large amounts of food when not feeling physically hungry
4. Eating alone because of being embarrassed by how much one is eating
5. Feeling disgusted with oneself, depressed, or very guilty after overeating

C. Marked distress regarding binge eating is present

D. The binge eating occurs, on average, at least 2 days a week for 6 months (Note: The method of determining frequency differs from that used for bulimia nervosa; future research should address whether the preferred method of setting a frequency threshold is counting the number of days on which binges occur or counting the number of episodes of binge eating)

E. The binge eating is not associated with the regular use of inappropriate compensatory behavior (e.g., purging, fasting, excessive exercise, etc.) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa

fused. Although substantial work has been done on BED, not all of these guidelines have been adequately addressed.

Over the past decade, the magnitude of research focusing on BED has increased substantially (6). A variety of self-report inventories, such as the Binge Eating Scale (BES) (7), the Three Factor Eating Questionnaire (8) and the Body Shape Questionnaire (9), as well as interview methods, such as the Structured Clinical Interview for the Diagnosis of DSM Disorders (SCID, 10) and the Eating Disorders Examination (EDE) (11), have been developed to assess binge eating in adults. However, attempts are still ongoing to refine the definition of a binge episode and to develop valid and reliable diagnostic criteria for BED. Researchers and clinicians are often unsuccessful in assessing what is an *unusually large amount of food* (12). First, they are inconsistent in recognizing bouts of overeating from grazing (i.e., eating continuously throughout the day instead of eating planned meals) and in deciphering what constitutes a truly large portion size from normal behavior, overindulgence, or circumstances (e.g., holiday). These inconsistencies make it difficult to determine the true number of binge episodes experienced by a patient or research participant. Second, researchers and clinicians (as well as patients) are unreliable in determining if loss of control was present during the binge eating episode (12). Because of subjective differences in the definition, loss of control is difficult to measure. Some individuals may report loss of control after eating a small amount of food (e.g., one cookie), whereas others may only experience a sense of loss of control after a much larger amount of food (e.g., a box of cereal). The EDE has a method for classifying types of overeating. An objective binge episode is one in which the amount eaten would be defined as relatively large (judged by the interviewer) and includes the patient's report of loss of control during the episode. A subjective binge episode is not viewed as large by the interviewer but the patient still reports loss of control. For example, the patient may have eaten a regular size candy bar but may have intended to only eat half of it. Alternatively, subjects may be classified with overeating episodes (either objective or subjective) when they do not experience loss of control over eating.

Although initially conceptualized primarily to be a disorder of adulthood, there is growing recognition that BED also occurs in adolescents and children. Such recognition has propelled the development of age-appropriate and age-relevant assessment measures. Assessment measures for children include the Eating Disorders Examination adapted for children (ChEDE) (13) and the Questionnaire of Eating and Weight Patterns - Adolescent version (14). Researchers have posited that broader, flexible criteria be used to measure BED in children (15-17), and Marcus and Kalarchian (15) recently proposed provisional criteria for measuring BED in children (see Table 2) based on a review and synthesis of findings from previous research studies. On the basis of these criteria, Shapiro et al (18) developed a brief structured, interviewer-administered scale (Children's Binge Eating Disorder Scale, C-BEDS) to measure BED in children aged 5-13. Results showed a strong association between diagnoses from the C-BEDS and SCID. However, the C-BEDS may be more developmentally appropriate for children and better able to identify subsyndromal BED. If used by physicians and other health providers, this brief measure may assist with identifying early onset binge eating behaviors and avoiding the associated consequences, including adult BED, obesity, and associated comorbidities.

Table 2 Provisional research criteria for diagnosing binge eating disorder in children (from 15)

A. Recurrent episodes of being eating
An episode of binge eating is characterized by both of the following:
1. Food seeking in absence of hunger (e.g. after a full meal)
2. A sense of lack of control over eating (e.g., endorse that "When I start to eat, I just can't stop")

B. Binge episodes are associated with one or more of the following:
1. Food seeking in response to negative affect (e.g., sadness, boredom, restlessness)
2. Food seeking as a reward
3. Sneaking or hiding food

C. Symptoms persist over a period of 3 months

D. Eating is not associated with the regular use of inappropriate compensatory behaviors (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa

MANAGEMENT OF BED

Goals for treatment

The primary goal for BED treatment is to achieve abstinence from binge eating. In overweight individuals with BED, treatment goals are often twofold: abstinence from binge eating and sustainable weight loss. Given comorbidity profiles, treatment must also often target anxiety and depression commonly associated with BED.

The literature on BED treatment covers a wide range of putative therapeutic agents and modalities. Those with the most substantial empirical support to date include the use of certain medications and certain behavioral interventions, alone or in combination. Evidence supporting self-help and other approaches is less strong (19,20).

Treatment approaches

Pharmacotherapy

The medications most widely studied thus far in randomized controlled trials (RCTs) include second-generation antidepressants (21-25), tricyclic antidepressants (26), anticonvulsants (27), and sibutramine (28). However, the majority of published RCTs have been limited in scope,

with samples being relatively small (fewer than 500 total participants in eight medication RCTs comprising primarily Caucasian women over age 18).

Among selective serotonin reuptake inhibitors (SSRIs), fluoxetine and fluvoxamine have received the most attention thus far. After 12 weeks, both fluoxetine (average dose 71.3 mg/day) (21) and fluvoxamine (average dose 239 mg/day) (22) were associated with reduced binge frequency and depressed mood. Using a larger sample (85 BED patients) but a shorter treatment period (9 weeks), Hudson et al (24) reported a significantly greater rate of reduction in binge frequency and body mass index (BMI) as well as greater improvement in illness severity with fluvoxamine (50-300 mg/day) compared to placebo. However, fluvoxamine did not demonstrate superiority over placebo in terms of remission rate or change in depression scores. Moreover, endpoint BMI was not reported. Thus, the group receiving fluvoxamine experienced more rapid reductions in binge eating and weight than the placebo group, but these changes did not appear to yield clinically significant effects with respect to binge abstinence and weight loss.

Sertraline and citalopram also show some promise in the treatment of BED. In two 6-week treatment trials, McElroy et al studied the effects of sertraline (mean dose 187 mg/day) versus placebo (23) and citalopram (40-60 mg/day) versus placebo (25) on binge frequency, weight, and mood in individuals with BED. Compared with placebo, both sertraline and citalopram were associated with reduced binge eating, weight loss, and illness severity ratings, but neither medication was clearly superior to placebo in terms of remission rate, and the initial rapid response in binge eating observed with citalopram was not sustained over time. Citalopram, but not sertraline, was associated with reduced depression ratings compared to placebo.

Tricyclic antidepressants are also of interest in the treatment of BED. Laederach-Hoffmann et al (26) studied 31 overweight individuals with BED over a 32-week period, providing standard bi-weekly diet counseling and psychological support augmented with either imipramine (25 mg three times a day) or placebo. At 8 and 32 weeks, binge eating episodes, depressed mood, and body weight decreased significantly in the imipramine-treated group. However, abstinence rates from binge eating were not reported.

Medications that suppress appetite directly or that are associated with weight loss as a side effect have also been examined in the treatment of BED. Examples include the anticonvulsant agent topiramate, which is associated with weight loss in some patients, and sibutramine, which is marketed for the treatment of obesity. In a recent study, topiramate (average dose 212 mg/day) was administered for 14 weeks to obese individuals with BED with a score greater than 15 on the Yale-Brown Obsessive Compulsive Scale for Binge Eating (YBOCS-BE) (27). Relative to placebo, topirimate yielded a significantly greater percentage reduction in binge episodes, binge days per week, and YBOCS-BE score, but did not differ with respect to weight loss, illness severity, or depression. Appolinario et al (28) studied the effects of 12 weeks of sibutramine treatment (15 mg/day) in individuals with BED and a BES score of at least 17. The sibutramine-treated group showed significantly greater decreases in binge days per week, BES scores, and self-reported depression scores compared to the placebo group. At week 12, the sibutramine group had lost on average 7.4 kg, whereas the placebo group had gained weight.

In summary, pharmacotherapy can be useful in the treatment of BED. Specifically, certain second-generation antidepressants, anticonvulsants, and anti-obesity medications have been associated with reduced binge frequency and in some cases reduced negative affect in individuals with BED. However, overall, studies have been hampered by high drop out and placebo response rates and by the failure to measure abstinence as a primary outcome and to report long-term post-intervention data. These limitations make it difficult to estimate the magnitude of clinical significance of any observed effects attributed to medication. Further study will be needed to determine the full utility and limitations of pharmacotherapy in the treatment of BED.

Behavioral therapy

Cognitive-behavioral therapy (CBT) has been the most commonly tested behavioral therapeutic approach for BED (29-31). Other approaches include dialectical behavior therapy (DBT), self-help, exercise, and virtual reality therapy (32-38). Studies have examined the effect of CBT alone as well as in combination with other manipulations concerning the level of spousal or therapist involvement or complementary body exposure treatment. The majority of behavioral therapy trials have enrolled relatively small samples of individuals with BED, usually female and over 18 years of age.

CBT for BED is rooted in the idea that inaccurate thoughts (about body image, for example) lead to inappropriate food consumption (i.e., excess quantity in a short time with accompanying feelings of loss of control), and that learning to adjust or restructure one's binge-triggering thoughts can reduce binge behavior. CBT can be delivered one-on-one or in a group setting, independently or in combination with other psychotherapy approaches.

Several studies have shown that CBT reduces binge frequency, related psychological aspects of binge eating (restraint, disinhibition, and hunger), depressed mood, and ratings of illness severity in individuals with BED (29-31). CBT may also increase the likelihood of abstinence from binge eating (31). However, CBT does not appear to lead to significant changes in body weight. Moreover, augmentation strategies such as CBT plus increased spousal involvement with therapy (31) or body exposure treatment (30) have not revealed any clear advantages over CBT alone. Thus, as currently conceptualized, CBT may be effective in helping patients improve their sense of control over binge eating behavior, but not over their weight concerns.

DBT fosters the development of skills in the domains of mindfulness, emotion regulation, interpersonal effectiveness, and distress tolerance. One study suggests that DBT principles may be useful in the management of BED. Telch et al (32) studied 20 weeks of DBT versus waiting list control in 44 women with DSM-IV BED. DBT led to greater reduction in binge days and binge episodes and in weight, shape, and eating concerns. However, the two groups did not differ in weight loss or in change in depression or anxiety.

Several studies have examined the effect of self-help strategies in BED. Interventions have been delivered in various formats, including with and without a facilitator or therapist, with or without structure, etc. Carter and Fairburn compared self-help using a book (33) with waiting list control in 72 women with BED and weekly binges (34). Self-help (both with and without a facilitator) led to greater reductions in the mean number of binge days and in clinical severity, while also improving abstinence and cessation rates and EDE scores. However, self-help did not produce significant weight loss in either group. Adding a facilitator had no appreciable effect over self-help alone. Similarly, Peterson et al (35,36) found self-help, regardless of the degree of facilitator involvement, to be beneficial in terms of reduced binge behavior, improved eating attitudes, and higher abstinence rates, but not in terms of reducing depression scores or BMI.

Other "alternative" approaches such as exercise (37,38) and virtual reality therapy (39) are beginning to be explored in the treatment of BED, but currently sufficient data do not exist to make any recommendations.

In summary, behavioral therapies offer some promise in the treatment of BED. CBT, DBT and self-help approaches are all associated with reductions in binge behavior, but the clinical significance of these findings remains uncertain in the absence of data on abstinence during active treatment and longer-term follow-up. Moreover, as examined to date, these behavioral therapies do not result in marked weight loss, which is a critical concern for the significant number of BED sufferers who are overweight. Somewhat paradoxically, there is some indication that drop out during self-help intervention may be inversely related to the degree of involvement by a professional facilitator/therapist. Further studies are needed to clarify these observations.

Combining pharmacotherapy and behavioral therapy

Several studies have examined the potential benefit of combining medication with behavioral treatment vs. either therapy delivered alone in the management of BED.

In their 16-week trial, Grilo et al (40) compared fluoxetine (60 mg/day) versus placebo either alone or with CBT. Results indicated that CBT plus fluoxetine (as well as CBT alone) was superior to fluoxetine alone and placebo in remission rate and in reducing binge frequency, eating and shape concerns, disinhibition, and depression. Weight loss did not differ across groups, however.

Agras et al (41) evaluated the effects of traditional weight loss therapy alone vs. CBT supplemented with weight loss therapy vs. CBT supplemented with weight loss therapy plus desipramine (300 mg/day). Binge eating was significantly reduced after 12 weeks in both groups receiving CBT; however, this effect did not persist at 36 weeks of treatment. Average weight loss was greatest in the weight loss therapy group in the early stages of treatment, but over time (i.e., at 3-month follow-up) the group receiving desipramine lost the most weight. Desipramine showed no clear advantage in reducing symptoms of depression.

Grilo et al (42) investigated the effect of CBT alone and in combination with the lipase inhibitor orlistat (120 mg three times/day) in 50 obese individuals with BED. CBT plus orlistat was associated with greater initial weight loss and a greater remission rate after 12 weeks of treatment. However, these potential benefits were not accompanied by any improvements in eating-related measures or depression, and they were not maintained at 2-month follow-up.

Taken together, these studies suggest that augmentation of CBT with certain medications may provide additional benefit over CBT alone or medication alone strategies in the early stages of BED treatment. However, the long-term benefit of such combined approaches is less certain.

Treatment harms and factors contributing to treatment efficacy

Throughout the BED treatment literature, the most commonly reported harms were those associated with the side effects of second-generation antidepressants, such as sedation, dry mouth, headache, and sexual dysfunction/decreased libido (43). In the studies reviewed here, for example, compared to placebo, insomnia was more pronounced in those receiving fluvoxamine or sertraline, and constipation was more pronounced in those receiving sibutramine or imipramine. Other side effects, such as nausea, sweating and fatigue, dry mouth, blurred vision, and gastrointestinal upset, were also reported. Notably, 24% of individuals treated with desipramine and 20% of individuals treated with topiramate dropped out due to medication side effects. Harms associated with psychotherapy are not reported as frequently, but may include increased mood dysregulation after cessation of active treatment, and should be monitored and given appropriate attention.

Specific factors that contribute to treatment efficacy in BED are not well understood. Limited data suggest that early abstinence from binge eating is associated with significantly greater weight loss (41), and that higher initial self-esteem may account for a small but significant percent of variance in outcome (33). There are deficiencies in the research literature regarding treatment efficacy by subgroups, as well. In general, males, ethnic minorities, and children are understudied. Initial findings require replication, and larger more culturally diverse samples need to be

studied before an accurate picture of individual difference factors in BED outcome can emerge.

Treatment drop out and placebo response

Our understanding of treatment options for BED is limited by several consistent methodological problems in the research literature: drop out and placebo response rates that are often high and unevenly distributed across treatment groups, and the failure to report abstinence rates and long-term follow-up data. Among pharmacotherapy trials reviewed here, drop out rates ranged from a low of 7% (with imipramine) to a high of 57% (with fluoxetine); rates for citalopram (16%), fluvoxamine (20%), sibutramine (23%), orlistat (24%), sertraline (28%), and topiramate (47%) were intermediate. Placebo response rates were also highly variable (6% to 39%). In psychotherapy trials, drop out was also extensive and highly variable, and not always consistent with hypothesized effects of the therapeutic manipulation (i.e., facilitator involvement). Across studies, drop out from CBT (14% to 34% in studies reviewed), DBT (18%), and self-help (0% to 27%) was on par with or perhaps slightly lower compared to certain pharmacotherapies, particularly fluoxetine, suggesting good acceptability of these treatment approaches to most patients. Evidence that combination therapies are more or less acceptable and tolerable for patients is mixed, including improved drop out from weight loss therapy plus CBT vs. weight loss alone (41), but not from treatment with fluoxetine plus CBT vs. fluoxetine alone (40). Lastly, the vast majority of published treatment trials have not followed participants for extended periods after acute treatment ends, so that the utility of these interventions in the long-term management of BED is uncertain.

CONCLUSIONS

Several issues regarding the diagnosis and management of BED remain open to research. Controversies in diagnosis include the lack of empirically validated criteria, the lack of a universally recognized operational definition of a "binge episode", and the lack of age-appropriate assessment instruments to be used in children and adolescents with BED.

Short-term, placebo-controlled medication-only trials provide limited evidence that SSRIs can be useful in reducing target eating, psychiatric, and weight symptoms in individuals with BED. However, this evidence must be viewed tentatively, because it is derived from a collection of studies plagued by high drop out and placebo response rates. Non-SSRI agents such as sibutramine and topiramate may also be beneficial in terms of weight reduction among individuals with BED, but definitive conclusions about their longer-term clinical utility await further details regarding abstinence and remission. Similarly, more studies are needed to confirm the therapeutic potential of low-dose imipramine to augment more traditional weight management and psychotherapy strategies.

In terms of behavioral interventions, CBT is effective in reducing binge frequency (whether reported as binge days or binge episodes) and in improving the psychological features of BED such as restraint, hunger, and disinhibition. CBT's effect on binge frequency, in particular, apparently leads to greater rates of sustained (up to four months after treatment) abstinence. The validity of CBT for reducing symptoms of depression in this patient population is unclear. Likewise, additional studies are needed to confirm the findings of decreased binge eating, eating-related psychopathology, and negative mood with DBT. Self-help approaches may provide viable alternatives, as they have shown efficacy in decreasing binge eating and key psychological features associated with BED. Abstinence from binge eating may hinge on treatment expectancies about weight loss and improved mood – the practitioner must be savvy about these treatment limitations, convey them through patient education, and monitor their impact on long-term adherence. Although non-weight focused behavioral strategies may not promote significant weight loss, they may be associated with less weight gain over time in individuals with BED. The importance of weight maintenance vs. weight loss or gain in treatment adherence and remission warrants further study. Finally, to date, most behavioral studies have suffered from marked drop out, thus our understanding of CBT as well as other behavioral therapies for the treatment of BED is still limited. Specific unaddressed questions include whether calories previously consumed as binges become distributed over nonbinge meals after treatment, which would contribute to the absence of weight change in CBT, and whether treatment alters the way in which patients label binges and nonbinge meals.

Questions remain as to the added benefit of combining pharmacological and psychotherapy approaches (i.e., medication plus CBT), which improve both binge eating and weight loss outcomes. Specifically, additional studies are needed to determine which medications given under which circumstances and to which patients optimally produce and maintain weight loss. Because weight-loss medications generally exert their effects only during active treatment (44), questions remain about pharmacotherapy duration and its relation to remission of behavioral and psychological symptoms and to long-term weight outcome. In addition, further studies are needed to better understand factors that serve as binge triggers (i.e., food cravings, mood) (45).

In order to move our understanding of BED treatment forward, new methods that enhance motivation and retention in medication trials need to be developed, and optimal strategies for maintaining treatment gains must be determined. The metric by which we evaluate treatment success must also be refined and standardized to focus on abstinence from binge eating (not merely reduced binge frequency) as the critical outcome. In addition, abstinence

should be evaluated independent of weight loss, yet weight loss must not be overlooked as a potentially significant moderator of long-term adherence and treatment satisfaction. Studies that target relapse prevention in BED also warrant a high research priority. Future studies should also carefully document and control for placebo response, which has been shown to be high (yet possibly transitory) in BED (22,46,47). Advances regarding treatment-resistant BED patients are likely to build on lessons learned from recent depression-treatment trials regarding potential drug augmentation and sequential medication benefits (48), and from ongoing and future studies targeting CBT non-responders. Finally, additional studies of DBT (for example, which can articulate which aspects of DBT are most applicable to the complex emotional and behavioral features of BED) are warranted.

Research is needed on innovative medications and behavioral treatments that explore novel modalities to reduce the subjectively reinforcing properties of binge eating. This likely includes new information technologies (such as e-mail, the Internet, personal digital assistants, text messaging, and other technological advances) that can be used to enhance treatment, particularly for those patients experiencing shame, denial, and interpersonal deficits or facing limited availability of specialty care. Our group (49) recently compared preliminary feasibility and acceptability of CD-ROM-delivered cognitive-behavioral therapy (CD-ROM CBT) to 10 weekly group CBT sessions and to a waiting list control in 66 overweight individuals with BED. Results were promising in terms of drop out and continued use of the CD after treatment. Also, the majority of waiting list participants elected to receive CD-ROM CBT over group CBT treatment at the end of the waiting period. Thus, preliminarily, CD-ROM appears to be an acceptable and at least initially preferred method of CBT delivery for overweight individuals with BED. The use of technology as a means of treatment delivery is emerging (50); further studies are needed in order to bridge the gap between clinical research and population-based delivery for the treatment of BED.

In summary, individuals with BED can benefit from pharmacotherapy and psychotherapy both alone and in combination. Greater clarity is required to determine how best to achieve both abstinence from binge eating and sustainable weight loss. As these stories unfold, important insights will likely emerge regarding BED and its place within the broader context of our current obesity epidemic (51,52).

References

1. Stunkard AJ. Eating patterns and obesity. Psychiatr Q 1959;33: 284-95.
2. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th ed., text revision (DSM-IV-TR). Washington: American Psychiatric Association, 2000.
3. Garfinkel P, Lin E, Goering P et al. Bulimia nervosa in a Canadian community sample: prevalence and comparison of subgroups. Am J Psychiatry 1995;152:1052-8.
4. Sullivan PF, Bulik CM, Kendler KS. The epidemiology and classification of bulimia nervosa. Psychol Med 1998;28:599-610.
5. Blashfield RK, Sprock J, Fuller AK. Suggested guidelines for including or excluding categories in the DSM-IV. Compr Psychiatry 1990;31:15-9.
6. Walsh BT. The current status of binge eating disorder. Int J Eat Disord 2003;34:S1.
7. Gormally J, Black S, Daston S et al. The assessment of binge eating severity among obese persons. Addict Behav 1982;7:47-55.
8. Stunkard AJ, Messick S. The Three Factor Eating Questionnaire to measure dietary restraint, disinhibition, and hunger. J Psychosom Res 1985;29:71-81.
9. Cooper PJ, Taylor MJ, Cooper Z et al. The development and validation of the Body Shape Questionnaire. Int J Eat Disord 1987; 6:485-94.
10. First M, Spitzer R, Gibbon M et al. Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Patient Edition. New York: Biometrics Research, New York State Psychiatric Institute, 1997.
11. Fairburn CG, Cooper Z. The Eating Disorder Examination (12th ed.). In: Fairburn CG, Wilson GT (eds). Binge eating: nature, assessment, and treatment. New York: Guilford, 1993:317-60.
12. Cooper Z, Fairburn CG. Refining the definition of binge eating disorder and nonpurging bulimia nervosa. Int J Eat Disord 2003; 34:S89-S95.
13. Bryant-Waugh R, Cooper P, Taylor C et al. The use of the Eating Disorder Examination with children: a pilot study. Int J Eat Disord 1996;19:391-7.
14. Johnson W, Grieve F, Adams C et al. Measuring binge eating in adolescents: Adolescent and Parent version of the Questionnaire of Eating and Weight Patterns. Int J Eat Disord 1999;26:301-14.
15. Marcus MD, Kalarchian MA. Binge eating in children and adolescents. Int J Eat Disord 2003;34(Suppl.):S47-S57.
16. Nicholls D, Chater R, Lask B. Children into DSM don't go: a comparison of classification systems for eating disorders in childhood and early adolescence. Int J Eat Disord 2000;28:317-24.
17. Decaluwe V, Braet C, Fairburn CG. Binge eating in obese children and adolescents. Int J Eat Disord 2003;33:78-84.
18. Shapiro JR, Woolson W, Hamer RM et al. Evaluating binge eating in children: development of the Children's Binge Eating Scale (C-BEDS). Int J Eat Disord 2007;40:82-9.
19. RTI-UNC Evidence-Based Practice Center. Management of eating disorders. Rockville: Agency for Healthcare Research and Quality, 2006.
20. Brownley KA, Berkman ND, Sedway JA et al. Binge eating disorder treatment: a systematic review of randomized controlled trias. Int J Eat Disord 2007;40:337-48.
21. Arnold LM, McElroy SL, Hudson JI et al. A placebo-controlled, randomized trial of fluoxetine in the treatment of binge-eating disorder. J Clin Psychiatry 2002;63:1028-33.
22. Pearlstein T, Spurell E, Hohlstein LA et al. A double-blind, placebo-controlled trial of fluvoxamine in binge eating disorder: a high placebo response. Arch Women Ment Health 2003;6:147-51.
23. McElroy SL, Casuto LS, Nelson EB et al. Placebo-controlled trial of sertraline in the treatment of binge eating disorder. Am J Psychiatry 2000;157:1004-6.
24. Hudson JI, McElroy SL, Raymond NC et al. Fluvoxamine in the treatment of binge-eating disorder: a multicenter placebo-controlled, double-blind trial. Am J Psychiatry 1998;155:1756-62.
25. McElroy SL, Hudson JI, Malhotra S et al. Citalopram in the treatment of binge-eating disorder: a placebo-controlled trial. J Clin Psychiatry 2003;64:807-13.
26. Laederach-Hofmann K, Graf C, Horber F et al. Imipramine and diet counseling with psychological support in the treatment of obese binge eaters: a randomized, placebo-controlled double-blind study. Int J Eat Disord 1999;26:231-44.
27. McElroy SL, Arnold LM, Shapira NA et al. Topiramate in the treat-

ment of binge eating disorder associated with obesity: a randomized, placebo-controlled trial. Am J Psychiatry 2003;160:255-61.
28. Appolinario JC, Bacaltchuk J, Sichieri R et al. A randomized, double-blind, placebo-controlled study of sibutramine in the treatment of binge-eating disorder. Arch Gen Psychiatry 2003;60:1109-16.
29. Wilfley DE, Welch RR, Stein RI et al. A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. Arch Gen Psychiatry 2002;59:713-21.
30. Hilbert A, Tuschen-Caffier B. Body image interventions in cognitive-behavioural therapy of binge-eating disorder: a component analysis. Behav Res Ther 2004;42:1325-39.
31. Gorin A, Le Grange D, Stone A. Effectiveness of spouse involvement in cognitive behavioral therapy for binge eating disorder. Int J Eat Disord 2003;33:421-33.
32. Telch CF, Agras WS, Linehan MM. Dialectical behavior therapy for binge eating disorder. J Consult Clin Psychol 2001;69:1061-5.
33. Fairburn CG. Overcoming binge eating. New York: Guilford, 1995.
34. Carter JC, Fairburn CG. Cognitive-behavioral self-help for binge eating disorder: a controlled effectiveness study. J Consult Clin Psychol 1998;66:616-23.
35. Peterson CB, Mitchell JE, Engbloom S et al. Group cognitive-behavioral treatment of binge eating disorder: a comparison of therapist-led versus self-help formats. Int J Eat Disord 1998;24:125-36.
36. Peterson CB, Mitchell JE, Engbloom S et al. Self-help versus therapist-led group cognitive-behavioral treatment of binge eating disorder at follow-up. Int J Eat Disord 2001;30:363-74.
37. Levine MD, Marcus MD, Moulton P. Exercise in the treatment of binge eating disorder. Int J Eat Disord 1996;19:171-7.
38. Pendleton VR, Goodrick GK, Poston WS et al. Exercise augments the effects of cognitive-behavioral therapy in the treatment of binge eating. Int J Eat Disord 2002;31:172-84.
39. Riva G, Bacchetta M, Baruffi M et al. Virtual-reality-based multidimensional therapy for the treatment of body image disturbances in binge eating disorders: a preliminary controlled study. IEEE Trans Inf Technol Biomed 2002;6:224-34.
40. Grilo CM, Masheb RM, Wilson GT. Efficacy of cognitive behavioral therapy and fluoxetine for the treatment of binge eating disorder: a randomized double-blind placebo-controlled comparison. Biol Psychiatry 2005;57:301-9.
41. Agras WS, Telch CF, Arnow B et al. Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder: an additive design. Behav Ther 1994;25:225-38.
42. Grilo CM, Masheb RM, Salant SL. Cognitive behavioral therapy guided self-help and orlistat for the treatment of binge eating disorder: a randomized, double-blind, placebo-controlled trial. Biol Psychiatry 2005;57:1193-201.
43. Gartlehner G, Hansen RA, Kahwati L et al. Drug class review on second generation antidepressants: final report. Chapel Hill: Agency for Healthcare Research and Quality, 2006.
44. Yanovski SZ, Yanovski JA. Obesity. N Engl J Med 2002;346:591-602.
45. Stein RI, Kenardy J, Wiseman CV et al. What's driving the binge in binge eating disorder? A prospective evaluation of precursors and consequences. Int J Eat Disord (in press).
46. Jacobs-Pilipski MJ, Wilfley DE, Crow SJ et al. Placebo response in binge eating disorder. Int J Eat Disord 2007;40:204-11.
47. Carter WP, Hudson JI, Lalonde JK et al. Pharmacologic treatment of binge eating disorder. Int J Eat Disord 2003;34:S74-S88.
48. Trivedi MH, Fava M, Wisniewski SR et al. Medication augmentation after the failure of SSRIs for depression. N Engl J Med 2006; 354:1243-52.
49. Shapiro JR, Reba-Harrelson L, Dymek-Valentine M et al. Feasibility and acceptability of CD-ROM-based cognitive-behavioural treatment for binge eating disorder. Eur Eat Disord Rev 2007;15: 175-84.
50. Tate DF, Zabinski MF. Computer and internet applications for psychological treatment: update for clinicians. J Clin Psychol 2004; 60:209-20.
51. Mokdad AH, Bowman BA, Ford ES et al. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001; 286:1195-200.
52. Flegal KM, Carroll MD, Ogden CL et al. Prevalence and trends in obesity among US adults, 1999-2000. JAMA 2002;288:1723-7.

The concept of mental disorder: diagnostic implications of the harmful dysfunction analysis

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What do we mean when we say that a mental condition is a medical disorder rather than a normal form of human suffering or a problem in living? The status of psychiatry as a medical discipline depends on a persuasive answer to this question. The answers tend to range from value accounts that see disorder as a sociopolitical concept, used for social control purposes, to scientific accounts that see the concept as strictly factual. I have proposed a hybrid account, the harmful dysfunction (HD) analysis, that incorporates both value and scientific components as essential elements of the medical concept of disorder, applying to both physical and mental conditions. According to the HD analysis, a condition is a disorder if it is negatively valued ("harmful") and it is in fact due to a failure of some internal mechanism to perform a function for which it was biologically designed (i.e., naturally selected). The implications of this analysis for the validity of symptom-based diagnostic criteria and for challenges in cross-cultural use of diagnostic criteria are explored, using a comparison of the application of DSM diagnostic criteria in the U.S. and Taiwan.

Key words: Psychopathology, diagnosis, nosology, philosophy of psychiatry, mental disorder, harmful dysfunction, cross-cultural diagnosis, validity of diagnostic criteria, false positives

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The concept of mental disorder is at the foundation of psychiatry as a medical discipline, at the heart of scholarly and public disputes about which mental conditions should be classified as pathological and which as normal suffering or problems of living, and has ramifications for psychiatric diagnosis, research, and policy. Although both normal and disordered conditions may warrant treatment, and although psychiatry arguably has other functions beyond the treatment of disorder, still there exists widespread concern that spurious attributions of disorder may be biasing prognosis and treatment selection, creating stigma, and even interfering with normal healing processes. However, no consensus exists on the meaning of "mental disorder". The upcoming revisions of the DSM-IV and ICD-10 offer an opportunity to confront these conceptual issues and improve the validity of psychiatric diagnosis.

I approach this problem via a conceptual analysis that asks: what do we mean when we say that a problematic mental condition, such as adolescent antisocial behavior, a child's defiant behavior toward a parent, intense sadness, intense worry, intense shyness, failure to learn to read, or heavy use of illicit drugs, is not merely a form of normal, albeit undesirable and painful, human functioning, but indicative of psychiatric disorder? The credibility and even the coherence of psychiatry as a medical discipline depends on there being a persuasive answer to this question. The answer requires an account of the concept of disorder that generally guides such judgments.

Among existing analyses of "mental disorder", a basic division is between value and scientific approaches. As Kendell put it: "The most fundamental issue, and also the most contentious one, is whether disease and illness are normative concepts based on value judgments, or whether they are value-free scientific terms; in other words, whether they are biomedical terms or sociopolitical ones" (1). I have proposed a hybrid account, the "harmful dysfunction" (HD) analysis of the concept of mental disorder (2-8). According to the HD analysis, a disorder is a *harmful dysfunction*, where "harmful" is a value term, referring to conditions judged negative by sociocultural standards, and "dysfunction" is a scientific factual term, referring to failure of biologically designed functioning. In modern science, "dysfunction" is ultimately anchored in evolutionary biology and refers to failure of an internal mechanism to perform one of its naturally selected functions.

In this article, I explore the considerable explanatory power of the HD analysis for understanding the distinction between mental disorder and other problematic mental conditions. I also illustrate the implications of the analysis for assessing the validity of DSM and ICD diagnostic criteria, and for understanding some of the conceptual challenges in applying diagnostic criteria across cultures, using the example of transplantation of DSM criteria to Taiwan.

WHY PSYCHIATRY CAN'T ESCAPE THE CONCEPT OF MENTAL DISORDER

The diagnostic criteria of the DSM and the ICD are currently the primary arbiters of what is disordered vs. nondisordered in most clinical practice and research. But they are clearly not conceptually final arbiters. The criteria are regularly revised to make them more valid in indicating disorder and to eliminate false positives, implicitly recognizing that "errors" in the criteria are possible. Moreover, both the popular press and critics within the mental health professions challenge the validity of the criteria in picking out mental disorder, and these disputes do not seem entirely arbitrary, but rather often seem to appeal to an underlying shared notion of disorder. In-

deed, professionals often classify conditions using the "not otherwise specified" category, which requires a sense of what is and is not a disorder independent of specific diagnostic criteria.

Granting the common observation that there is no "gold standard" laboratory test or physiological indicator for mental disorders and that current criteria are fallible, it might still be asked: why must we grapple with the elusive concept of disorder itself when there are so many empirical techniques for identifying disorders? The reality is that all of the tests that are commonly used to distinguish disorder from nondisorder rest on implicit assumptions about the concept of disorder; otherwise, it is not clear whether the test is distinguishing disorder from nondisorder, one disorder from another disorder, or one nondisordered condition from another. Common tests of validity such as statistical deviance, family history/genetic loading, predictive validity, Kendell's discontinuity of distribution, factor analytic validity, construct validity, syndromal co-occurrence of symptoms, response to medication, Robins and Guze criteria, Meehl's taxometric analysis, and all other such guides can identify a valid construct and separate one such construct from another. But whether the distinguished constructs are disorder versus nondisorder goes beyond the test's capabilities. Every such test is equally satisfied by myriad normal as well as disordered conditions. Even the currently popular (in the U.S.) use of role impairment does not inherently distinguish disorder from nondisorder (and for this reason is generally avoided by the ICD), because there are many normal conditions, from sleep and fatigue to grief and terror, that not only impair routine role functioning but are biologically designed to do so. It only *seems* as though these various kinds of empirical criteria provide a stand-alone standard for disorder, because they are used within a context in which disorders – in some background conceptual sense – are already implicitly and independently inferred to exist, and the issue is simply to distinguish among disorders or to distinguish disorder from normality. This essential background assumption itself depends on the concept of disorder being deployed independently of the specific empirical test. Thus, there is no substitute for the concept of mental disorder as the ultimate standard. None of our empirical approaches work without a warrant in a conceptual analysis of disorder.

A further reason why we must rely on the concept of disorder is the lack of definitive etiological understanding of mental disorder and the consequent theoretical fragmentation of psychiatry, and thus the decision in the DSM and the ICD to provide theory-neutral criteria for diagnosing disorders. Etiological theory (e.g., return of the repressed, irrational ideas, serotonin deficit) would generally provide ways to distinguish disorder from nondisorder in a more developed science. The need to rely for now on theory-neutral criteria means that the concept of disorder itself, which is to some extent shared by various theories, offers the best way of judging whether a theory-neutral diagnostic criteria set picks out disorders rather than normal conditions (i.e., is *conceptually valid*) (2). Theory-neutral criteria work to the extent that they adhere to an implicit understanding of disorder versus nondisorder that is shared across most theoretical perspectives and allows a provisional basis for shared identification of disorders for research purposes.

ASSUMPTIONS UNDERLYING THE ANALYSIS OF MENTAL DISORDER

The HD analysis departs from two observations: first, the concept of "disorder" has been around in physical medicine and applied to some mental conditions for millennia and is broadly understood by lay people and professionals; and, second, a central goal of an analysis of "mental disorder" is to clarify and reveal the degree of legitimacy in psychiatry's claims to be a truly medical discipline rather than, as antipsychiatrists and others have claimed, a social control institution masquerading as a medical discipline. The approach to defining "mental disorder" that seems most relevant to the latter goal is a conceptual analysis of the existing meaning of "disorder" as it is generally understood in medicine and society in general, with a focus on whether and how this concept applies to the mental domain. The claim of psychiatry to be a medical discipline depends on there being genuine mental disorders in the same sense of "disorder" that is used in physical medicine. Any proposal to define "mental disorder" in a way unique to psychiatry that does not fall under the broader medical concept of disorder would fail to address this issue. Part of the challenge in resolving this issue is that the medical concept of disorder is itself subject to ongoing dispute. The HD analysis is aimed at addressing this challenge.

Because the analysis here ultimately concerns the general concept of disorder as applied to both mental and physical conditions, examples from both mental and physical domains are used to test the analysis. I use "internal mechanism" as a general term to refer both to physical structures and organs as well as to mental structures and dispositions, such as motivational, cognitive, affective, and perceptual mechanisms. Some writers distinguish between "disorder", "disease", and "illness"; I focus on "disorder" as the broader term that covers both traumatic injuries and diseases/illnesses, thus being closer to the overall concept of medical pathology.

I focus on the question of what makes a mental condition a disorder; I do not address how to delineate mental versus physical disorders. For present purposes, mental processes are simply those like emotion, thought, perception, motivation, language, and intentional action. There is no intended Cartesian implication about any special ontological status of the mental; it is just an identified set of functions and processes.

THE VALUE COMPONENT OF "DISORDER"

As traditional value accounts suggest, a condition is a mental disorder only if it is harmful according to social values and

thus at least potentially warrants medical attention. Medicine in general, and psychiatry in particular, are irrevocably value-based professions. "Harm" is construed broadly here to include all negative conditions.

Both lay and professional classificatory behaviors demonstrate that the concept of mental disorder contains a value component. For example, inability to learn to read due to a dysfunction in the corpus callosum (assuming that this theory of some forms of dyslexia is correct) is harmful in literate societies, but not harmful in preliterate societies, where reading is not a skill that is taught or valued, and thus not a disorder in those societies. Most people have what physicians call "benign anomalies", that is, minor malformations that are the result of genetic or developmental errors but that cause no significant problem, and such anomalies are not considered disorders. For example, benign angiomas are small blood vessels whose growth has gone awry, leading them to connect to the skin, but, because they are not harmful, they are not considered disorders. The requirement that there be harm also accounts for why simple albinism, heart position reversal, and fused toes are not generally considered disorders, even though each results from an abnormal breakdown in the way some mechanism is designed to function. Purely scientific accounts of "disorder", even those based on evolutionary function as is the analysis below (9-11), fail to address this value component.

In the DSM and ICD diagnostic criteria, the symptoms and clinical significance requirement generally ensure harm and that the condition is negatively valued. The dispute remains about whether "mental disorder" is purely evaluative or contains a significant factual component that can discriminate a potential domain of negative conditions that are disorders from those that are nondisorders.

There are many negative conditions that are not disorders, and many of them contain symptoms and are clinically significant in that they cause distress or role impairment (e.g., grief). The distinction between disorders and nondisorders thus seems to depend on some further criterion.

THE FACTUAL COMPONENT OF "DISORDER"

Contrary to those who maintain that a mental disorder is simply a socially disapproved mental condition (12,13), "mental disorder" as commonly used is just one category of the many negative mental conditions that can afflict a person. One needs an additional factual component to distinguish disorders from the many other negative mental conditions not considered disorders, such as ignorance, lack of skill, lack of talent, low intelligence, illiteracy, criminality, bad manners, foolishness, and moral weakness.

Indeed, both professionals and laypersons distinguish between quite similar negative conditions as disorders versus nondisorders. For example, illiteracy is not in itself considered a disorder, even though it is disvalued and harmful in our society, but a similar condition that is believed to be due to lack of ability to learn to read because of some internal neurological flaw or psychological inhibition is considered a disorder. Male inclinations to aggressiveness and inclination to sexual infidelity are considered negative but not generally considered disorders because they are seen as the result of natural functioning, although similar compulsive motivational conditions are seen as disorders. Grief is seen as normal, whereas similarly intense sadness not triggered by real loss is seen as disordered. A pure value account of "disorder" does not explain such distinctions among negative conditions.

Moreover, we often adjust our views of disorder based on cross-cultural evidence that may go against our values. For example, U.S. culture does not value polygamy, but we judge that it is not a failure of natural functioning, thus not disordered, based partly on cross-cultural data.

The challenge, then, is to elucidate the factual component. Based on common usage in the literature, I call this factual component a "dysfunction". What, then, is a dysfunction? An obvious place to begin is with the supposition that a dysfunction implies an unfulfilled function, that is, a failure of some mechanism in the organism to perform its function. However, not all uses of "function" and "dysfunction" are relevant. The medically relevant sense of "dysfunction" is clearly *not* the colloquial sense in which the term refers to failure of an individual to perform well in a social role or in a given environment, as in assertions like "I'm in a dysfunctional relationship" or "discomfort with hierarchical power structures is dysfunctional in today's corporate environment". These kinds of problems need not be individual disorders. A disorder is different from a failure to function in a socially or personally preferred manner precisely because a dysfunction exists only when something has gone wrong with functioning, so that a mechanism cannot perform as it is naturally (i.e., independently of human intentions) supposed to perform.

Presumably, then, the functions that are relevant are "natural functions", about which concept there is a large literature (12-27). Such functions are frequently attributed to inferred mental mechanisms that may remain to be identified, and failures labeled dysfunctions. For example, a natural function of the perceptual apparatus is to convey roughly accurate information about the immediate environment, so gross hallucinations indicate dysfunction. Some cognitive mechanisms have the function of providing the person with the capacity for a degree of rationality as expressed in deductive, inductive, and means-end reasoning, so it is a dysfunction when the capacity for such reasoning breaks down, as in severe psychotic states.

The function of a mechanism is important because of its distinctive form of explanatory power; the existence and structure of the mechanism is explained by reference to the mechanism's effects. For example, the heart's effect of pumping the blood is also part of the heart's explanation, in that one can legitimately answer a question like "why do we have hearts?" or "why do hearts exist?" with "because hearts pump the blood". The effect of pumping the blood also

enters into explanations of the detailed structure and activity of the heart. Talk of "design" and "purpose" in the case of naturally occurring mechanisms is just a metaphorical way of referring to this unique explanatory property that the effects of a mechanism explain the mechanism. So, "natural function" can be analyzed as follows: *a natural function of an organ or other mechanism is an effect of the organ or mechanism that enters into an explanation of the existence, structure, or activity of the organ or mechanism*. A "dysfunction" exists when an internal mechanism is unable to perform one of its natural functions (this is only a first approximation to a full analysis; there are additional issues in the analysis of "function" that cannot be dealt with here (8,21,24)).

The above analysis applies equally well to the natural functions of mental mechanisms. Like artifacts and organs, mental mechanisms, such as cognitive, linguistic, perceptual, affective, and motivational mechanisms, have such strikingly beneficial effects and depend on such complex and harmonious interactions that the effects cannot be entirely accidental. Thus, functional explanations of mental mechanisms are sometimes justified by what we know about how people manage to survive and reproduce. For example, a function of linguistic mechanisms is to provide a capacity for communication, a function of the fear response is to avoid danger, and a function of tiredness is to bring about rest and sleep. These functional explanations yield ascriptions of dysfunctions when respective mechanisms fail to perform their functions, as in aphasia, phobia, and insomnia, respectively.

"Dysfunction" is thus a purely factual scientific concept. However, discovering what in fact is natural or dysfunctional (and thus what is disordered) may be difficult and may be subject to scientific controversy, especially with respect to mental mechanisms, about which we are still largely ignorant. This ignorance is part of the reason for the high degree of confusion and controversy concerning which conditions are really mental disorders. However, functional explanations can be plausible and useful even when little is known about the actual nature of a mechanism or even about the nature of a function. For example, we know little about the mechanisms underlying sleep, and little about the functions of sleep, but circumstantial evidence persuades us that sleep is a normal, biologically designed phenomenon and not (despite the fact that it incapacitates us for roughly one-third of our lives) a disorder; the circumstantial evidence enables us to distinguish some normal versus disordered conditions related to sleep despite our ignorance.

Obviously, one can go wrong in such explanatory attempts; what seems non-accidental may turn out to be accidental. Moreover, cultural preconceptions may easily influence one's judgment about what is biologically natural. But, often one is right, and one is making a factual claim that can be defeated by evidence. Functional explanatory hypotheses communicate complex knowledge that may not be so easily and efficiently communicated in any other way.

Today, evolutionary theory provides the best explanation of how a mechanism's effects can explain the mechanism's presence and structure. In brief, those mechanisms that had effects on the organism that contributed to the organism's reproductive success over enough generations thereby increased in frequency and hence were "naturally selected" and exist in today's organisms. Thus, an explanation of a mechanism in terms of its natural function may be considered a roundabout way of referring to a causal explanation in terms of natural selection. Since natural selection is the only known means by which an effect can explain a naturally occurring mechanism that provides it, evolutionary explanations presumably underlie all correct ascriptions of natural functions. Consequently, an evolutionary approach to mental functioning (7,24) is central to an understanding of psychopathology.

One might object that what goes wrong in disorders is sometimes a social function that has nothing to do with natural, universal categories. For example, reading disorders seem to be failures of a social function, because there is nothing natural or designed about reading. However, illiteracy involves the very same kind of harm as reading disorder, yet it is not considered a disorder. Inability to read is only considered indicative of disorder when circumstances suggest that the reason for the inability lies in a failure of some brain or psychological mechanism to perform its natural function. There are many failures of individuals to fulfill social functions, and they are not considered disorders unless they are attributed to a failed natural function.

If one looks down the list of disorders in the DSM, it is apparent that by and large it is a list of the various ways that something can go wrong with the seemingly designed features of the mind. Very roughly, psychotic disorders involve failures of thought processes to work as designed; anxiety disorders involve failures of anxiety- and fear-generating mechanisms to work as designed; depressive disorders involve failures of sadness and loss-response regulating mechanisms; disruptive behavior disorders of children involve failures of socialization processes and processes underlying conscience and social cooperation; sleep disorders involve failure of sleep processes to function properly; sexual dysfunctions involve failures of various mechanisms involved in sexual motivation and response; eating disorders involve failures of appetitive mechanisms, and so on. There is a certain amount of nonsense in the DSM and criteria are often overly inclusive. However, the vast majority of categories are inspired by conditions that even a lay person would correctly recognize as a failure of designed functioning.

When we distinguish normal grief from pathological depression, or normal delinquent behavior from conduct disorder, or normal criminality from antisocial personality disorder, or normal unhappiness from adjustment disorder, or illiteracy from reading disorder, we are implicitly using the "failure-of-designed-function" criterion. All of these conditions – normal and abnormal – are disvalued and harmful conditions, and the effects of the normal and pathological conditions can be quite similar behav-

iorally, yet some are considered pathological and some not. The natural-function criterion explains these distinctions.

It bears emphasis that even biological conditions that are harmful in the current environment are not considered disorders if they are considered designed features. For example, the taste preference for fat is not considered a disorder, even though in today's food-rich environment it may kill you, because it is considered a designed feature that helped us to obtain needed calories in a previous food-scarce environment. Higher average male aggressiveness is not considered a mass disorder of men even though in today's society it is arguably harmful, because it is considered the way men are designed (of course, there are aggressiveness disorders; here as elsewhere, individuals may have disordered responses of designed features).

In sum, a mental disorder is a harmful mental dysfunction. If the HD analysis is correct, then a society's categories of mental disorder offer two pieces of information. First, they indicate a value judgment that the society considers the condition negative or harmful. Second, they make the factual claim that the harm is due to a failure of the mind to work as designed; this claim may be correct or incorrect, but in any event reveals what the society thinks about the natural or designed working of the human mind.

IMPLICATIONS OF THE HD ANALYSIS FOR VALIDITY OF DIAGNOSTIC CRITERIA

One of the disadvantages of pure social-constructivist views of mental disorder, like antipsychiatric views, is that they offer no place to stand from which to critique current diagnostic criteria and to improve their validity. Once one has a conceptual analysis of disorder that offers a "place to stand" in evaluating whether diagnostic criteria identify disorders, one can consider whether current criteria get the intended distinction right. A distinction central to an adequate assessment is whether the client's problem is a mental disorder or a problem in living that involves a normal though problematic reaction to stressful environmental conditions. The way we think about a case may influence the treatment we think most appropriate, so that, for example, thinking of a client's condition as a mental disorder tends to suggest that something is wrong internally and that the locus of intervention should be the client's mental functioning rather than the client's relationship to the environment. There are many other potentially harmful effects of such misclassification as well, ranging from stigma to confusing research results about etiology and treatment when disordered and nondisordered clients are mixed together.

The international use of DSM-style symptom-based criteria to diagnose mental disorder raises two basic challenges. The first is that symptom-based criteria themselves, even as used within the U.S., fail to take context into account and thus fail to adequately identify conditions due to dysfunctions. Criteria are consequently often too broad and incorrectly include normal reactions under the "disorder" category. Here are three brief examples from earlier work of mine (6,28).

Major depressive disorder

The DSM-IV criteria for major depressive disorder contain an exclusion for uncomplicated bereavement (up to two months of symptoms after loss of a loved one are allowed as normal) but no exclusions for equally normal reactions to other major losses, such as a terminal medical diagnosis in oneself or a loved one, separation from one's spouse, the end of an intense love affair, or loss of one's job and retirement fund. Reactions to such losses may satisfy DSM-IV diagnostic criteria but are not necessarily disorders. If one's reaction to such a loss includes, for example, just two weeks of depressed mood, diminished pleasure in usual activities, insomnia, fatigue, and diminished ability to concentrate on work tasks, then one's reaction satisfies DSM-IV criteria for major depressive disorder, even though such a reaction need not imply pathology any more than it does in bereavement. Clearly, the essential requirement that there be a dysfunction in a depressive disorder – perhaps one in which loss-response mechanisms are not responding proportionately to loss as designed – is not adequately captured by DSM-IV criteria (29,30).

Because of these flaws, the epidemiological data on prevalence of depression can be misleading, yielding potentially inflated estimates of the social and economic costs of depression. Based on international epidemiological studies using symptom-based criteria, the World Health Organization (WHO) has publicized the apparently immense costs of depression. However, the claimed enormity of this burden relative to other serious diseases, and the consequent influence on priorities, may result from the failure to distinguish depressive disorders from normal sadness. The WHO calculations of disease burden are extremely complex, but arise from two basic components: the number of people who suffer from a condition and the amount of disability and premature death the condition causes. The first component of burden, the frequency of the condition, derives from symptom-based definitions that estimate that 9.5% of women and 5.8% of men suffer from depression in a 1-year period. The second component, disability, is ordered into seven classes of increasing severity, stemming from the amount of time lived with a disease weighted by the severity of the disease. The severity scores come from consensual judgments of health workers from around the world that are applied to all cases of the disease. Depression is placed in the second most severe category of illness, behind only extremely disabling and unremitting conditions such as active psychosis, dementia, and quadriplegia, and is considered comparable to the conditions of paraplegia and blindness. This extreme degree of severity assumes that all cases of depression share the depth, chronicity, and recurrence that are characteristic of the severe conditions that health workers see in their practices. But,

the epidemiological studies encompass everyone who meets symptom criteria, a group that, due to the possible confounding of normal sadness with disorder, may be heterogeneous to a greater degree than clinical patients would indicate, yielding an invalid overall estimation of disease burden. Unraveling these confusions could lead to a more optimal distribution of WHO's health resources.

Conduct disorder

The DSM-IV diagnostic criteria for conduct disorder allow the diagnosis of adolescents as disordered who are responding with antisocial behavior to peer pressure, threatening environment, or abuses at home (31). For example, if a girl, attempting to avoid escalating sexual abuse by her stepfather, lies to her parents about her whereabouts and often stays out late at night despite their prohibitions, and then, tired during the day, often skips school, and her academic functioning is consequently impaired, she can be diagnosed as conduct disordered. Rebellious kids or kids who fall in with the wrong crowd and who skip school and repetitively engage in shoplifting and vandalism also qualify for diagnosis. However, in an acknowledgment of such problems, there is a paragraph included in the "Specific culture, age, and gender features" section of the DSM-IV text for conduct disorder which states that "consistent with the DSM-IV definition of mental disorder, the conduct disorder diagnosis should be applied only when the behavior in question is symptomatic of an underlying dysfunction within the individual and not simply a reaction to the immediate social context". If these ideas had been incorporated into the diagnostic criteria, many false positives could have been eliminated. Unfortunately, in epidemiological and research contexts, such textual nuances are likely ignored.

Social phobia

Whereas social phobia is a real disorder in which people can sometimes not engage in the most routine social interaction, current criteria allow diagnosis when someone is, say, intensely anxious about public speaking in front of strangers. But, it remains unclear whether such fear is really a failure of normal functioning or rather an expression of normal range danger signals that were adaptive in the past, when failure in such situations could lead to ejection from the group and a consequent threat to survival. This diagnosis seems potentially an expression of American society's high need for people who can engage in occupations that require communicating to large groups (32,33).

IMPLICATIONS OF THE HD ANALYSIS FOR CROSS-CULTURAL USE OF DIAGNOSTIC CRITERIA

A second problem that arises in the use of symptom-based diagnostic criteria is specific to the international context: due to local cultural conditions, the symptomatic expression of a dysfunction, or the symptomatic indicators of dysfunction versus normality, or the values that determine that a condition is negative, may vary for a great number of reasons. To illustrate this problem, I return to each of the above diagnostic categories and suggest how additional problems might occur in using the DSM criteria for these disorders in the context of Taiwanese society.

Depression

The classic finding is that Asian populations express their depression through an "idiom of distress" that focuses on somatic complaints rather than more mental DSM symptoms (34,35). This poses a challenge in applying DSM criteria. However, the data suggest that, if asked, Asian populations do often report the DSM-type symptoms as well, so that this may be an issue of self-presentation rather than actual variation in the symptomatic expression of a dysfunction. Another issue concerns gender expectations: in Taiwan (especially among older generations), even more than in the U.S., the woman is expected to have primary responsibility for the home, which can be constraining. Folk understanding of female versus male nature tends to allow for a large amount of normal expression of depressive-like misery expressed by women as part of their "natural" life situation and innate tendencies. Different expectations apply to males. Thus, especially in applying DSM criteria to some older women, there might be a challenge in deciding whether the symptoms indicate a disorder (as they might in the U.S.) or are just a culturally sanctioned normal response to difficult circumstances.

Conduct disorder

In Taiwanese society, expectations and supervision of some children and adolescents appear to be more demanding and more rigid than in the U.S.. In some cases, this is because of the academic testing system, in which a youth's entire future may depend on his or her performance on a single test. These factors could affect the interpretation of antisocial behavior in several ways. For example, early misbehavior could more frequently be a normal response to excessive family pressure. On the other hand, some children may not express inherent antisocial tendencies until a later age than would be typical in U.S., because of the greater constraints of the Taiwanese cultural environment. It is also possible that Taiwanese hold a culturally implicit theory of adolescent development that is less accepting of youthful misbehavior as normal than is the American implicit theory, leading to overpathologization.

Social phobia

DSM-IV criteria for social phobia require anxiety only about social interactions with unfamiliar people. One can be perfectly comfortable with one's family and with those one knows, but still be diagnosed with social phobia if he feels anxious in certain situations with

strangers (e.g., public speaking). There may be a strong cultural loading here that poses challenges for the Taiwanese diagnostician. These criteria are influenced by American culture's belief in individuality, independence from family, and open interactions of unfamiliars. In contrast, some Taiwanese, at least of older generations, may have been socialized to think primarily of the family as a safe haven and to see unfamiliar people as requiring more caution. The DSM-IV criteria may potentially pathologize what might be considered normal among Taiwanese given local socialization. It should be emphasized that these observations may apply more to older Taiwanese.

As these examples suggest, the HD analysis allows much room for cross-cultural variation in diagnosis due to many nuanced sources not limited to culture-specific syndromes. However, the HD analysis also reflects the reality that cultures, whatever their values, cannot construct disorders from whole cloth; a culture is only correct in labeling a condition it considers undesirable as a disorder if the condition involves a failure of biologically designed functioning. Thus, cultures can be wrong about whether a condition is a disorder or normal, as Victorian physicians were wrong to think that clitoral orgasm was a disorder, ante-bellum confederate U.S. physicians were wrong to think that slaves who ran away from their slavery were disordered, and some cultures in which schistosomiasis is endemic are wrong to think that its symptoms are part of normal functioning.

CONCLUSIONS

Careful attention to the concept of mental disorder that underlies psychiatry suggests that, contrary to various critics, there is indeed a coherent medical concept of mental disorder in which "disorder" is used precisely as it is in physical medicine. Once this concept is made explicit, it offers a "place to stand" in evaluating whether current symptom-based DSM and ICD diagnostic criteria are accomplishing their goal of identifying psychiatric disorders as opposed to normal problematic mental conditions. I have argued that there is a long way to go in this regard. I suggest that the upcoming revisions of both manuals create a formal mechanism for reviewing each diagnostic criteria set for possible conceptual flaws leading to false positives, so that psychiatric diagnosis need not be afflicted by manifest weaknesses that are apparent to the press and the lay public yet go ignored by the profession.

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References

1. Kendell RE. What are mental disorders? In: Freedman AM, Brotman R, Silverman I et al (eds). Issues in psychiatric classification: science, practice and social policy. New York: Human Sciences Press, 1986: 23-45.
2. Wakefield JC. The concept of mental disorder: on the boundary between biological facts and social values. Am Psychol 1992; 47:73-88.
3. Wakefield JC. Disorder as harmful dysfunction: a conceptual critique of DSM-III-R's definition of mental disorder. Psychol Rev 1992;99:232-47.
4. Wakefield JC. Limits of operationalization: a critique of Spitzer and Endicott's (1978) proposed operational criteria for mental disorder. J Abnorm Psychol 1993;102:160-72.
5. Wakefield JC. DSM-IV: are we making diagnostic progress? Contemporary Psychology 1996;41:646-52.
6. Wakefield JC. Diagnosing DSM, Part 1: DSM and the concept of mental disorder. Behav Res Ther 1997;35:633-50.
7. Wakefield JC. Evolutionary versus prototype analyses of the concept of disorder. J Abnorm Psychol 1999;108:374-99.
8. Wakefield JC. Disorder as a black box essentialist concept. J Abnorm Psychol 1999; 108:465-72.
9. Boorse C. On the distinction between disease and illness. Philosophy and Public Affairs 1975;5:49-68.
10. Boorse C. What a theory of mental health should be. J Theory Soc Behav 1976;6: 61-84.
11. Boorse C. Health as a theoretical concept. Philosophy of Science 1977;44.
12. Houts AC. Harmful dysfunction and the search for value neutrality in the definition of mental disorder: response to Wakefield, part 2. Behav Res Ther 2001;39:1099-132.
13. Sedgwick P. Psycho politics. New York: Harper & Row, 1982.
14. Boorse C. Wright on functions. Philos Rev 1976;85:70-86.
15. Cummins R. Functional analysis. J Philos 1975;72:741-65.
16. Elster J. Explaining technical change. Cambridge: Cambridge University Press, 1983.
17. Hempel CG. The logic of functional analysis. In: Hempel CG (ed). Aspects of scientific explanation and other essays in the philosophy of science. New York: Free Press, 1965:297-330.
18. Klein DF. A proposed definition of mental illness. In: Spitzer RL, Klein DF (eds). Critical issues in psychiatric diagnosis. New York: Raven, 1978:41-71.
19. Moore MS. Discussion of the Spitzer-Endicott and Klein proposed definitions of mental disorder (illness). In: Spitzer RL, Klein DF (eds). Critical issues in psychiatric diagnosis. New York: Raven, 1978: 85-104.
20. Nagel E. Teleology revisited and other essays in the philosophy and history of science. New York: Columbia University Press, 1979.
21. Wakefield JC. Aristotle as sociobiologist: the 'function of a human being' argument, black box essentialism, and the concept of mental disorder. Philosophy, Psychiatry, and Psychology 2000;7:17-44.
22. Wakefield JC. Spandrels, vestigial organs, and such: reply to Murphy and Woolfolk's "The harmful dysfunction analysis of mental disorder". Philosophy, Psychiatry, and Psychology 2000;7:253-70.
23. Wakefield JC. Dysfunction as a factual component of disorder: reply to Houts, part 2. Behav Res Ther 2003;41:969-90.
24. Wakefield JC. Biological function and dysfunction. In: Buss D (ed). Handbook of evolutionary psychology. New York: Oxford Press, 2005:878-902.
25. Woodfield A. Teleology. Cambridge: Cambridge University Press, 1976.
26. Wright L. Functions. Philos Rev 1973;82: 139-68.
27. Wright L. Teleological explanations. Berkeley: University of California Press, 1976.
28. Wakefield JC, First M. Clarifying the distinction between disorder and non-disorder: confronting the overdiagnosis ('false positives') problem in DSM-V. In: Phillips KA, First MB, Pincus HA (eds). Advancing DSM: dilemmas in psychiatric diagnosis. Washington: American Psychiatric Press, 2003:23-56.

29. 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.
30. Horwitz AV, Wakefield JC. The loss of sadness: how psychiatry transformed normal sorrow into depressive disorder. New York: Oxford University Press, 2007.
31. Wakefield JC, Pottick KJ, Kirk SA. Should the DSM-IV diagnostic criteria for conduct disorder consider social context? Am J Psychiatry 2002;159:380-6.
32. Wakefield JC, Horwitz AV, Schmitz M. Social disadvantage is not mental disorder: response to Campbell-Sills and Stein. Can J Psychiatry 2005;50:324-6.
33. Wakefield JC, Horwitz AV, Schmitz M. Are we overpathologizing social anxiety?: social phobia from a harmful dysfunction perspective. Can J Psychiatry 2005;50:317-9.
34. Kleinman AM. Rethinking psychiatry. New York: Free Press, 1988.
35. Lin KM, Lin M, Zheng Y. Neurasthenia and chronic fatigue syndrome: lessons from cross-cultural study. In: Yilmaz AT, Weiss MG, Riecher-Rossler A (eds). Cultural psychiatry: euro-international perspectives. Basel: Karger, 2001:68-80.

Does psychiatry need an overarching concept of "mental disorder"?

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Since 1992 (1), Jerome Wakefield has been expounding, with minor modifications, a persuasive and influential point of view on the concept of mental disorder as "harmful dysfunction" (HD), which postulates a conjunction of a value term (harm) and a factual scientific term (dysfunction). This "hybrid" definition resolves the previously irrevocable polarity between the "social-constructivist" position (mental disorder is a value-laden social construct with no counterpart in biomedical reality) and the "objectivist" position (mental disorders are natural entities that could be understood in biological terms). In the HD concept, the relativism of the social definition of "harm" is counterbalanced by a factual component of a malfunctioning internal mechanism causing objective dysfunction. Wakefield believes that the HD concept will provide psychiatry with an "ultimate standard" of what constitutes mental disorder and that this is essential to the credibility and coherence of psychiatry as a medical discipline. A notable merit of the application of the HD concept so far has been in the demonstration of the fallacies of the social-constructivist view and in the incisive critique of the "atheoretical" platform of DSM-III and its subsequent editions, as well as of the arbitrariness and over-inclusiveness of some of its categories.

Notwithstanding all this, Wakefield's conceptualization of mental disorder has attracted critique (2,3) of some of its basic assumptions and supporting evidence. While acknowledging that the HD concept can have an energizing impact on the much needed debate about the theoretical foundations of psychiatry, I wish to join the camp of critics and to argue that: a) the HD definition and the conceptual analysis on which it rests contains logical inconsistencies, cannot be generalized to the entire domain of psychiatric nosology, and postulates an untenable *a priori* boundary between disorder and non-disorder; b) the assumption of the HD concept that dysfunction is anchored in a "failure of the mind to work as designed" by the evolution of the species does not accord well with current knowledge in evolutionary genetics and neuroscience; and c) the HD concept is of limited practical utility, especially as regards day-to-day clinical decision making.

Conceptual analysis is basically about how we use language, i.e. explicating what we mean by "mental disorder". In the search for an overarching definition, Wakefield assumes that in every society there are widely shared intuitions about mental disorder which provide a base for consensual judgements on the subject that could be somehow reconciled with scientific evidence of dysfunction. Most cultures certainly have prototypes, beliefs and practices related to mental disorder but, apart from converging on its stigmatizing aspects, such folk taxonomies in diverse societies are unlikely to provide "an underlying shared notion of disorder" that could be part of a rational and universal definition of mental disorder. Even more importantly, folk prototypes typically deal in dichotomies and opposites, e.g, disease versus health and disorder versus non-disorder – a model that can hardly be squared with the biomedical science component of the bipartite HD definition. Both general medicine and psychiatry are increasingly concerned with multiple biological continua and dimensions rather than with either-or categories. Although some extreme values along such continua and dimensions can be represented as categories, there is a huge grey zone of graded transitions between the biological phenomena which simply cannot be fitted into a single dichotomy. Thus, the concept of unitary "mental disorder" in general is a construct which is unlikely to find a "natural kind" counterpart in objective reality.

As regards Wakefield's elucidation of "dysfunction" as the factual component of "disorder", I am puzzled as to why the long shot to evolutionary theory and natural selection is considered necessary or even central to an understanding of psychopathology. Evolutionary psychology and psychopathology are still sciences under construction that can hardly provide a factual basis for teasing out the neural mechanisms and cognitive processes underlying the symptoms and signs of specific mental disorders. The definition of dysfunction as a failure of an organ or mechanism to perform the "natural function" for which it had been "designed" by natural selection implies the existence of purpose-driven evolutionary processes resulting in pre-ordained, fixed structures and functions, presumably located within the human brain. This view ignores the fact that natural selection is an opportunistic process, not guided by purpose or design, and that its general outcome is an increasing inter-individual variability. If anything, this variability will result in wider ranges for the parameters defining specific brain functions and dysfunctions; in different thresholds at which individuals develop mental and behavioural disorders; and in inherently fuzzy boundaries between disorder and non-disorder (2). Lastly, the assumption that neural systems within the human brain perform fixed cognitive or emotional functions pre-ordained by natural selection ignores two widely accepted pieces of evidence from evolutionary biology and neuroscience: first, that some highly specialized human cognitive functions (e.g., reading or writing) evolve by piggy-backing on earlier, more primitive adaptive mechanisms, and are therefore neutral vis-à-vis reproductive fitness; and secondly, that the individual brain is a neural plasticity machine, in the sense that it constructs its own internal cognitive architecture in post-natal development, in an activity-dependent manner, interacting with its environment. Thus, the thresholds of vulnerability to dysfunction of any causes vary individually to an extent that would

make the discernment of a breakdown in a "natural function" implausible.

My last point is: does psychiatry really need an overarching and universal definition of "mental disorder"? Neither disease nor health has ever been strictly and unambiguously defined in terms of finite sets of observable referential phenomena. Medical textbooks rarely devote even passing reference to the subject, and it seems perfectly possible for a medical professional to practice medicine and treat illnesses without using an overarching concept of disease (4). To quote Jaspers (5), "the medical person is least concerned with what healthy and sick mean in general… we do not need the concept of 'illness in general' at all and we now know that no such general and uniform concept exists". Furthermore, "doctors do not concern themselves with maximizing the evolutionary advantages of the human race as a whole, but with aiding individuals" (6).

The matter is further complicated by the emergence of molecular genetic classifications of large groups of diseases, and the concomitant availability of genetic diagnostic tests, which raise the possibility that the entire taxonomy of human disease may eventually be revised. Predictive diagnostic testing in clinically asymptomatic individuals will probably become possible in Alzheimer's disease, certain cancers and, hypothetically, for some of the major psychiatric disorders in the long run. Besides the ethical questions and the psychosocial repercussions of predictive testing, a problem to be faced is that for large segments of society (including health professionals) the concept of disease may become synonymous with the carrier state for a particular set of genes, without any reference to actual HD, blurring even further the demarcation between disease and non-disease. Attempts at defining an all-embracing, abstract definition of "mental disorder" have limited clinical utility (7) and will do poorly in this context.

Generally, the trend of the past decades has been one towards a multidimensional or polythetic conceptualization of the phenomena of disease, with several, relatively independent dimensions: a) clinical syndrome(s); b) structural and/or functional deviations from the statistical average; c) aetiology and pathogenetic mechanisms; and d) personal distress, quality of life and social functioning. At present, the majority of putative nosological entities in psychiatry are at best conceived as *open concepts*, as proposed by Meehl (8), i.e., subject to ongoing modification as new knowledge accrues. Closure will only be attained when fundamental issues of aetiology and pathogenesis are ultimately resolved – which is a long-term agenda. For the time being, the rather "weak" ICD-10 descriptive statement that presence of a mental disorder presupposes "a *clinically recognizable set of symptoms* or behaviours associated *in most cases* with distress and with interference with personal function" will probably do better than attempts at a hard-and-fast definition.

In conclusion, adoption of a generic, presumably universal, definition of "mental disorder" would be premature. It may cause more harm than good to psychiatry.

References

1. Wakefield JC. The concept of mental disorder. Am Psychol 1992;47:373-88.
2. Lilienfeld SO, Marino L. Mental disorder as a Roschian concept: a critique of Wakefield's "harmful dysfunction" analysis. J Abnorm Psychol 1995;104:411-20.
3. Murphy D. Psychiatry in the scientific image. Cambridge: MIT Press, 2006.
4. Jablensky A. Disease and health in the cultural context. In: Gunn SWA, Mansourian PB, Davies AM et al (eds). Understanding the global dimensions of health. New York: Springer, 2005:231-9.
5. Jaspers K. General psychopathology. Birmingham: Birmingham University Press, 1963.
6. Toon PD. Defining "disease" – classification must be distinguished from evaluation. J Med Ethics 1981;7:197-201.
7. Kendell R, Jablensky A. Distinguishing between the validity and utility of psychiatric diagnoses. Am J Psychiatry 2003;160:4-12.
8. Meehl PE. Diagnostic taxa as open concepts: meta-theoretical and statistical questions about reliability and construct validity in the grand strategy of nosological revision. In Millon T, Klerman GL (eds). Contemporary directions in psychopathology: towards the DSM-IV. New York: Guilford, 1986:215-31.

Potential implications of the harmful dysfunction analysis for the development of DSM-V and ICD-11

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Now that the development of both DSM-V and ICD-11 is underway, we should consider the potential practical implications of Wakefield's harmful dysfunction analysis for the revisions of these classifications. A research agenda for DSM-V (1) was published in 2002 with the goal of stimulating "research and discussion in the field in preparation for the eventual start of the DSM-V revision process" (2) and included a chapter on "Basic nomenclature issues for DSM-V". Among its recommendations were suggestions that DSM-V include a "definition of mental disorder that can be used as a criterion for assessing potential candidates for inclusion in the classification", noting that the definition of mental disorder included in DSM-IV is not "cast in a way that allows it to be used as a criterion for deciding what is and is not a mental disorder", largely because "the definition

fails to define or explain the crucial term dysfunction" (3). One of the strengths of Wakefield's harmful dysfunction analysis is that it helps to elucidate the key concept of "dysfunction", which Wakefield refers to as the "factual" component of the definition of mental disorder. Wakefield defines dysfunction as the failure of some brain or psychological mechanism to perform its naturally designed function. Although our current superficial understanding of mental processes limits our ability to precisely discern the various naturally designed functions of the brain, this approach is conceptually very appealing, because, as Wakefield points out in his many examples, it conforms to our common sense notions of what is and what is not a mental disorder.

If past experience is any guide, the upcoming revisions of the DSM and ICD classifications will bring with them many proposals for adding new disorders (4). While some proposals might entail carving out a new disorder from an existing category (for example, the proposal to add bipolar II disorder to DSM-IV involved reclassifying cases that would have been diagnosed as major depressive disorder in DSM-III-R) and thus primarily involve the boundary with other mental disorders, many proposals involve new diagnostic entities that impact the boundary with normality. It is this latter group for which Wakefield's harmful dysfunction analysis will be most relevant for insuring that the diagnostic entities are defined in such a way as to meet the criteria for a mental disorder. The harmful dysfunction analysis stresses that any definition of mental disorder should include elements that indicate both the presence of a dysfunction (i.e., the failure of a naturally designed mechanism) and a significant negative impact related to that dysfunction in terms of distress or impairment.

Previous efforts to construct criteria sets have primarily focused on the "harm" component by either including lists of symptoms that are, especially in aggregate, inherently harmful in terms of causing the individual distress or impairment (e.g., recurrent panic attacks, phobic avoidance) or else including a clinical significance criterion that explicitly requires impairment or distress (e.g., "the symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning"). Much less commonly are criteria included that address the "dysfunction" component of the definition, i.e., criteria that clarify that the harmful symptoms are the result of a failure in the individual of some designed function as opposed to resulting from non-disorder-related causes, like inadequate educational or financial opportunities, relational conflicts, etc. Most often, criteria are added that exclude specific situations in which the harmful symptoms are clearly caused by something that does not represent a failure of a designed function. For example, the diagnostic criteria for selective mutism specifically exclude situations in which the failure to speak is due to a lack of knowledge of, or comfort with, the spoken language required in the social situation. Rarely, the failed mechanism is explicitly included in the definition of a disorder; for example, stuttering is defined as a "disturbance in the normal fluency and time patterning of speech [that is] inappropriate for the individual's age" (5).

To illustrate how the harmful dysfunction analysis might apply in the consideration of proposals to include new disorders in DSM-V or ICD-11, take for example compulsive sexual behavior disorder (6), which is likely to be proposed for inclusion in DSM-V and ICD-11. Given that there are certainly at least some cases of individuals whose lives have been ruined by an inability to control their sexual impulses, the issue is not whether compulsive sexual behaviour can ever be considered a disorder, but instead how to tailor the criteria set for compulsive sexual behaviour disorder so that it falls within the definition of mental disorder. Using the harmful dysfunction analysis as a guide, the definition would have to include clear parameters indicating the harm caused by the symptoms as well as an explicit indication of the nature of the dysfunction, in this case, an internal failure to keep sexual impulses under control. To further clarify the internal nature of the dysfunction, additional criteria might be added to exclude other non-disordered causes for high levels of sexual activity (e.g., naturally high libido, situations in which outlets for sexual impulses are otherwise severely restricted).

It should be noted that, as Wakefield has pointed out elsewhere (7,8), the analysis outlined above has never been methodically applied to the current DSM-IV criteria sets, leading to many potential false positives that stem from not excluding cases in which symptoms arise from a non-disordered cause. Thus, the aforementioned harmful dysfunction analysis should not just be applied in the construction of criteria sets for new disorders, but should be used to guide revisions of the existing criteria sets as well.

References

1. Kupfer D, First M, Regier D (eds). A research agenda for DSM-V. Washington: American Psychiatric Publishing, 2002.
2. Kupfer D, First M, Regier D. Introduction. In: Kupfer D, First M, Regier D (eds). A research agenda for DSM-V. Washington: American Psychiatric Publishing, 2002: xv-xxiii.
3. Rounsaville B, Alarcon RD, Andrews G et al. Basic nomenclature issues for DSM-V. In: Kupfer D, First M, Regier D (eds). A research agenda for DSM-V. Washington: American Psychiatric Publishing, 2002:1-30.
4. Pincus H, Frances A, Davis WW et al. DSM-IV and new diagnostic categories: holding the line on proliferation. Am J Psychiatry 1992;149:112-7.
5. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed., text revision. Washington: American Psychiatric Association, 2000.
6. Mick T, Hollander E. Impulsive-compulsive sexual behavior. CNS Spectrum 2006; 11:944-55.
7. Wakefield J. Diagnosing DSM-IV - Part I: DSM-IV and the concept of disorder. Behav Res Ther 1997;35:633-49.
8. Wakefield J, First M. Clarifying the distinction between disorder and non-disorder: confronting the overdiagnosis ("false positives") problem in DSM-V. In: Phillips K, First M, Pincus H (eds). Advancing DSM: dilemmas in psychiatric diagnosis. Washington: American Psychiatric Publishing, 2003:23-56.

Evolution is the scientific foundation for diagnosis: psychiatry should use it

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Psychiatry has struggled for centuries to get mental disorders recognized as diseases just like those in the rest of medicine. To pursue this goal, the DSM-IV and other new diagnostic systems define disorders based on the number, severity and duration of symptoms. The benefit is that two clinicians who examine the same patient will likely arrive at the same diagnosis. This seems scientific. At least we can measure something reliably!

However, as Wakefield points out, such diagnostic systems only appear scientific. They offer no basis for deciding what is a disorder, and what is not. Worse yet, while they are intended to make psychiatry more like the rest of medicine, they do the opposite. In the rest of medicine, doctors recognize disorders as conditions that arise from abnormal functioning of some useful system. They know the heart evolved to pump blood and that insufficient function results in congestive heart failure. Cardiac failure is the diagnosis whenever the heart is not performing its normal function, no matter what the cause.

The rest of medicine makes a sharp distinction between disorders and protective responses. This distinction is mostly missing in psychiatry. Renal failure, cancer and paralysis are disorders, but fever, cough and pain are not disorders, they are protective responses. Fever and cough regulation mechanisms can fail, but doctors hardly ever diagnose "fever disorder" or "cough disorder". Instead, they look for the problem that aroused these functional responses.

As Wakefield shows so clearly, psychiatric diagnosis ignores this fundamental distinction. Major depression is diagnosed whenever severe enough symptoms persist long enough, no matter what is happening in the person's life. The exception, the recent death of a loved one, shows why considering context is essential. Good psychiatrists examine the patient's life situation in detail to try to understand whether the depression symptoms arise from a normal response to the current life situation, an abnormality of the mood regulation system, or, as is usually the case, some of both.

This essential distinction between reactive and endogenous depression was at the heart of DSM-II, but was eliminated in the DSM-III and IV. Ever since, psychiatric diagnosis has appeared objective, while in fact separating itself dramatically from diagnosis in the rest of medicine which relies on recognizing dysfunction. Why did psychiatric diagnosis exclude consideration of context? There are two obvious reasons.

First, when diagnosis depends on assessing the severity of life problems, reliability decreases. Whether or not loss of a job is sufficient to explain depression symptoms depends on how good or bad the job was, whether it can be easily replaced, and the person's financial situation. All of these factors involve somewhat subjective judgments. Making these judgments means that two diagnosticians will be less likely to come to the same conclusion.

This can be difficult, but the rest of medicine does not ignore context. For instance, when evaluating pain, physicians judge if this patient's pain is within the normal range given the nature of the organic lesion, or if the pain regulation system is not working properly. The decision is often difficult, but doctors do not duck the problem by using only the severity and duration of symptoms to determine if the patient has "pain disorder". Instead, they use all their knowledge and experience to try to decide if this patient's pain is a normal response, or if the system that regulates pain is abnormal.

The second reason psychiatric diagnosis ignores context is because the architects of the DSM-III were so desperate to separate psychiatry from psychoanalysis that they decided to ignore all theory. As a result, we still lack the kind of functional understanding that physiology offers to the rest of medicine. However, a functional understanding is now available to psychiatry.

For instance, determining when an emotion is abnormal requires understanding what normal emotions are for (1). The same evolutionary thinking that has rapidly advanced the study of animal behavior is being applied to human emotions. Emotions evolved because they adjust the body to deal with situations that have occurred again and again over millions of years. No emotion is good or bad in general, and negative emotions such as anxiety and sadness are just as useful as positive emotions. Emotions are useful if they are expressed in the situation they evolved for, otherwise they are abnormal (2). We must learn to recognize those situations. More generally, individuals who lack emotions don't do well in life. On average, across evolutionary history, they had fewer children. People who have excessive emotions, or whose emotions are expressed in the wrong situation, also do not do well. A panic attack is life-saving when you are being chased by a lion but, in a romantic situation, panic can severely decrease reproductive success!

The judgment of dysfunction is based on understanding a trait's evolutionary function. This is exactly the same for psychiatry as it is in the rest of medicine. Wakefield argues persuasively that this provides a solid biological basis for deciding whether a condition is normal or abnormal (3). This seems radical, but it is, instead, a call to return psychiatric diagnosis to its proper grounding in biology (4). Adopting his perspective would bring psychiatric diagnosis back into the biological framework that functional understanding provides for the rest of medicine.

References

1. Nesse RM. Evolutionary explanations of emotions. Human Nature 1990;1:261-89.
2. Nesse RM. Proximate and evolutionary studies of anxiety, stress, and depression: synergy at the interface. Neurosci Biobehav Rev 1999;23:895-903.
3. Wakefield JC, Horwitz AV. The loss of sadness: how psychiatry transformed normal sorrow into depressive disorder. New York: Oxford University Press, 2007.
4. Nesse RM, Jackson ED. Evolution: psychiatric nosology's missing biological foundation. Clin Neuropsychiatry 2006;3:121-31.

Fanatical about "harmful dysfunction"

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Wakefield is fanatical about "harmful dysfunction". We should be thankful for that. For his robust rhetoric has spectacularly succeeded, where decades of rigorous argument have spectacularly failed, in getting values on the agenda of psychiatric classification.

Yes, *values*. As a good rhetoretician, Wakefield focuses his audience's attention where their interests lie, on the empirical elements in the meaning of "disorder": this is why so much of his extensive output is concerned with defending a definition of "dysfunction" derived from evolutionary biology. "Evolution" and "biology" both have a reassuringly empirical ring. And so far as the DSM at least is concerned, this is precisely where the interests of Wakefield's audience lie: DSM-IV is explicitly evidence-based (1); and the American Psychiatric Association's agenda for DSM-V is predominantly an empirical research agenda (2). But even as Wakefield offers his audience what they want, an empirical definition of dysfunction, he is getting them to accept what they might otherwise resist, that "disorder", the concept with which DSM is concerned, has also a *non*-empirical, and specifically an *evaluative*, element in its meaning as well.

There is a kind of conceptual conjuring trick at work here. Wakefield presents "harmful dysfunction" fact-side up, but it is the value-side that the trick is all about. The trick is well turned, rhetorically speaking. Superficially, the trick is about "dysfunction" (fact-side) and harm (value-side). Thus far Wakefield's audience may feel reassured that even if disorder is, as Wakefield calls it, a hybrid (fact + value) concept, it is the (supposedly value-free) concept of dysfunction that psychiatric classification is (really) all about. But the trick runs deeper than this. For by liberally employing terms like "failure", Wakefield shows that his definition of dysfunction also has an underlying value side as well as the fact side he presents us with (3). As with "disorder" then, so with "dysfunction", Wakefield is able to present his definition of "dysfunction" fact-side up, while all the time it is the hidden value-side that is doing the (logical) work.

Like all conjuring tricks, once it is recognized for what it is, it is easy enough to see how it is done. Wakefield's citations alone illustrate what have been called the "3Rs" of rhetoric, Repetition, Repetition, Repetition – nearly half of Wakefield's citations are *self*-citations. His citations also show a good deal of the fourth 'R', rhetorical revisionism.

In a less rhetorical piece, the British psychiatrist and epidemiologist, the late Robert Kendell, instead of being assigned the relatively trivial role of pointing out the importance of resolving the value status of psychiatric diagnostic concepts, might have been credited as the first, over twenty years before Wakefield, to apply evolutionary biology to the problems of psychiatric classification (4). In a less rhetorical piece, similarly, the American philosopher, Christopher Boorse, instead of being accused of failing "to address (the) value component", might have been credited as the first, over twenty years before Wakefield, to propose a hybrid, fact + value, analysis of the medical concepts, and in two of the very articles cited by Wakefield (5,6). In a less rhetorical piece, finally, the block of no less that 16 citations described by Wakefield as being about "natural functions", i.e., about functions defined value-free, might more accurately have been described as contributions to a still unresolved debate about whether or not functions, let alone *dys*functions, can be naturalized at all, and let alone in the way proposed by Wakefield (7).

It might be thought that the presence of these rhetorical devices in Wakefield's work undermines his position. But that would be to stand outside the paradigm. As *rhetorical* devices, they are appropriate, well deployed, and effective.

Problems internal to the paradigm, on the other hand, do become apparent when, in the last part of his paper, Wakefield seeks to apply his analysis to some of the problems of the DSM. Thus, the rhetorical need for a single oft-repeated message leaves Wakefield at risk of appearing insensitive to the limitations of his own approach. The examples he gives are real enough: there really are these problems with the DSM; and they really are in part due to the difficulties of defining disorder. But beyond an earlier promissory note on a future neuroscience, Wakefield fails to show what, if any, specific contribution his "harmful dysfunction" definition of disorder makes to resolving the problems in question. Wakefield's examples are thus exemplary in form but have no exemplary content.

A second and more serious problem internal to the paradigm arises from Wakefield's rhetorical need to focus his audience's attention on the empirical element in the meaning of "disorder". For this leaves him at risk of appearing to neglect the resources of the many *non*-empirical disciplines available for tackling (alongside and in partnership with empirical disciplines) the problems of psychiatric classification. Such resources

include, for example, work in the philosophy of physics on the local nature of scientific validity (8); work in the philosophy of mind on the irreducible role of individual judgement (as in "clinical judgement") (9); and, specifically on values, work in such areas as linguistic analysis (10), phenomenology (11) and analytic philosophy (12), relevant to improving the *processes* of psychiatric diagnostic classification, i.e., to improving how our classifications are first developed and then actually used in day-to-day practice.

Still, these resources will be of little effect unless values and other non-empirical elements in the meaning of disorder are at least on the agenda of psychiatric classification. That is why, if getting them on the agenda has taken a conceptual conjuring trick, we should be thankful that Wakefield is fanatical about "harmful dysfunction".

References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington: American Psychiatric Association, 1994.
2. Kupfer DJ, First MB, Regier DE (eds). A research agenda for DSM-V. Washington: American Psychiatric Association, 2002.
3. Fulford KWM. Nine variations and a coda on the theme of an evolutionary definition of dysfunction. J Abnorm Psychol 1999;108: 412-20.
4. Kendell RE. The concept of disease and its implications for psychiatry. Br J Psychiatry 1975;127:305-15.
5. Boorse C. On the distinction between disease and illness. Philosophy and Public Affairs 1975;5:49-68.
6. Boorse C. What a theory of mental health should be. J Theory Soc Behav 1976;6:61-84.
7. Allen C, Lauder G (eds). Nature's purposes: analyses of function and design in biology. Cambridge: MIT Press, 1998.
8. Fine A. The natural ontological attitude. In: Boyd R, Gasker P, Trout JD (eds). The philosophy of science. Cambridge: MIT Press, 1999:261-77.
9. Thornton T. Tacit knowledge as the unifying factor in EBM and clinical judgement. Philosophy Ethics and Humanities of Medicine 2006;1:2.
10. Sadler JZ. Values and psychiatric diagnosis. Oxford: Oxford University Press, 2004.
11. Stanghellini G. Deanimated bodies and disembodied spirits. Essays on the psychopathology of common sense. Oxford: Oxford University Press, 2004.
12. Fulford KWM, Broome M, Stanghellini G et al. Looking with both eyes open: fact *and* value in psychiatric diagnosis? World Psychiatry 2005;4:78-86.

A new way of reducing the prevalence of mental disorders?

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In his lucidly written paper, Jerome Wakefield argues that a condition can be regarded as a mental disorder if a) it is considered harmful and b) it is due to a dysfunction resulting from the failure of some internal mechanism (originally destined to perform the now deranged function). This definition should hold for "physical" and for "mental" disorders. Wakefield does not distinguish disease from disorder, although the two terms are not describing the same type of conditions.

Another distinction that is important in this discussion is that between a *disorder* (and a medical disease), the expressed *needs for care* and a *sickness* (a state defined by a society as requiring treatment or deserving sickness benefits) (1).

A significant proportion of people who have a disorder do not request nor receive treatment or care; a number of people who request and receive care do not have a medically recognized disorder; and finally most societies at some point of their history designate a particular pattern of behaviour as being sick (and therefore requiring treatment or incarceration or both) although the persons concerned do not request treatment and do not suffer from any discernible disorder.

Requiring, as Wakefield suggests, that both a negative value and a dysfunction must be present to define a condition as a disorder requiring attention of the health system may lead to a number of problems. Thus, for example, people with a dysfunction that is at present not leading to a disadvantage would be excluded from treatment or care: to take Wakefield's example, people with an abnormality of corpus callosum (for example due to some infectious and curable condition) leading to dyslexia would not be offered treatment in illiterate societies, because their dysfunction does not lead to immediate disadvantages. Poor people in rich and in poor countries have often no access to many things that are available to those who are rich: would that mean that the poor should not be given health care for their dysfunctions because they will not be in situations where these might be disturbing?

I share Wakefield's faith into our capacity to assess disturbances of "mental" functions with just as much precision as that of "physical functions". On the other hand, the differences between cultures make the "negative value" assessment of a particular "factual dysfunction" so different from one setting to another that it is difficult to imagine how any comparisons of "disorders" could be done if we define them as Wakefield proposes. I therefore believe that epidemiological (and other) studies that need to work with homogenous groups should define disorders in terms of "factual dysfunction" in Wakefield's terms and then use the results of these assessments in a manner congruent with the goal of the studies – for example, to assess the prevalence of a disorder or to use them as one of the bases for the assessment of needs for care.

In summary, I think that Wakefield's analysis of the concept of mental disorder is useful, because it makes us think about the nature of diseases and their meaning, but I disagree with his conclusion that the "negative value" of a

particular dysfunction should be decisive in defining the disorder. Like in the rest of medicine, the diagnosis of a disorder should be based on well-defined symptoms indicating a dysfunction and steer clear from mixing this assessment with the assessments of social desirability or of disability.

References

1. Sartorius N. Fighting for mental health. Cambridge: Cambridge University Press, 2002.

Wakefield's hybrid account of mental disorder

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Wakefield's question is what makes a mental condition a disorder. He formulates the question in two different ways: a) "*What do we mean* when we say that a mental condition is a medical disorder rather than, for instance, a normal form of human suffering?" and b) "Which mental conditions *should* be classified as pathological?" The latter question is far more significant, especially if we concede that no consensus exists on the meaning of "mental disorder". "Disorder" is regarded as a broad term "that covers both traumatic injuries and diseases/illnesses". This notion is more practically significant than, for instance, the notion of disease. The distinction between disease and injury has no practically important consequences, whereas the distinction between disorder and non-disorder can affect who is entitled to publicly funded health care, medical insurance reimbursement, or sick leave with compensation (1,2).

In Wakefield's view, mental disorders are harmful mental dysfunctions. This is presented as a hybrid account, i.e., as incorporating both a value component (harm) and a factual component (dysfunction). It is not clear whether Wakefield's account contains any value component, however, i.e., whether it is a proper hybrid account. Wakefield repeatedly uses phrases like "judged negative by sociocultural standards" or "harmful according to social values" to characterize the value component, but to say that a condition is deemed negative by "sociocultural standards" is really a factual statement. Moreover, to refer to existing sociocultural standards is only relevant if we want to *explain* why certain conditions are classified as disorders in a certain society, but not if we want to determine what conditions *should* be classified as pathological. The latter question is the important one and, to answer this question, we need to determine whether a condition is harmful, not whether it is regarded as such from any particular perspective. But let us assume that Wakefield's analysis is, in fact, a proper hybrid account. In this case, his account of the value component is probably too narrow, and the same holds for his account of the factual component.

Mental disorders typically involve some kind of harm to the individual who has the disorder, e.g. distress or disability, and we rely rather heavily on considerations of harm when drawing the line between disorder and non-disorder. This strongly suggests that the connection between disorder and harm is conceptual rather than contingent. Wakefield makes a stronger point than this, however, namely that harm to the individual is *necessary* for disorder, and that we need not rely on any other evaluative considerations to delineate the class of mental disorder. However, it seems that there are mental disorders that are classified as disorders in virtue of other evaluative considerations, e.g., that paedophilia and antisocial personality disorder count as disorders because they are abnormal and/or harmful to others. This suggests that we should not draw the line between disorder and non-disorder on the basis of harm-for-the-individual-evaluations alone, but that we must also make use of harmful-for-others-judgments and judgments of abnormality, including attributions of irrationality (3). This view gives us a less coherent concept of mental disorder, however, and it is incompatible with the idea that "mental disorder" can be defined in terms of necessary conditions that are jointly sufficient (3).

Wakefield's evolutionary account of disorder has been heavily criticized (1,4-7). Most objections purport to show that dysfunction (in Wakefield's sense) is not necessary for disorder, i.e. that someone may well suffer from a disorder even when there is no "evolutionary malfunction". Some of these objections try to establish that "many mental functions are not direct evolutionary adaptations, but rather adaptively neutral by-products of adaptations" (4), and that some disorders involve failed mechanisms that have no adaptive function, like spandrels, exaptations, or vestigal parts.

Other arguments purport to establish that disorders can be caused by mechanisms that are working exactly as designed by evolution, e.g., that some disorders are evolutionary adaptive reactions to "pathogenic inputs". Injuries due to external trauma involve dysfunctions, however, and so do inflammatory reactions, infectious diseases, and post-traumatic stress disorder. But consider "normal grief" vs. pathological bereavement (a possible component in depressive disorder) as two possible reactions to loss. Is the difference between these conditions really that some specific mechanism is malfunctioning in the second case but not in the first? To defend the dysfunction account by postulating a "loss-response mechanism" is rather farfetched. It seems more plausible to regard the two conditions as *different ways of functioning*, where "the depressed way of grieving" is far more harmful than the "normal" way. This suggests that the presence of a dysfunction is not essential to disorder. Moreover, the exclusion of normal grief from the class of mental disorder can be

questioned: for instance, it might be appropriate to regard grief as a mental injury and, if all injuries are disorders, so is grief. It can also be argued that people in grief are entitled to sick leave with compensation. Normality is simply not the issue here.

To conclude, Wakefield's idea that disorders are dysfunctions (defined in evolutionary terms) tends to exclude too much from the category of mental disorder. There are alternative views, but these views also suffer from certain weaknesses (1). This strongly suggests that we cannot save our linguistic intuitions unless we abandon the idea that "mental disorder" can be defined in terms of necessary conditions that are jointly sufficient (1).

References

1. Brülde B. The concept of mental disorder. Gothenburg: Department of Philosophy, 2003.
2. Brülde B. Art and science, facts and knowledge. Philosophy, Psychiatry, and Psychology (in press).
3. Brülde B. Mental disorder and values. Philosophy, Psychiatry, and Psychology (in press).
4. Lilienfeld SO, Marino L. Mental disorder as a Roschian concept: a critique of Wakefield's "harmful dysfunction" analysis. J Abnorm Psychol 1995;104:411-20.
5. Murphy D, Woolfolk RL. The harmful dysfunction analysis of mental disorder. Philosophy, Psychiatry, and Psychology 2000; 7:241-52.
6. Murphy D, Woolfolk RL. Conceptual analysis versus scientific understanding: an assessment of Wakefield's folk psychiatry. Philosophy, Psychiatry, and Psychology 2000; 7:271-92.
7. Nordenfelt L. On the evolutionary concept of health: health as natural function. In: Nordenfelt L, Liss P-E (eds). Dimensions of health and health promotion. Amsterdam: Rodopi Press, 2003:37-54.

The usefulness of Wakefield's definition for the diagnostic manuals

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No one has done more in the last decade or so to clarify and analyse the concept of mental disorder than Jerome Wakefield, and it is timely to consider his work during preparations for new editions of the DSM and the ICD. These will involve review of the reliability of diagnoses, and the various issues of validity of classification of symptoms into syndromes, and syndromes into higher-order categories.

The further and distinctive kind of validity to which Wakefield has consistently drawn attention since his first papers in the early 1990s is what he has called the problem of *conceptual validity*: to what extent do the manuals capture all and only the mental disorders (or mental and behavioural disorders), and to what extent have they left some out, or, most discussed, to what extent have they mistakenly included some non-disorders in. This is the "overinclusiveness" or "false positive" problem. The diagnostic criteria for some disorders are *too lax*, in the sense that particular presentations may satisfy them, but are – apparently – not cases of disorder. Wakefield has argued along these lines for many conditions, including major depressive disorder, conduct disorder and social phobia.

Wakefield has consistently linked the problem of conceptual validity of diagnosing disorder – are we really diagnosing *disorder*? – to the fundamental problem of *reliability* of diagnosis. Following Hempel's advice, the diagnostic manuals have sought to make symptom description as purely observational as possible, without speculations as to aetiology, and then (especially in the DSM) to have syndrome composition as arithmetically algorithmic as possible (symptom counts of more or less complicated kinds). Wakefield's argument has been that this methodology in effect detaches troublesome mental states and behaviours from their context, failing to take account of whether they are "normal" responses to adversities, or arise in understandable ways according to normal learning – as opposed to genuine disorders involving dysfunction.

So can Wakefield's analysis help sort out what are the "genuine disorders"? In brief form the analysis is: mental disorder = harmful dysfunction. This brief form is trivial – inasmuch as it substitutes "dysfunction" for "disorder" – and should not be mistaken for the non-trivial full version, which is (along the lines of): mental disorder = harmful failure of a natural mental or behavioural mechanism to function as designed in evolution. Can this help solve the problem of conceptual validity for the psychiatric manuals? Can it be used to make particular diagnostic criteria sets more valid, by excluding non-disordered conditions?

It may be that Wakefield's analysis of "mental disorder" is conceptually correct. I have argued elsewhere that it is not (1), but the issues are too long for here. It is fair to say in any case that no one has come up with such a rigorous definition that is better. So should it be put in the preambles to the DSM-V and ICD-11?

The problem here would be the fairly obvious one – signalled by Wakefield's own arguments – namely, that reliability would be seriously jeopardised. To establish that a condition is a *disorder* in the sense of Wakefield's analysis, we would have to establish, or at least have a consensus about, whether it arose because of or at least involved "failure of a natural mental or behavioural mechanism to function as designed in evolution". But as opposed to what? Behavioural scientists working in an evolutionary theoretic framework have suggested that failure of function in Wakefield's sense as a pathway to harmful conditions can be contrasted with, for instance, evolutionary design/current environment mismatch, or maladaptive learning (2,3). If these are the kinds of intended contrasts, we need to wait until the science

has been done to establish which types or sub-types of problems are "genuine disorders" in the sense of Wakefield's analysis, and which are not. And in the meantime, during what might be a long wait, we would need *another name* for the problems, not *disorder* (which in this scenario we are interpreting in Wakefield's sense), but perhaps, for instance, *mental health problems*, the criteria for which would have to be reliable enough for us to do meaningful, generalizable research. We would be back where we are with (another) change of name.

If we were to follow this tack we may eventually sort out what conditions or sub-types are "disorders" (in the sense required by Wakefield's evolutionary theoretic analysis) and which are not. The ones that are not – Wakefield concedes – may still be associated with harm and with risk of harm. They would therefore still be in need of treatment (in a general sense including watchful waiting). Indeed the harm or risk associated with the non-disorder variants may be as high as for the "genuine disorders" – we won't know this until the science has been done (it cannot be known from the armchair). We would have a manual of "mental disorders and related mental health problems" (somewhat like the relaxed full title of the ICD) in which the difference between the two is less important than the associated harm and risk and consequent need for clinical attention and research. The evolutionary theoretic definition would not have done much practical work, because actually what drives practice is harm and risk.

References

1. Bolton D. What is mental disorder? An essay in philosophy, science and values. Oxford: Oxford University Press (in press).
2. Cosmides L, Tooby J. Toward an evolutionary taxonomy of treatable conditions. J Abnorm Psychol 1999;108:453-64.
3. Richters JE, Hinshaw SP. The abduction of disorder in psychiatry. J Abnorm Psychol 1999;108:438-45.

Cultural psychiatry on Wakefield's procrustean bed

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Jerome Wakefield has advanced an account of mental disorder that aims to provide a way to distinguish *bona fide* psychiatric disorders from "problems in living". He claims that it is possible to strip away the normative component of a disorder to leave a notion of dysfunction that is a "purely factual scientific concept". According to Wakefield's harmful dysfunction analysis, something is a mental disorder if, and only if, there is a deviation from natural function and that deviation is harmful. Natural function is construed as the function selected for by evolution. We offer four reasons for doubting that natural function can be determined by an application of evolutionary theory and, thus, for doubting the validity of the harmful dysfunction analysis.

First, the boundary between function and dysfunction is indeterminate. Although some traits or states show points of rarity or abrupt transitions, in many instances, psychological function and dysfunction manifest as a spectrum. Some people are more naturally anxious than others, for example. How far away from statistical normality does a function have to be to count as dysfunction? It is hard to see how to answer this question without appealing to a notion of deviation that is harmful or undesirable. And, indeed, notions of mental disorder in many cultures are closely tied to inappropriate or problematic social behavior, not to notions of internal (psychological or physiological) functioning (1,2). This way of understanding dysfunction, however, collapses the putatively factual component of the harmful dysfunction analysis into the normative one.

Second, natural function may not be actual function. The existence of many traits may be explained not by the increased fitness they confer but by evolutionary conservatism. A trait may be present in a species because it was present in the evolutionary ancestors of the species and was conserved because it was harmless or intrinsic to developmental pathways (3). Similarly, some physical and psychological human traits may best be explained by the fact that they conferred some adaptive advantage on an evolutionary ancestor of ours rather than on us. The natural function of a system, in Wakefield's sense, may have little to do with its current function.

Third, natural brain function may not be actual brain function. Neural plasticity makes a divergence between natural and actual function particularly likely in psychiatric disorder. In most human beings, for example, primary visual cortex functions to extract information about the external world from light. But in people who have lost their sight (and are naïve to Braille), primary visual cortex becomes responsive to tactile information (4). The function of primary visual cortex, it seems, has more to do with input than with selection pressures. Were a virus to render all human beings blind, the actual function of primary visual cortex would *ipso facto* be tactile, and disorders of primary visual cortex would have nothing to do with the function for which it was selected. While the example is extreme, it points up the fact that brain function depends significantly on environment.

Fourth, mental function and dysfunction are essentially dependent on culture. Could an early hominid have had attention deficit/hyperactivity disorder (ADHD)? That depends on the tasks for which attention is required. The ability to sit still for long hours in a classroom is a significant part of what we now count as normal attention. Since no

such demand was placed on the early hominid child, he could have had normal attention even if his attention functions were, biologically speaking, identical to a child with ADHD. Note that this is not a case of the hominid child having a harmless dysfunction. To say that would be to beg the question of what a dysfunction is. Rather, the concept of normal mental function varies in part with the demands placed by culture on the mind. It cannot be determined by evolutionary theory alone.

As Wakefield notes, culture exerts profound effects on symptom experience and expression in ways that may make symptom-based diagnostic criteria difficult to apply (5). But culture may go well beyond this to influence the mechanisms of psychiatric disorders. For example, in Cambodia, dizziness can indicate a "wind" attack, a potentially serious illness (6). The orthostatic dizziness that sometimes follows standing up can therefore lead to panic in a Cambodian. A Canadian whose mental functions were identical might never suffer from panic attacks because he fails to have the relevant beliefs. What makes the Cambodian, but not the Canadian, disordered depends upon culture. Again, it would be misleading to claim that the Canadian has the same dysfunction as the Cambodian but that it is only harmful in Cambodia. The specific dysfunction depends essentially on task demands that, in turn, depend on culture.

Psychiatry is a young discipline. Wakefield is asking it to lie on a procrustean bed and lose the limbs that do not sit well with an evolutionary conception of dysfunction. But there is no reason why psychiatry should be shrunk to fit and no reason to restrict it at a time when we understand so little about mental disorder. We should rather enlarge its domain of theory and practice to understand developmental problems in both an evolutionary and a social context. In response to Wakefield's worry that without something like harmful dysfunction analysis we will have no way to critique diagnostic constructs and criteria, we suggest that an alternative "place to stand" is on an integrated conception of mental function that is responsive to a range of considerations, from evolutionary theory and neurobiology, through cultural context, to systematic analyses of the social functions of the diagnostic construct itself.

References

1. Haslam N. Dimensions of folk psychiatry. Rev Gen Psychol 2005;9:35-47.
2. Glovsky V, Haslam N. Acculturation and changing concepts of mental disorder: Brazilians in the USA. Transcult Psychiatry 2003;40:50-61.
3. Chittka L, Briscoe A. Why sensory ecology needs to become more evolutionary - insect color vision as a case in point. In: Barth DG, Schmid A (eds). The ecology of sensing. Berlin: Springer, 2001:19-38.
4. Sadato N, Okada T, Kubota K et al. Tactile discrimination activates the visual cortex of the recently blind naive to Braille: a functional magnetic resonance imaging study in humans. Neurosci Lett 2004;359:49-52.
5. Kirmayer LJ. Culture, context and experience in psychiatric diagnosis. Psychopathology 2005;38:192-6.
6. Hinton DE, Pich V, Safren SA et al. Anxiety sensitivity among Cambodian refugees with panic disorder: a factor analytic investigation. J Anxiety Disord 2006;20:281-95.

The concept of mental disorder: an African perspective

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The concept of mental disorder is determined by many factors, including the historical context, cultural influence, level of scientific knowledge and capacity to carry out scientific enquiry, level of education in certain circumstances, as well as many others. In putting together a method of classification of mental disorders, the expert's duty is primarily that of capturing and remaining faithful to the current level of knowledge in the subject, acknowledging that, in a matter of time, some or all the above factors could change to variable degrees, making what was clear as a mental disorder a few decades previously less clear in the next edition of the classification system.

In the earlier editions of the DSM, homosexuality was clearly categorized as a mental disorder and, by extension, a condition demanding or at least requiring medical treatment. In Western cultures, any suggestion that being gay or lesbian is anything but normal would now attract the wrath of society. The situation in Africa is quite the opposite, and many Africans still view gay and lesbian people as "mentally sick", because their sexual orientation is against the order of nature. In this regard, one could view the Africans as "uncivilized" or as holding a cultural belief that may or may not change in the course of time, much as it did in Western countries.

A similar but opposite position holds with respect to the circumcision of women, a practice also described as female genital mutilation (FGM). There are still very strong pockets of Africans who practice FGM, presumably in part as a cure for what Victorian physicians would have called "clitoral orgasm", a condition then requiring preventive surgery. Many Africans defend the cultural position with equal vigor to those who find it abnormal. There are those who would consider it a mental aberration to mutilate the genitalia of young women and children.

Anorexia nervosa is one of the leading causes of morbidity and mortality in adolescent girls in Western countries. Crisp et al (1) found a prevalence of one severe case in 200 girls in independent schools, while, among girls aged 16 years and over, the rate rose to one in a hundred for severe cases. In Africa, the condition is hardly known. Njenga and Kang'ethe (2) reported on a study in Kenya and concluded that "in a cumulative period of 320 years of practice, Kenyan psychiatrists had seen twenty cases of anorexia nervosa". Hulley et al (3) studied a sample of Kenyan and British female athletes and concluded that "the effects of cul-

ture were clear, women in the UK were more dissatisfied with their weight and shape and demonstrated significantly more eating disorder cases and associated psychopathology compared with the Kenyan women".

So, who is deluding who? Is refusal to eat food by "spoilt white girls a disease or simple foolishness?". Trying to explain to the hungry African mother and child that there are girls who die in Western countries because they refuse to eat food goes beyond reason and logic and would not make sense as a mental disorder, and yet in the West, there is no room for such a discussion.

Anorexia nervosa in fact raises many questions regarding its cause and origins. Is it primarily genetic, or is it a social construct of a search of thinness as required of females in Western societies, or is it a combination of both? Should we conclude that pursuit of a cultural belief, such as the belief that to be thin is good, is evidence of a mental disorder because it causes mortality and morbidity? How much is the desire for a thin body "normal" and how much of the same is abnormal, and who decides anyway? Are these cultural or biological conditions? The issue of dimensional and categorical systems of classification comes sharply into focus. The African is however clear! When food is available, one must eat to the full!

Historically, the African were believed to function as "lobotomized Europeans" (4), because of a smaller brain, and the desire to free himself from French colonialist rule was evidence of a mental disorder, a "fact" taught in French Universities in the 1960s (5). Few if any psychiatrists would now believe "the facts" as stated above, but in the 1950s and 1960s, these were the facts as understood by well educated, well meaning men and women of science. It is therefore with this knowledge that we must approach the subject of mental disorders with caution and humility, as we could, in a generation or two, be viewed much as Carothers is now viewed by many.

That said, however, we must pick up the courage of our conviction and do what man has done through the years, which is to create order from chaos, which is, after all, the whole purpose and function of a classification system. Our duty to posterity, therefore, is to use the best available tools, to carry out the ordering process and, even if we get it "wrong" in the eyes of the next generation, we will be able to stand firm and tall in the knowledge that no system of classification will remain unchanged for all time. It therefore stands to reason that the concept of what is and what is not a mental disorder is a dynamic one, which will change from time to time, from culture to culture and, as in the case of homosexuality, from generation to generation.

References

1. Crisp AH, Palmer RL, Kalucy RS. How common is anorexia nervosa? A prevalence study. Br J Psychiatry 1976;128:549-54.
2. Njenga FG, Kang'ethe RK. Anorexia nervosa in Kenya. East Afr Med J 2004;81:188-93.
3. Hulley A, Currie A, Njenga F et al. Eating disorders in elite female distance runners: effects of nationality and running environment. Psychology of Sport and Exercise 2007; 8:521-33.
4. Carothers JC. Frontal lobe function and the African. J Ment Sci 1951;97:12-48.
5. Fanon F. The wretched of the earth. London: Penguin Books, 1963.

Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative

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Data are presented on the lifetime prevalence, projected lifetime risk, and age-of-onset distributions of mental disorders in the World Health Organization (WHO)'s World Mental Health (WMH) Surveys. Face-to-face community surveys were conducted in seventeen countries in Africa, Asia, the Americas, Europe, and the Middle East. The combined numbers of respondents were 85,052. Lifetime prevalence, projected lifetime risk, and age of onset of DSM-IV disorders were assessed with the WHO Composite International Diagnostic Interview (CIDI), a fully-structured lay administered diagnostic interview. Survival analysis was used to estimate lifetime risk. Median and inter-quartile range (IQR) of age of onset is very early for some anxiety disorders (7-14, IQR: 8-11) and impulse control disorders (7-15, IQR: 11-12). The age-of-onset distribution is later for mood disorders (29-43, IQR: 35-40), other anxiety disorders (24-50, IQR: 31-41), and substance use disorders (18-29, IQR: 21-26). Median and IQR lifetime prevalence estimates are: anxiety disorders 4.8-31.0% (IQR: 9.9-16.7%), mood disorders 3.3-21.4% (IQR: 9.8-15.8%), impulse control disorders 0.3-25.0% (IQR: 3.1-5.7%), substance use disorders 1.3-15.0% (IQR: 4.8-9.6%), and any disorder 12.0-47.4% (IQR: 18.1-36.1%). Projected lifetime risk is proportionally between 17% and 69% higher than estimated lifetime prevalence (IQR: 28-44%), with the highest ratios in countries exposed to sectarian violence (Israel, Nigeria, and South Africa), and a general tendency for projected risk to be highest in recent cohorts in all countries. These results document clearly that mental disorders are commonly occurring. As many mental disorders begin in childhood or adolescents, interventions aimed at early detection and treatment might help reduce the persistence or severity of primary disorders and prevent the subsequent onset of secondary disorders.

Key words: Mental disorders, lifetime prevalence, projected lifetime risk, age-of-onset distribution

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Although psychiatric epidemiological surveys have been carried out since after World War II (1), absence of a common format for diagnosis hampered cross-national syntheses. This situation changed in the early 1980s, with the development of fully structured research diagnostic interviews (2) and the implementation of large-scale psychiatric epidemiological surveys in many countries (3-5). The World Health Organization (WHO) developed a diagnostic instrument, the WHO Composite International Diagnostic Interview (CIDI) (6,7), based on extensive cross-national field trials, for use in cross-national epidemiological surveys (8-14). In 1998, the WHO created the WHO International Consortium in Psychiatric Epidemiology (ICPE) to coordinate comparative analyses of these surveys. The ICPE launched the WHO World Mental Health (WMH) Survey Initiative shortly thereafter to conduct coordinated CIDI surveys in all parts of the world. The current report presents the first cross-national results regarding age of onset, lifetime prevalence, and projected lifetime risk of mental disorders from the 17 WMH surveys so far completed.

Data of this sort are sorely needed by policy planners to assess the societal burden of mental disorders, unmet need for treatment, and barriers to treatment. These data are especially important given evidence from the WHO Global Burden of Disease Study that mental disorders impose enormous burdens worldwide, due to their combination of high prevalence and high disability (15), and evidence that, despite efficacious treatments, substantial unmet need for

treatment exists throughout the world (16). While earlier studies found high lifetime prevalence and generally early age-of-onset distributions of mental disorders, they did not make systematic disorder-specific age-of-onset comparisons. The latter are important for targeting early interventions, which are coming to be seen as critical for an effective public health response to mental disorders (17-19). Previous studies also focused on lifetime prevalence (the proportion of the population with a lifetime disorder up to age at interview) rather than projected lifetime risk (the estimated proportion of the population who will have the disorder by the end of their life), even though the latter is more important for policy planning purposes. We consider both prevalence and risk in this report.

METHODS

Samples

WMH surveys were administered in Africa (Nigeria, South Africa); the Americas (Colombia, Mexico, United States), Asia and the Pacific (Japan, New Zealand, Beijing and Shanghai in the People's Republic of China, henceforth referred to as Metropolitan PRC), Europe (Belgium, France, Germany, Italy, the Netherlands, Spain, Ukraine) (20); and the Middle East (Israel, Lebanon). Seven of these countries are classified by the World Bank as less developed (China, Colombia, Lebanon, Mexico, Nigeria, South Africa, Ukraine), while the others are classified as developed (21).

Most WMH surveys were based on stratified multistage clustered area probability household samples. Samples of areas equivalent to counties or municipalities in the US were selected in the first stage, followed by one or more subsequent stages of geographic sampling (e.g., towns within counties, blocks within towns, households within blocks) to arrive at a sample of households. In each of them, a listing of household members was created and one or two people were selected to be interviewed. No substitution was allowed when the originally sampled household resident could not be interviewed. The household samples were selected from census area data in all countries other than France (where telephone directories were used) and the Netherlands (where postal registries were used). Several WMH surveys (Belgium, Germany, Italy) used municipal resident registries to select respondents without listing households. The Japanese sample is the only totally unclustered sample, with households randomly selected in each of the four sample areas and one random respondent selected in each sample household. Nine of the 17 surveys were based on nationally representative household samples, while two others were based on nationally representative household samples in urbanized areas (Colombia, Mexico).

All surveys were conducted face-to-face by trained lay interviewers in multi-stage household probability samples, with 85,052 respondents. Country-level samples ranged from 2372 (Netherlands) to 12,992 (New Zealand). The weighted average cross-national response rate was 71.1%, with a 45.9-87.7% range (Table 1).

The Part I interview schedule, completed by all respondents, assessed core diagnoses. All respondents who met criteria for any diagnosis plus a probability sub-sample of other Part I respondents were administered Part II, which assessed disorders of secondary interest and a wide range of correlates. Part I data were weighted to adjust for differential probabilities of selection and to match population distributions on socio-demographic and geographic data. The Part II sample was additionally weighted for the oversampling of Part I respondents with core disorders. The interview schedule and other study materials were translated using standardized WHO translation and back-translation protocols. Consistent interviewer training procedures and quality control monitoring were used in all surveys (22,23). Informed consent was obtained in all countries using procedures approved by local Institutional Review Boards.

Measures

Diagnoses were based on CIDI Version 3.0 (24), which generates both ICD-10 (25) and DSM-IV (26) diagnoses. DSM-IV criteria are used here to facilitate comparison with previous epidemiological surveys. Core diagnoses included anxiety disorders (panic disorder, agoraphobia without panic disorder, specific phobia, social phobia, generalized anxiety disorder, post-traumatic stress disorder, and separation anxiety disorder), mood disorders (major depressive disorder, dysthymic disorder, bipolar disorder I or II or subthreshold bipolar disorder), impulse control disorders (intermittent explosive disorder, oppositional-defiant disorder, conduct disorder, attention-deficit/hyperactivity disorder), and substance use disorders (alcohol and drug abuse with or without dependence). Not all disorders were assessed in all countries. The Western European countries did not assess bipolar disorders and drug dependence. Only three countries (Colombia, Mexico, United States) assessed all impulse control disorders.

The disorders that require childhood onset (oppositional defiant disorder, conduct disorder, and attention-deficit/hyperactivity disorder) were included in Part II and limited to respondents in the age range 18-39/44, because of concerns about recall bias among older respondents. All other disorders were assessed for the full sample age range. Organic exclusion rules and hierarchy rules were used to make all diagnoses other than substance use disorders, which were diagnosed without hierarchy, because abuse often is a stage in the progression to dependence. Clinical calibration studies (27) found CIDI to assess these disorders with generally good validity in comparison to blinded clinical reappraisal interviews using the Structured Clinical Interview for DSM-IV (SCID) (28). CIDI prevalence es-

Table 1 Sample characteristics of the World Mental Health Surveys

Country	Survey	Field dates	Age range	Sample size			Response rate
				Part I	Part II	Part II and age ≤ 44[a]	
Belgium	ESEMeD	2001-2	18+	2419	1043	486	50.6
Colombia	NSMH	2003	18-65	4426	2381	1731	87.7
France	ESEMeD	2001-2	18+	2894	1436	727	45.9
Germany	ESEMeD	2002-3	18+	3555	1323	621	57.8
Israel	NHS	2002-4	21+	4859	-	-	72.6
Italy	ESEMeD	2001-2	18+	4712	1779	853	71.3
Japan	WMHJ 2002-2003	2002-3	20+	2436	887	282	56.4
Lebanon	LEBANON	2002-3	18+	2857	1031	595	70.0
Mexico	M-NCS	2001-2	18-65	5782	2362	1736	76.6
Netherlands	ESEMeD	2002-3	18+	2372	1094	516	56.4
New Zealand	NZMHS	2004-5	16+	12992	7435	4242	73.3
Nigeria	NSMHW	2002-3	18+	6752	2143	1203	79.3
People's Republic of China	B-WMH S-WMH	2002-3	18+	5201	1628	570	74.7
South Africa	SASH	2003-4	18+	4315	-	-	87.1
Spain	ESEMeD	2001-2	18+	5473	2121	960	78.6
Ukraine	CMDPSD	2002	18+	4725	1720	541	78.3
United States	NCS-R	2002-3	18+	9282	5692	3197	70.9

ESEMeD - European Study of the Epidemiology of Mental Disorders; NSMH - Colombian National Study of Mental Health; NHS - Israel National Health Survey; WMHJ 2002-2003 - World Mental Health Japan Survey; LEBANON - Lebanese Evaluation of the Burden of Ailments and Needs of the Nation; M-NCS - Mexico National Comorbidity Survey; NZMHS - New Zealand Mental Health Survey; NSMHW - Nigerian Survey of Mental Health and Wellbeing; B-WMH - Beijing World Mental Health Survey; S-WMH - Shanghai World Mental Health Survey; SASH - South Africa Health Survey; CMDPSD - Comorbid Mental Disorders during Periods of Social Disruption; NCS-R - U.S. National Comorbidity Survey Replication

The response rate is calculated as the ratio of the number of households in which an interview was completed to the number of households originally sampled, excluding from the denominator households known not to be eligible either because of being vacant at the time of initial contact or because the residents were unable to speak the designated languages of the survey

[a]All countries were age restricted to ≤44, with the exception of Nigeria, People's Republic of China, and Ukraine, which were age restricted to ≤39

timates were not higher than SCID prevalence estimates. Retrospective age-of-onset reports were based on a question series designed to avoid the implausible response patterns obtained in using the standard CIDI age-of-onset question (29). Experimental research has shown that this question sequence yields responses with a much more plausible age-of-onset distribution than the standard CIDI age-of-onset question (30). Predictor variables included cohort (defined by ages at interview 18-34, 35-49, 50-64, 65+), sex, and education (students versus non-students with low, low-average, average-high, and high education categories based on country-specific distributions). Education was coded as a time-varying predictor by assuming an orderly educational history.

Analysis procedures

Age of onset and projected lifetime risk as of age 75 were estimated using the two-part actuarial method implemented in SAS 8.2 (31). Predictors were examined using discrete-time survival analysis with person-year as the unit of analysis (32). Standard errors were estimated using the Taylor series linearization method (33) implemented in the SUDAAN software system (34). Multivariate significance tests were made with Wald χ^2 tests, using Taylor series design-based coefficient variance-covariance matrices. Standard errors of lifetime risk were estimated using the jackknife repeated replication method (35) implemented in a SAS macro (31). Significance tests were all evaluated at the .05 level with two-sided tests.

RESULTS

Lifetime prevalence

The estimated lifetime prevalence of having one or more of the disorders considered here varies widely across the WMH surveys, from 47.4% in the United States to 12.0% in Nigeria. The inter-quartile range (IQR; 25th-75th percentiles across countries) is 18.1-36.1%. Symptoms consistent with the existence of one or more lifetime mental disorders were reported by more than one-third of respondents in five countries (Colombia, France, New Zealand, Ukraine, United States), more than one-fourth in six (Belgium, Germany, Lebanon, Mexico, The Netherlands, South Africa), and more than one-sixth in four (Israel, Italy, Japan, Spain). The re-

maining two countries, Metropolitan PRC (13.2%) and Nigeria (12.0%), had considerably lower prevalence estimates, that are likely to be downwardly biased (36, 37). Prevalence estimates for other developing countries were all above the lower bound of the inter-quartile range (Table 2).

All four classes of disorder were important components of overall prevalence. Anxiety disorders were the most prevalent in ten countries (4.8-31.0%, IQR 9.9-16.7%) and mood disorders in all but one other country (3.3-21.4%, IQR 9.8-15.8%). Impulse control disorders were the least prevalent in most countries that included a relatively full assessment of these disorders (0.3-25.0%, IQR 3.1-5.7%). Substance use disorders were generally the least prevalent elsewhere (1.3-15.0%, IQR 4.8-9.6). The Western European countries did not assess illicit drug abuse-dependence, though, leading to artificially low prevalence estimates (1.3-8.9%) compared to other countries (2.2-15.0%). Substance dependence was also assessed only in the presence of abuse, possibly further reducing estimated prevalence (38). Lifetime disorder co-occurrence was quite common, as seen by noting that the sum of prevalence across the four disorder types was generally between 30% and 50% higher than the prevalence of any disorder. Within-class co-occurrence cannot be seen in the reported results, but is even stronger than between-class co-occurrence (results available on request).

Age-of-onset distributions

Despite the wide cross-national variation in estimated lifetime prevalence, considerable cross-national consistency exists in standardized age-of-onset distributions (detailed results are not reported here, but are available on request).

Impulse control disorders have the earliest age-of-onset distributions, both in terms of early median ages of onset (7-9 years of age for attention-deficit/hyperactivity disorder, 7-15 for oppositional-defiant disorder, 9-14 for conduct disorder, and 13-21 for intermittent explosive disorder) and an extremely narrow age range of onset risk, with 80% of all lifetime attention-deficit/hyperactivity disorder beginning in the age range 4-11 and the vast majority of oppositional-defiant disorder and conduct disorder beginning between ages 5 and 15. Although the age-of-onset distribution is less concentrated for intermittent explosive disorder, fully half of all lifetime cases have onsets in childhood and adolescence.

The situation is more complex with anxiety disorders, as the age-of-onset distributions fall into two distinct sets. The phobias and separation anxiety disorder all have very early ages of onset (medians in the range 7-14, IQR 8-11). Generalized anxiety disorder, panic disorder, and post-traumatic stress disorder, in comparison, have much later age-of-onset distributions (median 24-50, IQR 31-41), with much wider cross-national variation than for the impulse control disorders or the phobias or separation anxiety disorder.

The age-of-onset distributions for mood disorders are similar to those for generalized anxiety disorder, panic disorder, and post-traumatic stress disorder. Prevalence is consistently low until the early teens, at which time a roughly linear increase begins that continues through late middle age, with a more gradual increase thereafter. The median age of onset of mood disorders ranges between the late 20s and the early 40s (29-43, IQR 35-40).

The age-of-onset distribution of substance use disorders is consistent across countries, in that few onsets occur prior to the mid teens and cumulative increase in onset is rapid in adolescence and early adulthood. Considerable cross-national variation exists, though, in the sharpness of the change in the slope as well as in the age range of this change. This cross-national variation leads to wider cross-national variation in both the median and the inter-quartile range of the age-of-onset distributions than for impulse control disorders or phobias or separation anxiety disorder, but lower variation than for mood disorders or other anxiety disorders.

Projected lifetime risk

Projected lifetime risk of any disorder as of age 75 is between 17% (United States) and 69% (Israel) higher than estimated lifetime prevalence (IQR 28-44%) (Table 2). The highest risk-to-prevalence ratios (57-69%) are in countries exposed to sectarian violence (Israel, Nigeria, and South Africa). Excluding these three, there is no strong difference in ratios of less developed (28-41%) versus developed (17-49%) countries. The highest class-specific proportional increase in projected risk is for mood disorders (45-170%, IQR 61-98%) and the lowest for impulse control disorders (0-14%, IQR 0-2%), consistent with the former having the latest and the latter having the earliest age-of-onset distribution. The projected lifetime risk estimates suggest that approximately half the population (47-55%) will eventually have a mental disorder in six countries (Colombia, France, New Zealand, South Africa, Ukraine, United States), approximately one-third (30-43%) in six other countries (Belgium, Germany, Israel, Lebanon, Mexico, the Netherlands), approximately one-fourth (24-29%) in three others (Italy, Japan, Spain), and approximately one-fifth (18-19%) in the remaining countries (Metropolitan PRC, Nigeria).

Cohort effects

Previous research has suggested that projected lifetime risk might be increasing in recent cohorts (39). Prospective tracking studies are required to monitor cohort effects directly. However, indirect approximations can be obtained in cross-sectional data using retrospective age-of-onset re-

Table 2 Lifetime prevalence and projected lifetime risk as of age 75 of DSM-IV disorders

Country	Any anxiety disorder					Any mood disorder					Any impulse control disorder					Any substance use disorder					Any disorder				
	Prevalence			Projected lifetime risk		Prevalence			Projected lifetime risk		Prevalence			Projected lifetime risk		Prevalence			Projected lifetime risk		Prevalence			Projected lifetime risk	
	%	N[a]	SE	%	SE	%	N[a]	SE	%	SE	%	N[a]	SE	%	SE	%	N[a]	SE	%	SE	%	N[a]	SE	%	SE
Belgium	13.1	219	1.9	15.7	2.5	14.1	367	1.0	22.8	1.7	5.2	31	1.4	5.2	1.4	8.3	195	0.9	10.5	1.1	29.1	519	2.3	37.1	3.0
Colombia	25.3	948	1.4	30.9	2.5	14.6	666	0.7	27.2	2.0	9.6	273	0.8	10.3	0.9	9.6	345	0.6	12.8	1.0	39.1	1432	1.3	55.2[d]	6.0
France	22.3	445	1.4	26.0	1.6	21.0	648	1.1	30.5	1.4	7.6	71	1.3	7.6	1.3	7.1	202	0.5	8.8	0.6	37.9	847	1.7	47.2	1.6
Germany	14.6	314	1.5	16.9	1.7	9.9	372	0.6	16.2	1.3	3.1	31	0.8	3.1	0.8	6.5	228	0.6	8.7	0.9	25.2	573	1.9	33.0	2.5
Israel	5.2	252	0.3	10.1	0.9	10.7	524	0.5	21.2	1.6	-[b]	-	-	-	-	5.3	261	0.3	6.3	0.4	17.6	860	0.6	29.7	1.5
Italy	11.0	328	0.9	13.7	1.2	9.9	452	0.5	17.3	1.2	1.7	27	0.4	-[c]	-	1.3	56	0.2	1.6	0.3	18.1	612	1.1	26.0	1.9
Japan	6.9	155	0.6	9.2	1.2	7.6	183	0.5	14.1	1.7	2.8	11	1.0	-[c]	-	4.8	69	0.5	6.2	0.7	18.0	343	1.1	24.4	1.8
Lebanon	16.7	282	1.6	20.2	1.8	12.6	352	0.9	20.1	1.2	4.4	53	0.9	4.6	1.0	2.2	27	0.8	-	-[c]	25.8	491	1.9	32.9	2.1
Mexico	14.3	684	0.9	17.8	1.6	9.2	598	0.5	20.4	1.7	5.7	152	0.6	5.7	0.6	7.8	378	0.5	11.9	1.0	26.1	1148	1.4	36.4[d]	2.1
Netherlands	15.9	320	1.1	21.4	1.8	17.9	476	1.0	28.9	1.9	4.7	37	1.1	4.8	1.1	8.9	210	0.9	11.4	1.2	31.7	633	2.0	42.9	2.5
New Zealand	24.6	3171	0.7	30.3	1.5	20.4	2755	0.5	29.8	0.7	-[b]	-	-	-	-	12.4	1767	0.4	14.6	0.5	39.3	4815	0.9	48.6	1.5
Nigeria	6.5	169	0.9	7.1	0.9	3.3	236	0.3	8.9	1.2	0.3	9	0.1	-[c]	-	3.7	119	0.4	6.4	1.0	12.0	440	1.0	19.5	1.9
PR China	4.8	159	0.7	6.0	0.8	3.6	185	0.4	7.3	0.9	4.3	37	0.9	4.9	0.9	4.9	128	0.7	6.1	0.8	13.2	419	1.3	18.0	1.5
South Africa	15.8	695	0.8	30.1	4.4	9.8	439	0.7	20.0	2.4	-[b]	-	-	-	-	13.3	505	0.9	17.5	1.2	30.3	1290	1.1	47.5	3.7
Spain	9.9	375	1.1	13.3	1.4	10.6	672	0.5	20.8	1.2	2.3	40	0.8	2.3	0.8	3.6	180	0.4	4.6	0.5	19.4	842	1.4	29.0	1.8
Ukraine	10.9	371	0.8	17.3	2.0	15.8	814	0.8	25.9	1.5	8.7	91	1.1	9.7	1.3	15.0	293	1.3	18.8	1.7	36.1	1074	1.5	48.9	2.5
United States	31.0	2692	1.0	36.0	1.4	21.4	2024	0.6	31.4	0.9	25.0	1051	1.1	25.6	1.1	14.6	1144	0.6	17.4	0.6	47.4	3929	1.1	55.3	1.2

[a]The numbers reported here are the numbers of respondents with the disorders indicated in the column heading. The denominators used to calculate prevalence estimates based on these numbers of cases are reported in Table 1. In the case of anxiety disorders and substance use disorders, the denominators are the numbers of respondents in the Part II sample. In the case of mood disorders, the denominators are the numbers of respondents in the Part I sample. In the case of impulse control disorders and any disorders, the denominators are the numbers of respondents aged ≤44 in the Part II sample

[b]Impulse control disorders not assessed

[c]Cell size was too small to be included in analysis

[d]Projected lifetime risk to age 65 due to the sample including only respondents up to age 65

ports. This was done in the WMH data using discrete-time survival analysis to predict onset of disorders across age groups 18-34, 35-49, 50-64, and 65+. As these surveys were completed between 2002 and 2005, the most recent cohorts (aged 18-34 at interview) roughly correspond to those born in the years from 1968+. Respondents aged 35-49 at interview correspond roughly to cohorts born in 1953-1970, while those aged 50-64 were born in 1938-1955, and those aged 65+ were born before 1938. Survival analysis finds that the odds ratios for anxiety, mood, and substance use disorders are generally higher in recent compared to older cohorts, while not for impulse control disorders (Tables 3-5). No meaningful difference exists between less developed and developed countries, although cross-national variation exceeds chance expectations.

DISCUSSION

Three possible biases could have led to under-estimating prevalence. First, people with mental illness have been found to be less likely than others to participate in surveys, because of sample frame exclusions (e.g., excluding homeless people), differential mortality, or greater reluctance to participate (40). Variation in the magnitude of such under-representation across countries could help account for the wide between-country variation in prevalence-risk estimates. Second, previous research suggests that lifetime prevalence is sometimes under-reported because of respondent reluctance to admit mental illness (41). This bias might be especially strong in less developed countries with no strong tradition of independent public opinion research, which could help account for the especially low prevalence-risk estimates in Nigeria and Metropolitan PRC. Third, interviewer error might have led to under-reporting, especially in countries where there was an indirect incentive to rush through interviews, because interviewers were paid by the interview rather than by the hour. The most plausible bias that could have led to over-estimating prevalence, in comparison, is that the interview thresholds for defining disorders might have been too liberal. However, as noted in the section on measures, clinical reappraisal studies carried out in some of the countries with the highest prevalence estimates found no evidence of such bias (27).

Two possible biases of other sorts are also noteworthy. First, the method used to estimate lifetime risk was based on the assumption of constant conditional risk of first onset in a given year of life across cohorts. The existence of an apparent cohort effect means that this assumption is incorrect, probably causing an under-estimation of lifetime risk in younger cohorts. Second, age of onset might have been recalled with error related to age at interview, which could produce the data pattern found here as indirect evidence for a cohort effect (42). Evidence for age-related bias has been documented in previous epidemiological research (29), although the novel probing strategy used in the WMH surveys has been shown to minimize this problem (30).

Based on these considerations, the wide cross-national variation in WMH prevalence and risk estimates should be

Table 3 Inter-cohort differences in lifetime risk of any DSM-IV anxiety disorder[a]

Country	18-34			35-49			50-64			65+[b]			χ^2	df	N
	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N			
Belgium	2.6*	1.3-5.0	254	1.6	0.8-3.2	331	1.3	0.6-2.6	278	1.0	-	180	14.2*	3	1043
Colombia	1.6*	1.2-2.1	1125	1.3	0.9-1.8	818	1.0	-	438	-	-	-	10.0*	2	2381
France	3.1*	1.5-6.4	388	3.2*	1.5-6.7	472	1.6	0.8-3.3	362	1.0	-	214	21.3*	3	1436
Germany	3.1*	1.9-5.1	316	2.3*	1.4-3.9	436	2.3*	1.3-4.1	345	1.0	-	226	21.8*	3	1323
Israel	4.7*	2.6-8.3	1627	2.7*	1.6-4.4	1302	2.1*	1.4-3.3	1069	1.0	-	861	27.3*	3	4859
Italy	1.5	0.7-3.0	496	1.6	0.9-2.8	516	1.3	0.8-2.2	454	1.0	-	313	3.3	3	1779
Japan	5.6*	2.2-13.8	155	2.8*	1.3-6.1	219	2.6*	1.2-5.6	295	1.0	-	218	14.9*	3	887
Lebanon	3.2*	1.6-6.2	349	2.5*	1.2-5.1	348	1.0	0.5-2.1	199	1.0	-	135	24.1*	3	1031
Mexico	2.4*	1.6-3.4	1183	1.6*	1.1-2.4	750	1.0	-	429	-	-	-	25.3*	2	2362
Netherlands	3.6*	2.1-6.1	264	4.5*	3.0-6.8	358	3.0*	2.0-4.6	302	1.0	-	170	60.6*	3	1094
New Zealand	3.4*	2.7-4.2	2394	2.6*	2.1-3.1	2474	2.1*	1.7-2.7	1517	1.0	-	927	126.3*	3	7312
Nigeria	3.1*	1.4-6.9	971	2.3*	1.1-4.9	549	2.8*	1.5-5.4	369	1.0	-	254	11.1*	3	2143
PR China	1.7	0.6-4.4	379	1.1	0.5-2.5	726	1.6	0.7-3.9	357	1.0	-	166	3.3	3	1628
South Africa	2.3*	1.3-4.0	2172	1.8*	1.1-3.1	1264	1.3	0.8-2.1	638	1.0	-	241	16.5*	3	4315
Spain	3.8*	2.2-6.5	545	2.8*	1.5-5.2	556	1.3	0.8-2.2	456	1.0	-	564	28.7*	3	2121
Ukraine	1.7*	1.1-2.6	420	1.0	0.6-1.6	434	1.0	0.7-1.6	412	1.0	-	454	6.5	3	1720
United States	3.5*	2.8-4.4	1939	3.4*	2.7-4.1	1831	2.5*	2.0-3.0	1213	1.0	-	709	159.2*	3	5692

[a]Based on discrete-time survival models with person-year as the unit of analysis, controls are time intervals
[b]Referent category
*Significant at the .05 level, two-sided test

Table 4 Inter-cohort differences in lifetime risk of any DSM-IV mood disorder[a]

Country	18-34			35-49			50-64			65+[b]			χ^2	df	N
	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N			
Belgium	11.3*	6.1-20.9	573	4.9*	3.2-7.5	775	3.6*	2.0-6.4	570	1.0	-	501	87.3*	3	2419
Colombia	6.3*	4.2-9.3	2000	2.3*	1.6-3.1	1577	1.0	-	849	-	-	530	92.7*	2	4426
France	9.0*	6.0-13.5	743	3.0*	2.2-4.2	942	1.8*	1.2-2.6	719	1.0	-	490	146.4*	3	2894
Germany	12.2*	7.1-21.0	815	5.2*	3.5-7.7	1180	2.4*	1.6-3.4	893	1.0	-	667	94.4*	3	3555
Israel	6.5*	4.5-9.4	1627	2.8*	2.0-4.0	1302	1.8*	1.3-2.5	1069	1.0	-	861	118.4*	3	4859
Italy	5.7*	3.8-8.4	1326	3.6*	2.6-5.0	1393	2.3*	1.6-3.3	1153	1.0	-	840	91.3*	3	4712
Japan	23.7*	13.4-42.0	410	7.7*	4.5-13.2	571	3.8*	2.4-5.8	764	1.0	-	691	146.2*	3	2436
Lebanon	6.2*	3.0-12.8	965	3.1*	1.4-6.7	931	1.7	0.8-3.2	553	1.0	-	408	60.5*	3	2857
Mexico	4.0*	2.6-6.1	2871	1.6*	1.1-2.3	1888	1.0	-	1023	-	-	646	65.0*	2	5782
Netherlands	11.7*	6.6-20.8	564	6.4*	4.0-10.2	729	2.9*	1.7-4.8	627	1.0	-	452	115.7*	3	2372
New Zealand	10.0*	8.2-12.2	3747	5.0*	4.1-6.0	4102	2.9*	2.4-3.6	2697	1.0	-	2244	653.9*	3	12790
Nigeria	3.7*	1.8-7.6	3175	1.8	0.9-3.6	1631	1.2	0.7-2.1	1104	1.0	-	842	19.4*	3	6752
PR China	20.8*	9.4-45.8	1209	4.4*	2.3-8.4	2261	2.5*	1.4-4.4	1184	1.0	-	547	76.5*	3	5201
South Africa	9.6*	5.5-16.7	2172	5.5*	3.1-9.9	1264	2.5*	1.4-4.4	638	1.0	-	241	95.6	3	4315
Spain	9.6*	6.6-13.9	1567	4.2*	3.0-5.9	1431	2.2*	1.6-3.0	1024	1.0	-	1451	176.3*	3	5473
Ukraine	1.9*	1.4-2.4	1194	1.0	0.8-1.3	1225	0.9	0.8-1.1	1180	1.0	-	1126	38.2*	3	4725
United States	9.5*	7.3-12.4	3034	5.0*	3.7-6.6	2865	3.0*	2.3-3.9	1922	1.0	-	1461	383.6*	3	9282

[a]Based on discrete-time survival models with person-year as the unit of analysis, controls are time intervals
[b]Referent category
*Significant at the .05 level, two-sided test

interpreted with caution, because it is likely over-estimated due to between-country differences in some of the biases enumerated above. The overall prevalence-risk estimates, which are consistent with previous cross-national research (8-14,39), are likely to be conservative, as the most plausible biases lead to under-estimation. The evidence for cohort effects is more difficult to judge, as both substantive and methodological interpretations are plausible. The options are either that the prevalence of mental disorders is on the rise or that prevalence is stable but under-estimated among older respondents.

Given the high prevalence-risk estimates even with the possibility of conservative bias, a question can be raised about the meaningfulness of these estimates. Our clinical reappraisal studies, consistent with comparable studies carried out in conjunction with previous community psychiatric epidemiological surveys (43), show that the high prevalence estimates are genuine (i.e., consistent with expert clinician judgments) rather than due to CIDI errors. It is important to recognize, though, that not all mental disorders are severe. WMH measures of disorder severity were applied only to 12-month cases, so we have no way to estimate severity of lifetime cases. Analysis of 12-month cases, though, finds the majority rated mild on a clinical rating scale with categories mild, moderate, and severe (22). These cases are nonetheless meaningful, because even mild cases can be impairing and often evolve into more serious disorders over time (44).

Table 5 Inter-cohort differences in lifetime risk of any DSM-IV substance use disorder[a]

Country	18-34			35-49			50-64			65+[b]			χ^2	df	N
	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N	OR	95% CI	N			
Belgium	5.0*	2.6-9.8	254	3.6*	1.7-7.3	331	2.6*	1.2-5.4	278	1.0	-	180	26.7*	3	1043
Colombia	2.3*	1.6-3.3	2000	1.1	0.7-1.6	1577	1.0	-	849	-	-	530	39.3*	2	4426
France	5.8*	3.3-10.0	388	3.3*	2.0-5.7	472	2.5*	1.4-4.2	362	1.0	-	214	44.1*	3	1436
Germany	5.6*	2.9-10.7	316	3.7*	2.0-6.8	436	3.9*	2.1-7.1	345	1.0	-	226	35.0*	3	1323
Israel	11.3*	5.9-21.6	1627	4.6*	2.4-9.0	1302	2.5*	1.2-5.1	1069	1.0	-	861	119.9*	3	4859
Italy	2.6*	1.0-6.7	496	1.8	0.8-4.1	516	1.6	0.6-3.9	454	1.0	-	313	5.5	3	1779
Japan	1.9	0.6-6.0	155	2.3*	1.1-4.9	219	2.5*	1.1-5.7	295	1.0	-	218	6.7	3	887
Lebanon[c]	-	-		-	-		-	-		-	-		-	-	-
Mexico	1.7*	1.3-2.4	2871	1.2	0.9-1.7	1888	1.0	-	1023	-	-	646	12.8*	2	5782
Netherlands	12.4*	7.0-21.8	264	7.0*	3.8-13.1	358	6.8*	3.4-13.9	302	1.0	-	170	85.3*	3	1094
New Zealand	8.1*	6.1-10.7	3747	3.5*	2.7-4.7	4102	2.5*	1.9-3.3	2697	1.0	-	2244	283.7*	3	12790
Nigeria	3.4*	1.1-10.1	971	4.9*	1.8-13.3	549	2.9	1.0-8.7	369	1.0	-	254	11.8*	3	2143
PR China	8.2*	1.0-67.2	379	4.0	0.6-28.2	726	1.5	0.2-11.2	357	1.0	-	166	31.9*	3	1628
South Africa	2.6*	1.3-5.4	2172	1.5	0.8-2.9	1264	1.0	0.6-1.9	638	1.0	-	241	29.1	3	4315
Spain	9.3*	3.6-24.2	545	5.0*	1.8-13.7	556	1.5	0.6-4.2	456	1.0	-	564	38.1*	3	2121
Ukraine	10.8*	5.8-20.1	420	5.0*	2.4-10.4	434	2.8*	1.3-5.8	412	1.0	-	454	116.4*	3	1720
United States	6.7*	4.6-10.0	1939	4.9*	3.5-7.0	1831	3.5*	2.4-5.3	1213	1.0	-	709	111.0*	3	5692

[a]Based on discrete-time survival models with person-year as the unit of analysis, controls are time intervals
[b]Referent category
[c]Cell size too small to be included in analysis
*Significant at the .05 level, two-sided test

The age-of-onset distributions reported here are consistent with those in previous epidemiological surveys (39,45). Given the enormous personal and societal burdens of mental disorders, the finding that many cases have early ages of onset suggests that public health interventions might profitably begin in childhood. Importantly, studies of initial contact with the treatment system (46-48) show that people with these early-onset disorders often wait more than a decade before seeking treatment, and present with seriously impairing disorders that might have been easier to treat if they had sought treatment earlier in the course of illness. Interventions aimed at early detection and treatment might help reduce the persistence or severity of these largely primary anxiety and impulse control disorders and prevent the onset of secondary disorders. More preclinical and clinical research is needed on treatments of early cases, though, to determine whether this is true. Epidemiological research is also needed on the long-term consequences of early interventions for long-term secondary prevention.

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References

1. Cooper B. Psychiatric epidemiology. London: Croom Helm, 1987.
2. Robins LN, Helzer JE, Croughan JL et al. National Institute of Mental Health Diagnostic Interview Schedule: its history, characteristics and validity. Arch Gen Psychiatry 1981;38:381-9.
3. Cross-National Collaborative Group. The changing rate of major depression: cross-national comparisons. JAMA 1992;268:3098-105.
4. Weissman MM, Bland RC, Canino GJ et al. The cross-national epidemiology of panic disorder. Arch Gen Psychiatry 1997;54:305-9.
5. Weissman MM, Bland RC, Canino GJ et al. Cross-national epidemiology of major depression and bipolar disorder. JAMA 1996; 276:293-9.
6. Wittchen HU. Reliability and validity studies of the WHO Composite International Diagnostic Interview (CIDI): a critical review. J Psychiatr Res 1994;28:57-84.
7. World Health Organization. Composite International Diagnostic Interview (CIDI, Version 1.0). Geneva: World Health Organization, 1990.
8. Andrade L. Lifetime prevalence of mental disorders in a catchment area in São Paulo, Brazil. Presented at the 7th Congress of the International Federation of Psychiatric Epidemiology, Santiago, August 1996.
9. Bijl RV, van Zessen G, Ravelli A et al. The Netherlands Mental Health Survey and Incidence Study (NEMESIS): objectives and design. Soc Psychiatry Psychiatr Epidemiol 1998;33:581-6.
10. Caraveo J, Martinez J, Rivera B. A model for epidemiological studies on mental health and psychiatric morbidity. Salud Mental 1998;21:48-57.
11. Kessler RC, McGonagle KA, Zhao S et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Results from the National Comorbidity Survey. Arch Gen Psychiatry 1994;51:8-19.
12. Kylyc C. Mental health profile of Turkey: main report. Ankara: Ministry of Health Publications, 1998.
13. Vega WA, Kolody B, Aguilar-Gaxiola S et al. Lifetime prevalence of DSM-III-R psychiatric disorders among urban and rural Mexican Americans in California. Arch Gen Psychiatry 1998;55:771-8.
14. Wittchen H-U, Perkonigg A, Lachner G et al. Early Developmental Stages of Psychopathology study (EDSP): objectives and design. Eur Addict Res 1998;4:18-27.
15. Murray CJL, Lopez AD. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020. Cambridge: Harvard University Press, 1996.
16. Bijl RV, de Graaf R, Hiripi E et al. The prevalence of treated and untreated mental disorders in five countries. Health Aff 2003;22: 122-33.
17. Amminger GP, Leicester S, Yung AR et al. Early-onset of symptoms predicts conversion to non-affective psychosis in ultra-high risk individuals. Schizophr Res 2006;84:67-76.
18. McGue M, Iacono WG. The association of early adolescent problem behavior with adult psychopathology. Am J Psychiatry 2005; 162:1118-24.
19. Thompson KN, Conus PO, Ward JL et al. The initial prodrome to bipolar affective disorder: prospective case studies. J Affect Disord 2003;77:79-85.
20. Alonso J, Angermeyer MC, Bernert S et al. Sampling and methods of the European Study of the Epidemiology of Mental Disorders (ESEMeD) project. Acta Psychiatr Scand 2004;420(Suppl.):8-20.
21. World Bank. World development indicators 2003. Washington: World Bank, 2003.
22. Demyttenaere K, Bruffaerts R, Posada-Villa J et al. Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. JAMA 2004;291:2581-90.
23. Kessler RC, Merikangas KR. The National Comorbidity Survey Replication (NCS-R): background and aims. Int J Methods Psychiatr Res 2004;13:60-8.
24. Kessler RC, Ustun TB. The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). Int J Methods Psychiatr Res 2004;13:93-121.
25. World Health Organization. International classification of diseases (ICD-10). Geneva: World Health Organization, 1991.
26. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington: American Psychiatric Association, 1994.
27. Haro JM, Arbabzadeh-Bouchez S, Brugha TS et al. Concordance of the Composite International Diagnostic Interview Version 3.0 (CIDI 3.0) with standardized clinical assessments in the WHO World Mental Health Surveys. Int J Methods Psychiatr Res 2006; 15:167-80.
28. First MB, Spitzer RL, Gibbon M et al. Structured Clinical Interview for DSM-IV Axis I Disorders, research version, non-patient edition (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute, 2002.
29. Simon GE, VonKorff M. Recall of psychiatric history in cross-sectional surveys: implications for epidemiologic research. Epidemiol Rev 1995;17:221-7.
30. Knauper B, Cannell CF, Schwarz N et al. Improving the accuracy of major depression age of onset reports in the US National Comorbidity Survey. Int J Methods Psychiatr Res 1999;8:39-48.
31. SAS Institute. SAS/STAT software: changes and enhancements, Release 8.2. Cary: SAS Publishing, 2001.
32. Efron B. Logistic regression, survival analysis, and the Kaplan-Meier curve. J Am Stat Assoc 1988;83:414-25.
33. Wolter KM. Introduction to variance estimation. New York: Springer, 1985.
34. Research Triangle Institute. SUDAAN: Professional Software for Survey Data Analysis. Research Triangle Park: Research Triangle Institute, 2002.
35. Kish L, Frankel MR. Inferences from complex samples. J Royal Stat Soc 1974;36:1-37.

36. Gureje O, Lasebikan VO, Kola L et al. Lifetime and 12-month prevalence of mental disorders in the Nigerian Survey of Mental Health and Well-Being. Br J Psychiatry 2006;188:465-71.
37. Shen YC, Zhang MY, Huang YQ et al. Twelve-month prevalence, severity, and unmet need for treatment of mental disorders in metropolitan China. Psychol Med 2006;36:257-67.
38. Hasin DS, Grant BF. The co-occurrence of DSM-IV alcohol abuse in DSM-IV alcohol dependence: results of the National Epidemiologic Survey on Alcohol and Related Conditions on heterogeneity that differ by population subgroup. Arch Gen Psychiatry 2004; 61:891-6.
39. WHO International Consortium in Psychiatric Epidemiology. Cross-national comparisons of the prevalences and correlates of mental disorders. Bull World Health Organ 2000;78:413-26.
40. Allgulander C. Psychoactive drug use in a general population sample, Sweden: correlates with perceived health, psychiatric diagnoses, and mortality in an automated record-linkage study. Am J Publ Health 1989;79:1006-10.
41. Cannell CF, Marquis KH, Laurent A. A summary of studies of interviewing methodology: 1959-1970. Vital Health Stat 1977;2:69.
42. Giuffra LA, Risch N. Diminished recall and the cohort effect of major depression: a simulation study. Psychol Med 1994;24:375-83.
43. Kessler RC, Wittchen H-U, Abelson JM et al. Methodological studies of the Composite International Diagnostic Interview (CIDI) in the US National Comorbidity Survey. Int J Methods Psychiatr Res 1998;7:33-55.
44. Kessler RC, Merikangas KR, Berglund P et al. Mild disorders should not be eliminated from the DSM-V. Arch Gen Psychiatry 2003;60:1117-22.
45. Christie KA, Burke JDJ, Regier DA et al. Epidemiologic evidence for early onset of mental disorders and higher risk of drug-abuse in young-adults. Am J Psychiatry 1988;145:971-5.
46. Christiana JM, Gilman SE, Guardino M et al. Duration between onset and time of obtaining initial treatment among people with anxiety and mood disorders: an international survey of members of mental health patient advocate groups. Psychol Med 2000; 30:693-703.
47. Olfson M, Kessler RC, Berglund PA et al. Psychiatric disorder onset and first treatment contact in the United States and Ontario. Am J Psychiatry 1998;155:1415-22.
48. Wang PS, Angermeyer M, Borges G et al. Delay and failure in treatment seeking after first onset of mental disorders in the World Health Organization's World Mental Health Survey Initiative. World Psychiatry 2007;6:177-85.

Delay and failure in treatment seeking after first onset of mental disorders in the World Health Organization's World Mental Health Survey Initiative

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Data are presented on patterns of failure and delay in making initial treatment contact after first onset of a mental disorder in 15 countries in the World Health Organization (WHO)'s World Mental Health (WMH) Surveys. Representative face-to-face household surveys were conducted among 76,012 respondents aged 18 and older in Belgium, Colombia, France, Germany, Israel, Italy, Japan, Lebanon, Mexico, the Netherlands, New Zealand, Nigeria, People's Republic of China (Beijing and Shanghai), Spain, and the United States. The WHO Composite International Diagnostic Interview (CIDI) was used to assess lifetime DSM-IV anxiety, mood, and substance use disorders. Ages of onset for individual disorders and ages of first treatment contact for each disorder were used to calculate the extent of failure and delay in initial help seeking. The proportion of lifetime cases making treatment contact in the year of disorder onset ranged from 0.8 to 36.4% for anxiety disorders, from 6.0 to 52.1% for mood disorders, and from 0.9 to 18.6% for substance use disorders. By 50 years, the proportion of lifetime cases making treatment contact ranged from 15.2 to 95.0% for anxiety disorders, from 7.9 to 98.6% for mood disorders, and from 19.8 to 86.1% for substance use disorders. Median delays among cases eventually making contact ranged from 3.0 to 30.0 years for anxiety disorders, from 1.0 to 14.0 years for mood disorders, and from 6.0 to 18.0 years for substance use disorders. Failure and delays in treatment seeking were generally greater in developing countries, older cohorts, men, and cases with earlier ages of onset. These results show that failure and delays in initial help seeking are pervasive problems worldwide. Interventions to ensure prompt initial treatment contacts are needed to reduce the global burdens and hazards of untreated mental disorders.

Key words: Treatment seeking, anxiety disorders, mood disorders, substance use disorders

(World Psychiatry 2007;6:177-185)

Worldwide, mental disorders inflict tremendous morbidity, mortality, and impairment (1,2). Although the armamentarium of effective treatments keeps growing, few nations seem able or willing to pay for their widespread use (3). Indeed, the majority of people with recent episodes of mental illnesses continue to go untreated, even in economically-advantaged societies (4). This reality has left many nations searching for strategies to use what limited resources they do have as efficiently as possible in an effort to alleviate burden given current constraints (5).

One promising strategy is to emphasize use of treatment resources earlier in the disease courses of affected individuals, before many negative sequelae from mental illnesses develop (6). Such an approach is supported by several lines of research. Data from preclinical studies suggest that neural "kindling" can cause untreated disorders to become more frequent, spontaneous, severe, and treatment refractory (7). Epidemiologic studies suggest that school and job failure, teenage child-bearing, and early, violent, or unstable marriages are associated with early-onset untreated mental disorders (8-10). Single disorders often progress to complex comorbid disorders that are more difficult to treat and more likely to recur than less complex conditions (11). In addition, clinical trials have shown that timely intervention can prevent suicidality (12).

A crucial first step in reducing delays in seeking treatment after first onset of a mental disorder is to document the current state of affairs with regard to the delays that currently exist in the population and the predictors of those delays. Unfortunately, very little is known about ini-

tial treatment contact, as mental health services research has focused on recent treatment of current episodes rather than initial treatment of incident cases (13). However, the few existing studies that have examined initial treatment seeking have found that many lifetime cases eventually make contact, but usually after delaying years from when the disorders began (14-16).

A second critical step is identifying what nations can concretely do to shorten periods of untreated mental illness. Although countries employ a wide variety of national policies, delivery system designs, and means of financing mental health services, the impacts of these on delays in initial treatment seeking are unknown. Perhaps the only way to shed light on these impacts is to compare delays across countries with different policy, delivery system, and financing features (3,17). Unfortunately, very few such cross-national studies of delays have been conducted (14,15).

The current report begins to address these issues by analyzing data from the World Health Organization (WHO)'s World Mental Health (WMH) Initiative, a program of coordinated surveys being conducted in 28 developed and developing countries (1). We start by constructing cumulative lifetime probability of treatment contact curves to estimate probabilities of help-seeking for mental disorders and the typical duration of delays. We do so separately for 15 countries in which WMH surveys are now complete. To begin to understand potential determinants as well as developing and targeting future interventions, we also examine correlates of failure to make initial treatment contact.

METHODS

Samples

Countries with completed WMH surveys used in these analyses included Belgium, Colombia, France, Germany, Israel, Italy, Japan, Lebanon, Mexico, the Netherlands, New Zealand, Nigeria, People's Republic of China (Beijing and Shanghai), Spain and the United States. Employing designations made by the World Bank (18), China, Colombia, Lebanon, Mexico and Nigeria were categorized as less developed and the remainder as developed. Trained lay interviewers conducted all surveys face-to-face among multistage household probability samples. Individual country sample sizes ranged from 2,372 in the Netherlands to 12,992 in New Zealand, and the total sample size was 76,012. Response rates in individual countries ranged from 45.9% in France to 87.7% in Colombia and the weighted average response rate across all countries was 71.1%. Details on response rates and other design issues are presented in the paper by Kessler et al (19).

Part I of the survey contained core diagnostic assessments and was completed by all respondents. All Part I respondents who met criteria for any disorder and a sub-sample of approximately 25% of others were administered Part II, which assessed correlates, service use, and disorders of secondary interest. Details concerning the standardized survey methods (e.g., interviewer training procedures, WHO translation protocols for all study materials, and quality control procedures for interviewer and data accuracy) employed in all WMH surveys are available elsewhere (1,20,21). Informed consent was obtained prior to beginning all interviews. Informed consent procedures and human subjects safeguards were approved by the Institutional Review Boards of organizations coordinating the survey in each country.

Diagnostic assessments

The WHO's Composite International Diagnostic Interview (CIDI) Version 3.0 (22,23) was used to assess mental disorders using DSM-IV criteria. Disorders considered in this report include mood disorders (major depressive episode, dysthymia, and bipolar disorder I or II, or subthreshold bipolar disorder), anxiety disorders (panic disorder, specific phobia, social phobia, generalized anxiety disorder), and substance use disorders (alcohol and drug abuse and dependence). Lifetime prevalence and age of onset were assessed separately for each disorder (19). All diagnoses are considered with organic exclusions and without diagnostic hierarchy rules.

Blinded clinical reappraisal studies using the Structured Clinical Interview for DSM-IV (SCID) (25) have shown generally good concordance between DSM-IV diagnoses based on the CIDI 3.0 and the SCID for anxiety, mood, and substance use disorders (22). The recent clinical reappraisal studies carried out in four WMH countries (the United States, Italy, Spain, and France, with total N=468) have provided evidence for a good concordance between CIDI-3.0 diagnoses and diagnoses based on blinded re-interviews, with area under the receiver operator characteristics curve ranging between 0.71 and 0.93 for lifetime mood/anxiety disorders, and between 0.83 and 0.88 for 12-month mood/anxiety disorders (26).

Initial treatment contacts

In each CIDI diagnostic section, respondents were asked whether they ever in their life talked to a medical doctor or other professional about the disorder under investigation. When asking this question, interviewers clarified that the term "other professional" was intended to apply broadly and include a wide range such as psychologists, counselors, spiritual advisors, herbalists, acupuncturists, and any other healing professionals. Respondents who reported that they ever talked to any professional about the disorder being assessed were then asked how old they were the first time they did so. Responses to this question were used to define ages of first treatment contact. Data from WMH countries (e.g., South Africa, Ukraine) in which dis-

order-specific questions about treatment were not asked are not included in this analysis.

Predictor variables

Predictors included age of onset of the disorder being assessed, cohort, and gender. Age of onset was categorized separately for each country as early (25th percentile), early-average (50th percentile), late-average (75th percentile), and late onset. Cohort was defined by age at interview and categorized as 18-34, 35-49, 50-64, 65+ years.

Analysis procedures

Estimated projections of the cumulative probability of treatment contact in the year of disorder onset and by 50 years after onset were made using the actuarial method of survival analysis (27) implemented in SAS (version 8.2, SAS Institute, Cary, N.C.). Separate curves were generated for each country. Typical durations of delay in initial treatment contact were defined as the median years from disorder onset to first treatment contact among cases that eventually made treatment contact. Correlates of treatment contact were examined separately for each disorder using discrete-time survival analysis (28) with person-year as the unit of analysis. Time-invariant predictors included age of onset of the disorder, cohort, and gender. The only time-varying predictor was the number of years since first onset of the disorder. Models were estimated among all respondents with the disorder to identify predictors of ever making treatment contact. Effects of weighting and clustering on significance tests were adjusted for using the Taylor series linearization method (29) implemented in SUDAAN (version 8.0.1, Research Triangle Institute, N.C.). Wald χ^2 tests using Taylor series design-based coefficient variance-covariance matrices were used to make multivariate significance tests in the discrete-time survival analyses. Statistical significance was evaluated using .05 level, two-sided tests.

RESULTS

Cumulative probabilities and median delays in treatment contact

The first column of Table 1 presents the proportions of lifetime cases with anxiety disorders making treatment contact in the year of disorder onset. The proportion ranged from a low of 0.8% in Nigeria to a high of 36.4% in Israel, with an inter-quartile range (IQR: 25th -75th percentiles) of 3.6-19.8%. The proportions of lifetime cases with anxiety disorders making treatment contact by 50 years are shown in the second column of Table 1 and ranged from 15.2% in Nigeria to 95.0% in Germany (IQR 44.7-90.7%). The median duration of delay among cases with anxiety disorders that eventually made treatment contact is shown in the third col-

Table 1 Proportional treatment contact in the year of onset of any anxiety disorder and median duration of delay among cases that subsequently made treatment contact

	Making treatment contact in year of onset, % (SE)	Making treatment contact by 50 years, % (SE)	Median duration of delay in years (SE)
The Americas			
Colombia	2.9 (0.6)	41.6 (3.9)	26.0 (1.5)
Mexico	3.6 (1.1)	53.2 (18.2)	30.0 (5.1)
USA	11.3 (0.7)	87.0 (2.4)	23.0 (0.6)
Europe			
Belgium	19.8 (2.8)	84.5 (4.9)	16.0 (3.5)
France	16.1 (1.8)	93.3 (1.9)	18.0 (1.8)
Germany	13.7 (1.8)	95.0 (2.3)	23.0 (2.3)
Italy	17.1 (2.1)	87.3 (8.5)	28.0 (2.2)
Netherlands	28.0 (3.7)	91.1 (2.8)	10.0 (1.6)
Spain	23.2 (2.0)	86.6 (5.2)	17.0 (3.2)
Africa and Middle East			
Israel	36.4 (0.9)	90.7 (1.3)	3.0 (0.1)
Lebanon	3.2 (1.1)	37.3 (11.5)	28.0 (3.9)
Nigeria	0.8 (0.5)	15.2 (2.6)	16.0 (4.2)
Asia and the Pacific			
Japan	11.2 (2.4)	63.1 (6.2)	20.0 (2.4)
PR China	4.2 (2.0)	44.7 (7.2)	21.0 (3.1)
Oceania			
New Zealand	12.5 (0.8)	84.2 (2.5)	21.0 (0.8)

Table 2 Proportional treatment contact in the year of onset of any mood disorder and median duration of delay among cases that subsequently made treatment contact

	Making treatment contact in year of onset, % (SE)	Making treatment contact by 50 years, % (SE)	Median duration of delay in years (SE)
The Americas			
Colombia	18.7 (2.7)	66.6 (3.7)	9.0 (1.6)
Mexico	16.0 (2.2)	69.9 (8.5)	14.0 (3.1)
USA	35.4 (1.2)	94.8 (2.5)	4.0 (0.2)
Europe			
Belgium[a]	47.8 (2.7)	93.7 (2.5)	1.0 (0.3)
France[a]	42.7 (2.1)	98.6 (1.4)	3.0 (0.3)
Germany[a]	40.4 (3.8)	89.1 (5.0)	2.0 (0.4)
Italy[a]	28.8 (3.0)	63.5 (5.9)	2.0 (0.5)
Netherlands[a]	52.1 (2.9)	96.9 (1.7)	1.0 (0.3)
Spain[a]	48.5 (2.3)	96.4 (3.1)	1.0 (0.3)
Africa and Middle East			
Israel	31.9 (0.8)	92.7 (0.5)	6.0 (0.3)
Lebanon	12.3 (2.0)	49.2 (5.2)	6.0 (2.1)
Nigeria	6.0 (1.7)	33.3 (7.2)	6.0 (3.3)
Asia and the Pacific			
Japan	29.6 (4.0)	56.8 (7.3)	1.0 (0.7)
PR China	6.0 (2.2)	7.9 (2.6)	1.0 (2.0)
Oceania			
New Zealand	41.4 (1.3)	97.5 (1.0)	3.0 (0.2)

[a]Used major depressive episode instead of any mood disorder

umn of Table 1. Among the fraction of cases making treatment contact, delays were shortest in Israel (median delay of 3.0 years) and longest in Mexico (median delay of 30.0 years). There were statistically significant differences between countries ($F_{15,726}$=95,259.7; p<0.001) and generally longer delays in developing vs. developed countries (detailed results are not reported, but are available on request).

As shown in Table 2, the proportions of lifetime cases with mood disorders making treatment contact in the year of disorder onset ranged from lows of 6.0% in Nigeria and China to a high of 52.1% in the Netherlands (IQR 16.0-42.7%). The proportions of cases with mood disorders making treatment contact by 50 years ranged from 7.9% in China to 98.6% in France (IQR 56.8-96.4%). Among cases with mood disorders eventually making treatment contact, the median duration of delay was shortest in three Western European (Belgium, the Netherlands, and Spain) and two Asian (China and Japan) countries (median delay of 1.0 years in each) and longest in Mexico (median delay of 14.0 years). The delays among cases with mood disorders were significantly different across countries ($F_{15,726}$=47,368.1; p<0.001) (detailed results are not reported, but are available on request). Comparison of Tables 1 and 2 reveals that delays were generally shorter for mood than anxiety disorders.

The proportions of lifetime cases with substance use disorders making treatment contact in the year of disorder onset ranged from a low of 0.9% in Mexico to a high of 18.6% in Spain (IQR 2.8-13.2%) (see Table 3). By 50 years, the proportions of cases with substance use disorders making treatment contact ranged from 19.8% in Nigeria to 86.1% in Germany (IQR 25.7-66.6%). Cases with substance use disorders eventually making treatment contact had the shortest delays in Spain (median delay of 6.0 years) and the longest in Belgium (median delay of 18.0 years). The delays among cases with substance use disorders were significantly different across countries ($F_{15,726}$=21,505.3; p<0.001) (detailed results are not reported, but are available on request). The delays among cases with substance use disorders appeared to be generally intermediate between those for mood and anxiety disorders.

Table 3 Proportional treatment contact in the year of onset of any substance use disorder and median duration of delay among cases that subsequently made treatment contact

	Making treatment contact in year of onset, % (SE)	Making treatment contact by 50 years, % (SE)	Median duration of delay in years (SE)
The Americas			
Colombia	3.6 (0.8)	23.1 (7.1)	11.0 (5.0)
Mexico	0.9 (0.5)	22.1 (4.8)	10.0 (3.3)
USA[a]	10.0 (0.8)	75.5 (3.8)	13.0 (1.2)
Europe			
Belgium	12.8 (4.8)	61.2 (17.7)	18.0 (5.8)
France	15.7 (5.4)	66.5 (14.1)	13.0 (3.7)
Germany	13.2 (5.7)	86.1 (8.6)	9.0 (3.9)
Italy	- [b]	- [b]	- [b]
Netherlands	15.5 (5.4)	66.6 (7.9)	9.0 (3.1)
Spain	18.6 (7.6)	40.1 (14.1)	6.0 (4.9)
Africa and Middle East			
Israel	2.0 (0.5)	48.0 (2.4)	12.0 (0.5)
Lebanon[a]	- [b]	- [b]	- [b]
Nigeria[a]	2.8 (1.7)	19.8 (7.2)	8.0 (1.8)
Asia and the Pacific			
Japan[a]	9.2 (5.1)	31.0 (7.8)	8.0 (4.6)
PR China[a]	2.8 (1.8)	25.7 (9.0)	17.0 (3.7)
Oceania			
New Zealand	6.3 (0.8)	84.8 (15.4)	17.0 (1.3)

[a]Assessed in the part II sample
[b]Disorder was omitted due to insufficient cases (n<30)

Correlates of lifetime treatment contact

Results from the discrete time survival models of lifetime treatment contact for anxiety disorders are shown in Table 4. Female gender was significantly associated with a higher likelihood of making initial treatment contact in four countries. Significant, monotonic relationships between being in younger cohorts and higher probabilities of treatment contact existed in 12 out of the 13 countries with significant cohort differences. Cases with earlier ages of onset of their anxiety disorders were significantly less likely to make treatment contact in 14 countries.

Correlates of lifetime treatment contact for mood disorders are shown in Table 5. Female gender was significantly associated with higher likelihoods of treatment contact in three countries. Significant, generally monotonic relationships between being in younger cohorts and higher probabilities of treatment contact existed in 10 countries. Earlier ages of onset were significantly associated with lower likelihoods of making treatment contact for mood disorders in 13 countries.

For substance use disorders, female gender was significantly associated with greater initial treatment contact in one country (see Table 6). There were significant, generally monotonic relationships between being in younger cohorts and higher probabilities of initial treatment contact in eight countries. Having an earlier age of onset was significantly associated with a lower likelihood of making treatment contact for substance use disorders in eight countries.

DISCUSSION

Several potential limitations should be kept in mind when interpreting these results. Most important is the potential that respondents failed to recall events occurring over their lifetimes. For example, those not seeking treatment may have been more likely to forget or normalize symptoms than cases who sought treatment. Unfortunately, we cannot evaluate this possibility or whether it occurred differentially across countries. However, it is worth noting that, to the extent this occurred, we have underestimated failures and delays in initial treatment seeking.

Table 4 Socio-demographic predictors of lifetime treatment contact for any anxiety disorder

Country	Sex			Cohort (age at interview)							Age of onset						
	Female		χ^2	Age 18-34		Age 35-49		Age 50-64		χ^2	Early		Early-average		Late-average		χ^2
	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	
The Americas																	
Colombia	1.1	(0.7-1.8)	0.1	3.4	(1.4-8.2)	1.6	(0.8-3.3)	1.0	-	9.6	0.2	(0.1-0.3)	0.3	(0.2-0.6)	0.3	(0.1-0.5)	33.4
Mexico	1.1	(0.6-1.8)	0.1	2.3	(0.8-6.4)	2.3	(0.8-6.4)	1.0	-	2.6	0.2	(0.1-0.3)	0.2	(0.1-0.3)	0.2	(0.1-0.3)	59.1
USA	1.3	(1.0-1.6)	5.4	2.5	(1.9-3.3)	1.4	(1.1-1.8)	1.2	(0.9-1.6)	62.6	0.2	(0.2-0.2)	0.2	(0.2-0.3)	0.2	(0.2-0.3)	326.4
Europe																	
Belgium	1.2	(0.7-2.1)	0.4	4.7	(1.6-13.6)	3.0	(1.2-7.5)	1.3	(0.6-2.8)	14.8	0.1	(0.1-0.3)	0.1	(0.0-0.3)	0.2	(0.1-0.5)	63.5
France	1.5	(1.1-2.1)	8.8	4.5	(2.5-8.1)	2.3	(1.3-4.2)	1.3	(0.7-2.5)	52.2	0.2	(0.1-0.3)	0.2	(0.1-0.3)	0.3	(0.2-0.5)	82.4
Germany	1.5	(1.1-2.1)	6.6	4.5	(2.7-7.5)	2.3	(1.5-3.7)	1.5	(0.8-2.9)	59.8	0.2	(0.1-0.3)	0.2	(0.1-0.3)	0.2	(0.1-0.5)	43.5
Italy	1.1	(0.7-1.5)	0.1	2.6	(1.3-5.2)	2.1	(1.2-3.7)	1.4	(0.7-2.9)	16.0	0.1	(0.1-0.2)	0.1	(0.1-0.2)	0.3	(0.2-0.5)	101.8
Netherlands	1.1	(0.7-1.6)	0.2	3.0	(1.8-5.1)	2.5	(1.6-3.7)	1.0	-	26.8	0.1	(0.0-0.2)	0.1	(0.1-0.3)	0.4	(0.2-0.7)	52.0
Spain	1.0	(0.7-1.6)	0.0	3.3	(1.9-5.7)	2.0	(1.1-3.7)	0.8	(0.5-1.3)	38.5	0.1	(0.0-0.1)	0.1	(0.0-0.2)	0.2	(0.1-0.4)	96.2
Africa and Middle East																	
Israel	1.0	(0.6-1.5)	0.0	5.0	(1.8-13.9)	3.2	(1.4-7.4)	1.9	(0.9-4.0)	10.0	0.4	(0.2-1.0)	0.5	(0.3-1.1)	0.6	(0.3-1.2)	3.7
Lebanon	0.5	(0.2-1.2)	2.5	1.9	(0.2-20.0)	1.3	(0.1-11.3)	0.8	(0.1-6.9)	2.6	0.1	(0.0-0.3)	0.2	(0.1-0.4)	0.7	(0.3-1.5)	28.7
Nigeria	1.1	(0.4-3.3)	0.0	0.6	(0.1-3.0)	0.1	(0.0-0.7)	0.3	(0.1-1.9)	7.9	0.3	(0.2-0.7)	0.6	(0.2-2.0)	0.5	(0.2-1.5)	10.1
Asia and the Pacific																	
Japan	0.9	(0.5-1.6)	0.3	5.6	(1.8-17.2)	1.7	(0.8-3.7)	1.3	(0.5-3.3)	14.1	0.1	(0.0-0.1)	0.1	(0.1-0.2)	0.4	(0.2-1.0)	63.5
PR China	1.0	(0.4-2.3)	0.0	4.6	(1.4-15.6)	2.1	(0.9-5.0)	1.0	-	6.7	0.3	(0.1-0.9)	0.2	(0.0-1.0)	0.7	(0.2-2.4)	8.3
Oceania																	
New Zealand	1.3	(1.1-1.5)	8.6	4.3	(2.9-6.3)	2.4	(1.7-3.4)	1.7	(1.3-2.4)	68.8	0.1	(0.1-0.1)	0.1	(0.1-0.2)	0.2	(0.2-0.2)	461.0

Table 5 Socio-demographic predictors of lifetime treatment contact for any mood disorder

Country	Sex			Cohort (age at interview)							Age of onset						
	Female		χ^2	Age 18-34		Age 35-49		Age 50-64		χ^2	Early		Early-average		Late-average		χ^2
	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	
The Americas																	
Colombia	1.5	(0.9-2.3)	2.7	3.2	(1.3-7.7)	1.7	(1.0-3.2)	1.0	-	6.7	0.2	(0.1-0.4)	0.3	(0.2-0.7)	0.8	(0.5-1.3)	33.6
Mexico	1.6	(1.0-2.4)	4.6	2.1	(0.9-4.9)	1.7	(0.8-3.3)	1.0	-	3.1	0.3	(0.2-0.6)	0.5	(0.2-0.9)	0.8	(0.4-1.6)	25.1
USA	1.3	(1.1-1.5)	10.2	4.4	(3.2-6.1)	3.1	(2.3-4.1)	1.9	(1.4-2.6)	115.5	0.2	(0.1-0.3)	0.3	(0.2-0.3)	0.4	(0.3-0.6)	176.7
Europe																	
Belgium[a]	1.4	(0.9-2.1)	2.5	3.9	(1.2-12.5)	3.9	(1.5-10.5)	1.7	(0.7-4.0)	14.5	0.2	(0.1-0.6)	0.4	(0.2-0.9)	0.6	(0.4-0.9)	14.2
France[a]	1.3	(0.9-1.8)	2.9	5.7	(3.1-10.5)	4.4	(2.4-8.0)	2.0	(1.1-3.5)	44.3	0.2	(0.1-0.4)	0.4	(0.2-0.8)	0.6	(0.3-1.2)	54.9
Germany[a]	1.2	(0.8-2.0)	0.9	1.9	(0.7-5.1)	1.2	(0.6-2.8)	1.2	(0.5-2.5)	6.3	0.3	(0.1-0.6)	0.5	(0.2-1.0)	1.1	(0.5-2.1)	22.5
Italy[a]	1.4	(0.9-2.0)	2.6	1.4	(0.7-2.8)	1.6	(0.8-2.9)	1.1	(0.6-2.1)	2.8	0.4	(0.2-0.8)	0.8	(0.4-1.6)	0.8	(0.4-1.4)	15.7
Netherlands[a]	0.9	(0.7-1.3)	0.1	3.9	(1.7-8.9)	2.7	(1.6-4.4)	1.0	-	18.5	0.1	(0.0-0.3)	0.3	(0.1-0.6)	0.5	(0.3-0.8)	27.1
Spain[a]	1.2	(0.8-1.8)	1.1	1.9	(0.9-3.8)	2.7	(1.4-5.1)	1.3	(0.8-2.1)	11.3	0.4	(0.2-0.8)	0.4	(0.2-0.9)	0.7	(0.4-1.2)	8.3
Africa and Middle East																	
Israel	1.1	(0.9-1.5)	0.7	5.4	(2.9-10.0)	4.0	(2.3-6.8)	2.3	(1.4-3.7)	30.9	0.3	(0.2-0.6)	0.4	(0.2-0.6)	0.6	(0.4-1.0)	20.8
Lebanon	1.1	(0.7-1.8)	0.2	13.8	(2.3-83.0)	8.8	(1.5-51.1)	5.0	(0.8-30.8)	13.4	0.4	(0.2-0.8)	0.2	(0.1-0.7)	0.7	(0.3-1.4)	10.6
Nigeria	1.4	(0.5-3.6)	0.5	2.7	(0.3-22.4)	0.5	(0.1-3.7)	1.0	-	6.8	2.6	(0.2-33.6)	1.2	(0.0-31.2)	3.3	(0.3-41.1)	3.0
Asia and the Pacific																	
Japan	1.6	(0.8-3.5)	1.7	3.9	(1.1-13.4)	2.0	(0.7-6.2)	1.5	(0.6-4.2)	5.0	0.2	(0.0-0.6)	0.5	(0.2-1.3)	0.8	(0.4-1.9)	9.8
PR China	0.8	(0.2-3.6)	0.1	0.7	(0.2-2.9)	0.4	(0.1-1.3)	1.0	-	2.4	0.5	(0.1-3.3)	0.4	(0.1-1.7)	0.5	(0.1-1.9)	2.3
Oceania																	
New Zealand	1.4	(1.2-1.6)	16.9	3.7	(2.7-5.2)	2.3	(1.7-3.1)	1.6	(1.2-2.2)	84.1	0.2	(0.2-0.3)	0.3	(0.3-0.4)	0.6	(0.5-0.8)	205.6

[a]Used major depressive episode instead of any mood disorder

Even when events were recalled, they may have been dated inaccurately. The most common form of dating error is telescoping, in which past experiences are recalled as having occurred more recently than they actually did. Questions that focused memory search and bounded recall uncertainty were embedded in WMH surveys to help respon-

Table 6 Socio-demographic predictors of lifetime treatment contact for any substance use disorder

Country	Sex		χ^2	Cohort (age at interview)						χ^2	Age of onset						χ^2
	Female			Age 18-34		Age 35-49		Age 50-64			Early		Early-average		Late-average		
	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	
The Americas																	
Colombia	0.8	(0.3-2.5)	0.1	9.1	(1.6-51.0)	5.3	(1.0-28.2)	1.0	-	6.7	0.2	(0.0-0.9)	0.4	(0.1-2.1)	0.2	(0.0-0.9)	7.9
Mexico	2.8	(0.8-9.5)	2.9	3.6	(0.7-18.1)	0.8	(0.2-2.9)	1.0	-	8.0	0.8	(0.2-3.6)	1.3	(0.3-5.7)	1.7	(0.5-5.5)	2.0
USA[a]	1.2	(0.8-1.6)	1.0	3.4	(1.7-6.8)	1.7	(0.9-3.1)	1.3	(0.7-2.3)	18.2	0.6	(0.4-0.8)	0.6	(0.4-0.8)	0.6	(0.4-0.8)	14.4
Europe																	
Belgium	0.7	(0.1-8.3)	0.1	35.9	(1.1-1163.4)	35.9	(1.1-1163.4)	35.9	(1.1-1163.4)	4.5	0.1	(0.0-0.2)	0.1	(0.0-0.2)	0.1	(0.0-0.2)	25.7
France	0.8	(0.2-3.2)	0.2	0.2	(0.0-3.2)	0.7	(0.1-4.8)	1.0	-	2.1	0.4	(0.1-2.6)	0.4	(0.1-2.6)	0.4	(0.1-2.6)	1.0
Germany	1.4	(0.4-5.3)	0.2	4.3	(0.5-37.5)	4.3	(0.5-37.5)	1.0	-	1.9	0.2	(0.0-1.2)	0.1	(0.0-0.3)	1.0	(0.3-3.1)	12.6
Italy	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b
Netherlands	0.6	(0.1-2.9)	0.4	1.4	(0.1-24.1)	1.7	(0.1-19.6)	0.4	(0.0-5.1)	2.1	0.0	(0.0-0.7)	0.2	(0.0-1.1)	0.1	(0.0-0.3)	18.3
Spain	1.5	(0.1-41.2)	0.1	8.1	(1.4-46.8)	1.0	-	1.0	-	5.8	0.0	(0.0-0.1)	0.0	(0.0-0.7)	0.2	(0.0-1.7)	16.0
Africa and Middle East																	
Israel	0.2	(0.0-1.3)	2.8	9.5	(1.8-49.7)	3.8	(1.0-14.7)	1.0	-	7.3	0.7	(0.2-2.8)	0.3	(0.1-1.5)	2.2	(0.7-7.6)	8.5
Lebanon[a]	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b	_b
Nigeria[a]	_b	_b	_b	4.7	(0.6-34.6)	2.3	(0.7-7.9)	1.0	-	3.5	0.1	(0.0-1.7)	0.5	(0.1-3.0)	0.2	(0.0-2.8)	3.1
Asia and the Pacific																	
Japan[a]	0.4	(0.1-3.3)	0.7	3.6	(0.1-203.0)	0.3	(0.1-0.7)	0.3	(0.1-0.7)	9.5	0.2	(0.0-5.3)	0.4	(0.0-3.1)	1.3	(0.3-5.2)	2.5
PR China[a]	0.4	(0.0-6.4)	0.5	1.8	(0.2-20.1)	0.5	(0.1-2.0)	1.0	-	3.0	0.5	(0.1-3.1)	0.5	(0.1-3.1)	0.8	(0.1-5.9)	0.6
Oceania																	
New Zealand	1.3	(1.0-1.7)	4.6	5.6	(2.8-11.0)	3.1	(1.6-5.9)	1.8	(0.9-3.5)	47.1	0.4	(0.3-0.6)	0.3	(0.2-0.4)	0.4	(0.3-0.5)	63.2

[a]Assessed in the part II sample
[b]Disorder was omitted due to insufficient lifetime cases (n<30)

dents recall age of onset and age of initial treatment contact (23,30). However, to the extent these efforts were not successful, it is again likely that delays in initial treatment seeking have been underestimated.

Our examinations of contacts with providers in the prior year have revealed that many fail to result in adequate treatment (4). To the extent that initial contacts with providers also fail to result in any treatment or in adequate regimens, we have underestimated failure and delays in receipt of *effective* treatment. Furthermore, we were only able to study predictors of failure to make treatment contact that could be retrospectively dated. We also limited potential predictors to variables for which *a priori* hypotheses have been raised regarding treatment delay or failure, to reduce the possibility of chance findings (14-16).

Finally, we cannot be certain that the failures and delays in initial treatment seeking observed here are of clinical or public health significance. Alternatively, those who failed to make prompt initial contacts may have largely had self-limiting or less serious disorders (31). However, our earlier analyses of the U.S. data revealed that even those with severe and impairing disorders have substantial delays in initial treatment contact (16). Furthermore, the preclinical, epidemiologic, and trial data reviewed above suggest that even milder disorders, if left untreated, lead to greater severity, additional psychiatric comorbidity, and negative social and occupational functioning (8-10).

Keeping these limitations in mind, our results reveal two major problems in the initial treatment-seeking process for mental disorders that are occurring throughout the world. On one hand, many lifetime cases never make any treatment contact for their disorders, particularly in developing countries, where the financial and structural barriers to accessing mental health services are most formidable (3). Failure to seek help also appears to be greatest for conditions with low perceived needs for treatment, such as substance use disorders, for which over half of lifetime cases failed to make any treatment contact in the majority of countries (13,32).

Even among cases that do eventually seek help, a second major source of unmet need for mental health care is the pervasive delays before treatment contacts are made. The typical delays observed here last for years or even decades after disorder onset. Initial treatment contacts appear to be fastest for mood disorders, perhaps because these disorders have been targeted in some countries by educational campaigns, primary care quality improvement programs, and treatment advances (33-35). On the other hand, the longer delays for anxiety disorders may be due to the earlier age of onset of some conditions (e.g., phobias), fewer associated impairments, and even fear of providers or treatments involving social interactions (e.g., talking therapies, group settings, waiting rooms) (4,13,36).

Women have been shown in prior research to be faster than men at translating nonspecific feelings of distress into conscious recognition that they have emotional problems, perhaps explaining the significantly higher rates of initial

treatment contact by women in some countries (37). More recent cohorts were also significantly more likely to make eventual treatment contact, perhaps suggesting a positive outcome of programs recently attempted in some countries to destigmatize and increase awareness of mental illness, of screening and outreach initiatives, of the introduction and direct-to-consumer promotion of new treatments, and of expansion of insurance programs (1,33-35,38-42). Consistent with prior research (14-16), early-onset disorders were associated with lower probabilities of initial treatment contact in most countries. One explanation for this finding may be that minors need the help of parents or other adults to seek treatment, and recognition is often low among these adults unless symptoms are severe (43,44). In addition, child and adolescent-onset mental disorders may be associated with normalization of symptoms or development of coping strategies (e.g., social withdrawal in social phobias) that interfere with help-seeking later in life. Finally, lack of accessible child mental health services may also be an important issue in many countries.

While these results document the failure and delay in initial treatment seeking for mental disorders that are occurring worldwide, additional research will be needed to clarify what policy makers can concretely do to address them at the local and national levels. At the local level, it is critical to identify whether and through what specific programs long periods of untreated mental illness can be reduced. Cost-efficient interventions that can be applied in schools, clinics, or health care systems, consisting of aggressive outreach and prompt treatment of new cases, are just emerging. Long-term intervention trials currently in the field will shed light on the extent to which these model programs prevent subsequent negative clinical, social, educational, and occupational outcomes (45,46). Programs of public education, school or primary care-based screening, disease management, or coordination and referral between non-health care and health care professions, may also prove helpful in this regard (34,38,44,47-51).

Furthermore, it will be critical to clarify what can be done at the national level to minimize failure and delay in initial treatment contact. General and mental health care policies, delivery system designs, and levels or mechanisms of financing mental health services may have enormous impacts on the timeliness of treatment seeking. Unfortunately, policy makers currently lack rigorous data on these impacts, including whether impacts are positive, negative, as intended, or inadvertent. Linking data such as those of the WHO Project Atlas on existing policies, delivery systems, and financing of mental health care, to WMH survey data on failure and delay in initial treatment, may offer a novel way to shed light on these impacts and help guide future policy decisions (3,17).

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References

1. Demyttenaere K, Bruffaerts R, Posada-Villa J et al. Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. JAMA 2004;291:2581-90.
2. Murray CJL, Lopez AD. The global burden of disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020. Cambridge: Harvard University Press, 1996.
3. Saxena S, Sharan P, Saraceno B. Budget and financing of mental health services: baseline information on 89 countries from WHO's Project Atlas. J Ment Health Policy Econ 2003;6:135-43.
4. Wang PS, Aguilar-Gaxiola E, Alonso J et al. Worldwide use of mental health services for anxiety, mood, and substance disorders: results from 17 countries in the WHO World Mental Health (WMH) Surveys. Lancet 2007;370:841-50.
5. World Health Organization. The world health report 2001. Mental health: new understanding, new hope. Geneva: World Health Organization, 2001.
6. Kohn R, Saxena S, Levav I et al. The treatment gap in mental health care. Bull World Health Organ 2004;82:858-66.
7. Post RM, Weiss SR. Sensitization and kindling phenomena in mood, anxiety, and obsessive-compulsive disorders: the role of serotonergic mechanisms in illness progression. Biol Psychiatry 1998;44:193-206.
8. Kessler RC, Berglund PA, Foster CL et al. Social consequences of psychiatric disorders, II: teenage parenthood. Am J Psychiatry 1997; 154:1405-11.
9. Kessler RC, Foster CL, Saunders WB et al. Social consequences of psychiatric disorders, I: educational attainment. Am J Psychiatry 1995;152:1026-32.
10. Kessler RC, Walters EE, Forthofer MS. The social consequences of psychiatric disorders, III: probability of marital stability. Am J Psychiatry 1998;155:1092-6.
11. Kessler RC, Price RH. Primary prevention of secondary disorders: a proposal and agenda. Am J Commun Psychol 1993;21:607-33.
12. Meltzer HY, Alphs L, Green AI et al. Clozapine treatment for suicidality in schizophrenia: International Suicide Prevention Trial (InterSePT). Arch Gen Psychiatry 2003;60:82-91.
13. Leaf PJ, Livingston MM, Tischler GL et al. Contact with health professionals for the treatment of psychiatric and emotional problems. Med Care 1985;23:1322-37.
14. Christiana JM, Gilman SE, Guardino M et al. Duration between onset and time of obtaining initial treatment among people with anxiety and mood disorders: an international survey of members of mental health patient advocate groups. Psychol Med 2000;30: 693-703.
15. Olfson M, Kessler RC, Berglund PA et al. Psychiatric disorder onset and first treatment contact in the United States and Ontario. Am J Psychiatry 1998;155:1415-22.
16. Wang PS, Berglund PA, Olfson M et al. Delays in initial treatment contact after first onset of a mental disorder. Health Serv Res 2004;39:393-415.
17. Mezzich JE. From financial analysis to policy development in mental health care: the need for broader conceptual models and partnerships. J Ment Health Policy Econ 2003;6:149-50.
18. World Bank. World development report 2004: making services work for poor people. Washington: International Bank for Reconstruction and Development/World Bank, 2003.
19. Kessler RC, Angermeyer M, Anthony JC et al. Lifetime prevalence and age-of-onset distributions of mental disorders in the World Health Organization's World Mental Health Survey Initiative. World Psychiatry 2007;6:168-76.
20. Alonso J, Angermeyer MC, Bernert S et al. Sampling and methods of the European Study of the Epidemiology of Mental Disorders (ESEMeD) project. Acta Psychiatr Scand 2004;420(Suppl.):8-20.
21. Kessler RC, Merikangas KR. The National Comorbidity Survey Replication (NCS-R): background and aims. Int J Methods Psychiatr Res 2004;13:60-8.
22. Kessler RC, Berglund P, Demler O et al. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry 2005;62: 593-602.
23. Kessler RC, Ustun TB. The World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI). Int J Methods Psychiatr Res 2004;13:93-121.
24. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th ed. Washington: American Psychiatric Association, 1994.
25. First MB, Spitzer RL, Gibbon M et al. Structured Clinical Interview for DSM-IV Axis I Disorders, research version, non-patient edition (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute, 2002.
26. Haro JM, Arbabzadeh-Bouchez S, Brugha TS et al. Concordance of the Composite International Diagnostic Interview Version 3.0 (CIDI 3.0) with standardized clinical assessments in the WHO World Mental Health Surveys. Int J Methods Psychiatr Res 2006; 15:167-80.
27. Halli SS, Rao KV. Advanced techniques of population analysis. New York: Plenum, 1992.
28. Efron B. Logistic regression, survival analysis, and the Kaplan-Meier curve. J Am Stat Assoc 1988;83:414-25.
29. Wolter K. Introduction to variance estimation. New York: Springer, 1985.
30. Kessler RC, Wittchen HU, Abelson JM et al. Methodological issues in assessing psychiatric disorder with self-reports. In: Stone AA, Turkkan JS, Bachrach CA et al (eds). The science of self-report: implications for research and practice. Mahwah: Lawrence Erlbaum, 2000:229-35.
31. 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 surveys' estimates. Arch Gen Psychiatry 2002;59:115-23.
32. Mojtabai R, Olfson M, Mechanic D. Perceived need and help-seeking in adults with mood, anxiety, or substance use disorders. Arch Gen Psychiatry 2002;59:77-84.
33. Hirschfeld RMA, Keller MB, Panico S et al. The National Depressive and Manic-Depressive Association consensus statement on the undertreatment of depression. JAMA 1997;277:333-40.
34. Jacobs DG. National depression screening day: educating the public, reaching those in need of treatment and broadening professional understanding. Harv Rev Psychiatry 1995;3:156-9.
35. Olfson M, Marcus SC, Druss B et al. National trends in the outpatient treatment of depression. JAMA 2002;287:203-9.
36. Solomon P, Gordon B. Outpatient compliance of psychiatric emergency room patients by presenting problems. Psychiatr Q 1988; 59:271-83.
37. Kessler RC, Brown RL, Broman CL. Sex differences in psychiatric help-seeking: evidence from four large-scale surveys. J Health Soc Behav 1981;22:49-64.
38. Regier DA, Hirschfeld RM, Goodwin FK et al. The NIMH Depression, Awareness, Recognition, and Treatment program: structure, aim, and scientific basis. Am J Psychiatry 1988;145:1351-7.
39. Ridgely MS, Goldman HH. Mental health insurance. In: Rochefort DA (ed). Handbook on mental health policy in the United States. Westport: Greenwood, 1989:341-61.
40. Rosenthal MB, Berndt ER, Donohue JM et al. Promotion of prescription drugs to consumers. N Engl J Med 2002;346:498-505.
41. Spitzer RL, Kroenke K, Williams JBW et al. Validation and utility of a self-report version of the PRIME-MD: the PHQ Primary Care Study. JAMA 1999;282:1737-44.
42. Sturm R. Tracking changes in behavioral health services: how

have carve-outs changed care? J Behav Health Serv Res 1999;26: 360-71.
43. Janicke DM, Finney JW, Riley AW. Children's health care use: a prospective investigation of factors related to care-seeking. Med Care 2001;39:990-1001.
44. Morrissey-Kane E, Prinz RJ. Engagement in child and adolescent treatment: the role of parental cognitions and attributions. Clin Child Fam Psychol Rev 1999;2:183-98.
45. Beidel DC, Turner SM, Morris TL. Behavioral treatment of childhood social phobia. J Consult Clin Psychol 2000;68:1072-80.
46. MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. Arch Gen Psychiatry 1999;56:1073-86.
47. Aseltine RH Jr, DeMartino R. An outcome evaluation of the SOS Suicide Prevention Program. Am J Publ Health 2004;94:446-51.
48. Carleton RA, Bazzarre T, Drake J et al. Report of the Expert Panel on Awareness and Behavior Change to the Board of Directors, American Heart Association. Circulation 1996;93:1768-72.
49. Connors CK. The Connors Rating Scales: use in clinical assessment, treatment planning and research. In: Maruish M (ed). Use of psychological testing for treatment planning and outcome assessment. Hillsdale: Lawrence Erlbaum, 1994:550-78.
50. Velicer WF, Hughes SL, Fava JL et al. An empirical typology of subjects within stage of change. Addict Behav 1995;20:299-320.
51. Weaver AJ. Has there been a failure to prepare and support parish-based clergy in their role as frontline community mental health workers: a review. J Pastoral Care 1995;49:129-47.

Burnout in psychiatrists

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Psychiatrists as a group are vulnerable to experiencing burnout, more so than other physicians and surgeons. In this paper, various definitions of burnout are reviewed and the tools available for quantifying burnout are compared. The factors that make psychiatry a stressful profession are also examined. These include factors such as patient violence and suicide, limited resources, crowded inpatient wards, changing culture in mental health services, high work demands, poorly defined roles of consultants, responsibility without authority, inability to effect systemic change, conflict between responsibility toward employers vs. toward the patient, and isolation. In order to investigate how exposure to such stressors results in burnout, two theoretical models are examined. Recommendations are also made, on the basis of anecdotal reports, for addressing burnout in psychiatrists.

Key words: Burnout, psychiatrists, stress management, workforce

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Psychiatrists have a stressful life. They use themselves as "tools" in their profession and experience a range of powerful emotions in their clinical work. The doctor-patient relationship in itself evokes emotions such as the need to rescue the patient, a sense of failure and frustration when the patient's illness progresses or does not respond to treatment, feelings of powerlessness against illness and its associated losses, grief, fear of becoming ill oneself, or a desire to separate from and avoid patients to escape these feelings (1). Given the personal nature of the relationship psychiatrists have to develop with their patients, these emotions are likely to be intensified in their context. Psychiatrists are also exposed to external stressors, due to the rapidly changing ways of service delivery, the widening gap between the way they are trained and the way they practice, and the increasingly complex administrative and legal frameworks.

While stressors may originate from a variety of sources and may vary with settings and disciplines, the outcome of chronic exposure to emotional and interpersonal stressors on the job is invariably burnout (2,3). This paper examines the concept and causes of burnout and the relevant interventions as they apply to psychiatrists.

BURNOUT: DEFINITIONS AND MEASURES

The concept of burnout was first introduced by Freudenberger (4). Since then, various definitions have emerged. Kuremyr et al (5) defined burnout as "an experience of physical, emotional and mental exhaustion caused by long-term involvement in situations that are emotionally demanding". Lee and Ashforth (6), referring to Maslach and Jackson's (7) work, defined burnout as a syndrome of emotional exhaustion (tiredness, somatic symptoms, decreased emotional resources, and a feeling that one has nothing left to give to others), depersonalization (developing negative, cynical attitudes and impersonal feelings towards their clients, treating them as objects) and lack of feelings of personal accomplishment (feelings of incompetence, inefficiency and inadequacy). This definition of burnout has been the most widely used in literature.

While burnout may have a negative impact on workforce, patient care and the individual's health, it may also play a protective role. The symptoms of burnout have been hypothesized to appear in order to protect human psyche against further damage in the face of "having no way out". Freudenberger (8) describes depersonalization as a means of protection against further emotional draining or a homeostatic mechanism in an emotionally exhausted worker. Along similar lines, one may argue that emotional exhaustion acts as a "brake" for individuals who may not know how or when to slow down. Negative changes in attitude (reduced work goals, loss of idealism, heightened self interest, increasing emotional detachment from clients) have been described by Benbow (3) as a form of coping.

Standardized and valid instruments have been developed for the measure of burnout. Two are currently popular: the Maslach Burnout Inventory (MBI, 9) and the Burnout Measure (BM, 10). The MBI gives scores on the three subscales or dimensions of depersonalization, emotional exhaustion and lack of personal accomplishment, by determining how people respond to each of 22 statements on a scale of 0-6. The higher the respondents score on depersonalization and emotional exhaustion, the higher their levels of burnout, while the lack of personal accomplishment scale measures in the opposite direction. The inventory has been found to be reliable, valid and easy to administer. The BM contains 21 items (rated on a 7-point frequency scale) grouped into three subscales (assessing physical exhaustion, mental exhaustion, and emotional exhaustion). Unlike the MBI, the items have no explicit association with work and are presented in random order. The subscales have shown good (.80 to .90 range) internal consistency (11), and the total scale has shown a 1-month test-retest reliability of .89 (12). As with the MBI, factor analytical studies suggest that the BM is a unidimensional measure (11). Others have shown a high correlation between the total BM scores and the scores on the MBI emotional exhaustion scale (12).

WHAT CAUSES BURNOUT IN PSYCHIATRISTS

In order to understand psychiatrists' susceptibility to burnout, one needs to examine the factors that make psychiatry a stressful profession. Deahl and Turner (13) identified violence and the fear of violence, limited resources, crowded inpatient wards and an increasing culture of blame creeping into the mental health services as the main sources of stress for psychiatrists. High work demands without adequate resources, poorly defined roles of consultants, responsibility without authority, inability to effect systemic change, conflict between responsibility toward employers vs. toward the patient, isolation of consultants in community mental health teams and lack of feedback were identified as sources of stress by Thompson (14). A qualitative study of mental health professionals in a well-resourced community mental health team including psychiatrists (15) identified administrative demands, lack of resources, work overload, responsibility for patients and relapsing patients as the top five sources of stress. Overt bureaucracy, high workload and the lack of free time were reported as the factors which may either be responsible for premature retirement by specialist psychiatrists or be reasons why juniors would not pursue psychiatry as a career option (16,17). A large survey of psychiatrists identified out of hours or long hours of duty, dealing with difficult and hostile relatives of patients, arranging admissions, paper work, balancing personal and professional lives and managing suicidal or homicidal patients as particularly stressful experiences (18).

It is important to note that not every psychiatrist who is exposed to such stressors for extended periods develops burnout. Holloway et al (19) describe an interactive model that examines the relationship of the external stressors outlined above with mediating factors and stress outcome. To cite their example, "the poorly functioning doctor who lacks appropriate coping mechanisms and ends up working in an impoverished service may well experience more occupational stresses than his or her more successful peer working within a well resourced and professionally rewarding service. Overwhelming personal or professional life events (e.g., a patient homicide) may lead to decompensation of even most resilient and best supplied professional". Positive motivating factors or sources of job satisfaction, such as appreciation for job done well, responsibility for others, personal advancement and salary enhancement, may play an important role in the final outcome of stress exposure.

An inverse relationship between stress and job satisfaction has been reported in lawyers (20), rehabilitation workers (21) and public service employees (22). Surprisingly, such relationship does not appear to exist for psychiatrists: despite experiencing depression and burnout, they can continue to enjoy their work and consistently score high in job satisfaction surveys – a finding reported from the UK (23), Australia (24) and the USA (25,26). One could speculate that psychiatrists as a group are so committed and passionate towards their work that the exhaustion associated with burnout does not dilute their pleasure derived from work. Alternatively, while psychiatrists may be good at picking up changes in their mood state, they may believe that practising psychiatry in an exhausted state is part of their job (27). This peculiarity of psychiatrists as a group renders any model that relies on job satisfaction as a protective factor weak.

A study of psychiatrists and psychiatric residents investigated the relationship between demographic factors, work and leisure activities. Personality was assessed by the Munich Personality Test (MPT) and burnout with the Tedium Measure (TM) (28). Psychiatric residents reported significantly higher scores on TM and neuroticism, but lower scores on frustration tolerance on MPT. The study found that neuroticism alone explained a substantial proportion of the total TM variance. Work-related variables turned out to be of a small importance only, whereas no influence could be demonstrated for different leisure activities. Another study (29) reported that, as a group, psychiatrists differed significantly on various personality measures from physicians in other disciplines. They scored higher than physicians and surgeons on items of neuroticism, openness and agreeableness, but lower on conscientiousness. Even though psychiatrists reported less clinical work demands, they reported higher work-related emotional exhaustion and severe depression than physicians and surgeons. These findings imply that the very personality characteristics that attract people towards pursuing psychiatry as a career may also render them sensitive to stressors.

Another recent paper (30) examined the interaction between four sets of factors proposed to be responsible for burnout: predisposing, precipitating, perpetuating and protective. Many of the factors that were recognized as external, internal and mediating in Holloway et al's model (19) were encompassed in the above four "P" model, which also identified some systemic factors responsible for burnout, raising the possibility that reducing stress through these systemic issues could reduce burnout in psychiatrists.

The above study (30) pointed out that psychiatrists may be predisposed to burnout due to their personality traits, which make them prone to internalize their stressful experiences. Their training experience may also play a significant role in the causation of stress and burnout: psychiatric trainees are more closely involved with people's personal difficulties than trainees in other disciplines, and often labour feelings of self-doubt, fear, and fatigue (31). Psychiatrists are trained in long-term verbal interventions, but they are invariably employed to deliver short-term and mainly biological treatment modalities (32). Furthermore, it appears that workload on psychiatrists is set to increase globally due to increasing population, a progressive move to community-based treatment, increasing involvement in administrative roles, increasing standards of practice, greater expectations by doctors to have time for study and relaxation, as well as diminishing numbers of those choosing to go into psychiatry (33-36). In other words, psychiatrists as a group are predisposed to experience stress due to internal and external factors.

Against the background of these predisposing factors, psychiatrists are invariably exposed to triggers that precipitate burnout. Violence perpetrated by patients is widely prevalent in mental health services (37-41) and is widely recognized as stressful for all psychiatrists, irrespective of their level of experience (42). Most psychiatrists experience patient suicide and are invariably adversely affected by it (43,44). On-call duties and dealing with difficult and hostile relatives have also been described as distressing events in psychiatrists' profession (18).

The final appearance of burnout may depend on how one perceives and responds to stressful situations. Factors that affect such appraisal styles (so-called perpetuating factors) are instrumental in determining whether the stress originated at work may or not translate into burnout. Gender plays a significant role in the perception and origin of stress and consequently in the way one responds to stress: women respond to stresses through career dilution and diminution (working part time) and/or by using strategies to limit demands on intimacy (45). Personality traits may also play a significant role in predisposing psychiatrists to experience burnout and in perpetuating the phenomenon once it sets in (46). Certain systemic factors have been identified that contribute to psychiatrists' stress and therefore possibly burnout: they include changes in health service delivery model, clinician management conflicts, and time management and resource issues (47,48).

PROTECTIVE FACTORS AND INTERVENTIONS FOR BURNOUT

There are factors protecting psychiatrists against burnout. Some evidence suggests that lifestyle factors and paying attention to one's non-professional life may have a protective effect (49). Academic work has been reported (50) to be negatively correlated with depersonalization, emotional exhaustion and overall stress, implying that personality traits of people with academic interests may have a protective effect against burnout (51). While adding teaching to clinical commitments may increase workload, work-related stress may in fact decrease as a result and indeed the sense of professional accomplishment may increase (52).

While the above-mentioned factors may have preventive effects against burnout, their utility once burnout sets in remains untested through well-designed studies. It is worth noting that not only intervention studies are lacking for psychiatrists' burnout, but there is a significant dearth of studies across all disciplines. A recent systematic review (53) of resident burnout found that insufficient data prevented drawing conclusions about causal relationships between stressors and burnout or indeed any attempts to identify at risk residents based on socio-demographic or personality factors. A systematic review of stress, burnout and coping found no studies had evaluated the use of stress-management interventions in psychiatrists (47). The review found three intervention studies that had used samples of "mental health professionals" including psychiatrists. However, for reasons identified above, it might not be appropriate to lump psychiatrists with other mental health professionals when it comes to either stressors or responses to stressors including burnout. In the absence of any well-designed interventional studies, one may have to look at anecdotal reports. Holloway et al (19) listed interventions focusing on the individual (such as social skills training, stress management interventions, social support and time management) and on the organization (defining role and job characteristics, improving interpersonal relationships, encouraging decentralization in the organizational structure and improving the physical environment of work place). The authors emphasized the importance of formal support through regular feedback and appraisal of psychiatrists' performance, which need to occur even in the absence of any identified problem.

CONCLUSIONS

Burnout is a serious consequence of chronic exposure to work-related stressors. As a group, psychiatrists are at a high risk of experiencing burnout, due to external factors such as work environment, internal factors such as personality and appraisal styles, and mediating factors such as support and resilience. The onset of burnout can be seen as a consequence of the interaction between predisposing, precipitating, perpetuating and protective factors. While factors that are protective against burnout and therefore may have a preventive role have been identified, there is a lack of studies evaluating the efficacy of interventions once burnout has set in. Anecdotal evidence suggests that support through peers, organization or family/friends may be effective against established burnout.

References

1. Meier DE, Back AL, Morrison RS. The inner life of physicians and care of the seriously ill. JAMA 2001;286:3007-14.
2. Farber BA. Introduction: a critical perspective on burnout. In: Farber BA (ed). Stress and burnout in the human service professions. New York: Pergamon, 1983:1-20.
3. Benbow S. Burnout: current knowledge and relevance to old age psychiatry. Int J Geriatr Psychiatry 1998;13:520-6.
4. Freudenberger HJ. Staff burnout. J Soc Issues 1974;30:159-65.
5. Kuremyr D, Kihlgren M, Norberg A et al. Emotional experiences, empathy and burnout among staff caring for demented patients at a collective living unit and a nursing home. J Adv Nurs 1994;19: 670-9.
6. Lee RT, Ashforth BE. On the meaning of Maslach's three dimensions of burnout. J Appl Psychol 1990;75:743-7.
7. Maslach C, Jackson SE. The measurement of experienced burnout. J Occup Behav 1981;2:99-113.
8. Freudenberger HJ. Burnout: contemporary issues, trends and concerns. In: Farber BA (ed). Stress and burnout in the human service professions. New York: Pergamon, 1983:23-8.
9. Maslach C, Jackson SE, Leiter MP. Maslach Burnout Inventory Manual, 3rd ed. Palo Alto: Consulting Psychologist Press, 1996.

10. Pines A, Aronson E. Career burnout: causes and cures. New York: Free Press, 1988.
11. Corcoran K. Measuring burnout: an updated reliability and convergent validity study. In: Crandall R, Perrewe PL (eds). Occupational stress: a handbook. Washington: Taylor and Francis, 1995:263-8.
12. Pines AP. On burnout and buffering effects of social support. In: Farber BA (ed). Stress and burnout in the human service professions. New York: Pergamon, 1988:155-74.
13. Deahl M, Turner T. General psychiatry in no-man's land. Br J Psychiatry 1997;171:6-8.
14. Thompson C. The mental state we are in: morale and psychiatry. Psychiatr Bull 1998;22:405-9.
15. Reid Y, Johnson S, Morant N et al. Explanations for stress and satisfaction in mental health professionals: a qualitative study. Soc Psychiatry Psychiatr Epidemiol 1999;34:301-8.
16. Kendell RE, Pearce A. Consultant psychiatrists who retired prematurely in 1995 and 1996. Psychiatr Bull 1997;21:741-5.
17. Mears A, Kendall T, Katona C et al. Career intentions in psychiatric trainees and consultants. London: Royal College of Psychiatrists, 2002.
18. Rathod S, Roy L, Ramsay M et al. A survey of stress in psychiatrists working in the Wessex Region. Psychiatr Bull 2000;24:133-6.
19. Holloway F, Szmukler G, Carson J. Support systems. 1. Introduction. Advances in Psychiatric Treatment 2000;6:226-35.
20. Jackson SE, Turner J, Brief AP. Burnout among public service lawyers. Unpublished manuscript, University of Michigan, Ann Arbor, 1985.
21. Riggar TF, Godley SH, Hafer M. Burnout and job satisfaction in rehabilitation administrators and direct service providers. Rehabil Counsel Bull 1984;27:151-60.
22. Zedeck S, Maslach C, Mosier K et al. Affective response to work and quality of family life: employee and spouse perspectives. J Soc Behav Personal 1988;3:135-57.
23. Prosser D, Johnson S, Kuipers E et al. Mental health,'burnout' and job satisfaction among hospital and community-based mental health staff. Br J Psychiatry 1996;69:334-7.
24. Rey JM, Walter G, Giuffrida M. Australian psychiatrists today: proud of their profession but stressed and apprehensive about the future. Aust N Zeal J Psychiatry 2004;38:105-10.
25. Snibbe JR, Radcliffe T, Weisberger C et al. Burnout among primary care physicians and mental health professionals in a managed health care setting. Psychol Rep 1989;65:775-80.
26. Vaccaro JV, Clark GH Jr. A profile of community mental health centre psychiatrists: results of a national survey. Commun Ment Health J 1987;23:282-9.
27. Firth-Cozens J, Greenhalgh J. Doctors' perceptions of the links between stress and lowered clinical care. Soc Sci Med 1997;44:1017-22.
28. Amstutz MC, Neuenschwander M, Modestin J. Burnout in psychiatric physicians. Results of an empirical study. Psychiatr Prax 2001;28:163-7.
29. Deary IJ, Agius RM, Sadler A. Personality and stress in consultant psychiatrists. Int J Soc Psychiatry 1996;42:112-23.
30. Kumar S, Hatcher S, Huggard P. Burnout in psychiatrists: an aetiological model. Int J Psychiatry Med 2005;35:405-16.
31. Hoop JG. Hidden ethical dilemmas in psychiatric residency training: the psychiatry resident as dual agent. Acad Psychiatry 2004; 28:183-9.
32. Hafner H. Psychiatry as a profession. Nervenarzt 2002;73:33-40.
33. Brockington I, Mumford D. Recruitment into psychiatry. Br J Psychiatry 2002;180:307-12.
34. Goldman W. Is there a shortage of psychiatrists? Psychiatr Serv 2001;52:1587-9.
35. Scully JH, Wilk JE. Selected characteristics and data of psychiatrists in the United States, 2001-2002. Acad Psychiatry 2003;27:247-51.
36. Kennedy P, Griffiths H. General psychiatrists discovering new roles for a new era and removing work stress. Br J Psychiatry 2001;179: 283-5.
37. Haller RM, Deluty RH. Assaults on staff by psychiatric inpatients. A critical review. Br J Psychiatry 1988;152:174-9.
38. Hubschmidt T. The prosecution of violent psychiatric inpatients; one respectable intervention. Psychiatr Prax 1996;23:26-8.
39. Drinkwater J. Violence in psychiatric hospitals. In: Feldman P (ed). Developments in the study of criminal behaviour, Vol. 2. Chichester: Wiley, 1982:111-30.
40. Hodgkinson PE, McIvor L, Phillips M. Patient assaults on staff in a psychiatric hospital: a two year retrospective study. Med Sci Law 1985;25:288-94.
41. Rice ME, Harris GT, Varney GW et al. Violence in institutions. Toronto: Hogrefe and Huber, 1989.
42. Poster EC. A multinational study of psychiatric nursing staffs' beliefs and concerns about work safety and patient assault. Arch Psychiatr Nurs 1996;10:365-73.
43. Guthrie E, Tattan T, Williams E et al. Sources of stress, psychological distress and burnout in psychiatrists: comparison of junior doctors, senior registrars and consultants. Psychiatr Bull 1999;23:207-12.
44. Alexander DA, Klein ST, Gray NM et al. Suicide by patients: questionnaire study of its effect on consultant psychiatrists. Br Med J 2000;320:1571-4.
45. Cartwright LK. Role montage: life patterns of professional women. J Am Med Women Assoc 1987;42:142-3.
46. Naisberg-Fennig S, Fennig S, Keinan G et al. Personality characteristics and proneness to burnout: a study among psychiatrists. Stress Med 1991;7:201-5.
47. Fothergill A, Edwards D, Burnard P. Stress, burnout, coping and stress management in psychiatrists: findings from a systematic review. Int J Soc Psychiatry 2004;50:54-65.
48. Snyder TG, Kumar S. Who do I serve? An experiential perspective of problems in retaining psychiatrists in New Zealand. Australasian Psychiatry 2004;12:401-3.
49. Garfinkel PE, Bagby RM, Schuller DR et al. Predictors of success and satisfaction in the practice of psychiatry: a preliminary follow-up study. Can J Psychiatry 2001;46:835-40.
50. Clark GH, Vaccaro JV. Burnout among CMHC psychiatrists and the struggle to survive. Hosp Commun Psychiatry 1987;38:843-7.
51. Agius RM, Blenkin H, Deary IJ et al. Survey of perceived stress and work demands of consultant doctors. Occup Environ Med 1996; 53:217-24.
52. Rutter H, Herzberg J, Paice E. Stress in doctors and dentists who teach. Med Educ 2002;36:543-9.
53. Thomas NK. Resident burnout. JAMA 2004;292:2880-9.

News from the WPA Secretariat

JOHN COX
WPA Secretary General

The institutional life of WPA is at times so frenetic that it is difficult to know what to highlight in this brief report. However, I am now over three-quarters through a six-year term of office and, consistent with the recommendations in the Manual of Procedures, I am beginning to reflect on what can yet be achieved, and indeed to plan for a smooth hand over to my successor. WPA is therefore, I believe, about to reap the benefits of its wise decision to have a Permanent Secretariat to ensure greater continuity between triennia.

When good things happen, it is important for them to be noticed and not submerged in the hurly-burly of daily activity. So here is some good news:

- The Jamaica Psychiatric Society and the Libyan Association of Psychiatry, Neurology and Neurosurgery are much welcomed as new *ad hoc* Member Societies, and the World Fellowship for Schizophrenia and Allied Disorders is a new *ad hoc* Affiliated Association.
- WPA News has changed its format and is now more colourful and substantial. Each region of the world has been featured in four recent issues, giving prominence to the huge amount of good work being done by the 18 Zonal Representatives.
- The Secretariat in Geneva is now staffed by our increasingly experienced administrators: Anna Engstrom from Sweden and Pamela Atiase from Switzerland/Ghana. The excellent suite of offices is to be further extended by our helpful hosts (Geneva University Hospitals) to include an additional office, cloakroom and kitchenette.
- The recent WPA Regional Meetings in Lima, Nairobi and Seoul have been characterised by strong regional commitment. The African Association of Psychiatrists and Allied Professionals (AAPAP) masterminded our first WPA Regional Meeting in sub-Saharan Africa. The meeting probably relaunched the African Journal of Psychiatry, and the commitment and vitality (including rhythm and dance) of the region were truly infectious.
- Your Secretary General was an invited speaker at a smaller co-sponsored meeting in Beirut. The presentations from Iraqi colleagues were poignant, and their links with psychiatrists in the area and across the world were tangibly appreciated.
- The new Section on Psychiatry in Developing Countries got off to a flying start in Lahore, where the launch of the South Asian Federation of Psychiatric Associations occurred – further evidence of growing regional coordination of WPA, facilitated by the impact of global communication systems but also thriving on direct personal contact.
- During the past year, I have had the privilege to chair the European Task Force, which brings together the WPA, the World Health Organization (Europe), the European Union of Medical Specialists (UEMS), as well as the Association of European Psychiatrists (AEP). The growing confidence of European psychiatry was visible at the leaders meeting in Madrid, despite large differences in human resource. The core debate about the role of psychiatrists in public mental health continued and the need for more specific training recognised; yet rightly Prof. Maj pointed out that there is a risk of overlooking our central responsibilities to prevent and manage mental disorders.
- The prime task of the Secretariat is to be a communication channel for this world network of member associations. It is our intention to improve further this process by the website management moving to Geneva, with a substantial debt to the pioneering work of Roger Montenegro in Buenos Aires.
- The WPA General Survey will be distributed in Autumn and provide the various WPA components with a vital opportunity to express their opinions about what WPA does well, as well as where and how improvements to our organisation can occur. In particular, we will seek opinions about the usefulness of paper mailings, and it is likely that the content of all present paper mailings will in future be on the website.
- In addition to this institutional work, I am grateful to colleagues across the world, who challenge and sustain my persisting academic and clinical interest in perinatal and transcultural psychiatry as well as medical ethics. There is a myth that WPA thinking is dominated by psychopharmacology. This subject is of course important, but the emphasis on explanatory pluralism in WPA is what I have found so refreshing in much of the work that we undertake.
- The new Section on Perinatal Psychiatry and Infant Mental Health, as well as the new Institutional Programme on Parental and Infant Mental Health (co-chaired with Sam Tyano), are both launched. They will provide a much-needed focus for the scientific work in this field, which so often is overlooked by maternal child health and children's services.

WPA at its best is like a scaffold that provides a structure within which Member Societies (small and large) can thrive, so that knowledge and clinical experience are exchanged.

Over the next twelve months, I hope that the Secretariat in Geneva will increasingly facilitate this work and that the most up-to-date communication systems will soon be in place. In this way our organisation, which has limited financial resources and yet ambitious values, will become stronger, with more social as well as financial capital. The challenges for the organisation are considerable, but I do not detect any faltering of the will to succeed.

It is much to be hoped that the larger Member Societies as stiffeners, including my own College, as well as those with smaller budgets, will increasingly recognise the opportunity to facilitate international educational exchange, and promote strategies for conflict resolution and the provision of psychiatric expertise to communities traumatised by violence and war.

Free and low-cost access to online WPA publications

Helen Herrman
WPA Secretary for Publications

Offering psychiatrists free and low-cost access to WPA publications is an important feature of the WPA publications program. Yet the psychiatrists and institutions eligible for these benefits are not always aware that they are available or how to obtain them. This article describes the needs for free access, especially in low- and middle-income countries, the ways it is provided, and some developments in the rapidly changing world of publishing.

The timely and effective dissemination of research results is essential for high standards and innovation in research and clinical services. Often in low- and middle-income countries only the free abstracts are available to clinicians. The abstracts tend to be short, poorly written and sometimes they are misleading (1). A 10/90 divide in the publication of internationally accessible mental health literature is also evident and remains unchanged. Recent studies reveal that most articles in high impact journals come from richer countries (2-4). A search of the Web of Science database of the Institute for Scientific Information (ISI) from 1992 to 2001 found that low- and middle-income countries (N=152) contributed only 6%, high-income countries (N=54) 94%, and 14 leading high-income countries 90% of internationally accessible mental health research (5). A joint statement from editors of scientific journals publishing mental health research and the World Health Organization (WHO) offered some steps to correct these imbalances (6).

The WPA publications program is committed to help correct this imbalance and improve information exchange between psychiatrists, scientists, other professionals, policy-makers, politicians and the general public, irrespective of their country of residence and its wealth. The program has two main goals. The first is to disseminate information on significant clinical, service and research developments, reaching as many psychiatrists as possible in the various countries of the world. The second is to encourage contributions from psychiatrists of all regions of the world, in the form of research papers, commentaries or reports on mental health or significant service innovations. These goals are being pursued in several ways: through *World Psychiatry* (the WPA official journal); through several series of books; in joint activities with the WPA educational program and scientific sections; and in collaborative activities with other organizations. Free and low-cost access to WPA publications is an important feature of the program. It is attained in two main ways.

First is the publication of *World Psychiatry* in its print and electronic versions. The journal is published in three editions (English, Spanish and Chinese) and is now indexed in PubMed, Current Contents/Behavioural Sciences, Current Contents/Clinical Medicine, and the Science Citation Index. It regularly publishes contributions from all regions of the world. It is alone recommended as an explicitly international journal to researchers and advocates of international mental health (2). *World Psychiatry* is sent free of charge to more than 32,000 psychiatrists in 121 countries. The journal is also produced in an electronic version, with free access through PubMed (www.pubmedcentral.nih.gov/tocrender.fcgi?journal=297&action=archive), the WPA website and the websites of several of WPA Member Societies and Sections. *World Psychiatry* is now an important means by which contributors from all countries can obtain wide dissemination for high quality research reports and inclusion in international data indexes. The free access contributes to the journal's potential to increase the global prominence of research from low- and middle-income countries.

Collaboration with the publishers of the WPA books provides the second significant means of free and low-cost access to WPA publications. WPA has a productive partnership with Wiley-Blackwell, resulting in the *Evidence and Experience* series as well as other books. Information on the series and print purchase is available through the series page (eu.wiley.com/WileyCDA/Section/id-303180.html). The latest book from Wiley-WPA, released in March 2007, is entitled *The Mental Health of Children and Adolescents: An Area of Global Neglect*. It is edited by H. Remschmidt, B. Nurcombe, M.L. Belfer, N. Sartorius and A. Okasha, and presents the highlights of the WPA Presidential Programme on Child Mental Health, constituting a global call to action for mental health workers and policy makers.

Wiley-Blackwell offers 15% discount to individual members of WPA Member Societies on the purchase price of all its books (see www.Wiley.com). All the WPA books are also available online through Wiley InterScience Online-Books™, with free abstracts and subscription access to the chapters. Psychiatrists in the wealthiest countries have access through individual or institutional accounts. An important benefit is available to those working in low- and middle-income countries: from the time of first publication of Wiley-Blackwell medical books, including all the WPA titles, people in low-income countries have free access through Health InterNetwork Access to Research Initiative (HINARI); those in middle-income countries pay a small amount. HINARI provides free or very low-cost online access to thousands of major journals and books in biomedical and related social sciences to local, non-profit institutions in developing countries. HINARI was launched in 2002 and is one of the world's largest collections of biomedical and health literature. There are presently 2000 institutions in 106 countries registered for HINARI. There are 113 countries eligible for HINARI. The WHO manages the HINARI website, with the support of the Yale University Library.

Other recent news is the finalising of an agreement between Wiley-Blackwell

and WPA to publish electronically through Wiley InterScience the major series called *Anthologies of Classic Psychiatry Texts*, directed by D. Moussaoui. This series collects classic texts never previously published in English, with bio-bibliographical notes on their authors. Permission to transfer copyright has been obtained by Wiley-Blackwell for the Anthologies of French, Italian and Spanish texts. The *German Anthology of Psychiatric Texts,* edited by H. Sass, has been published recently, and the same agreement will be sought by Wiley-Blackwell. These books will be available online (free and low-cost to those eligible as above). Wiley-Blackwell is also considering making these available via print on demand so that individuals can purchase the print edition, should they choose. The ready availability in perpetuity of these texts is a major resource for psychiatrists and scholars.

Rowman & Littlefield published in July 2006 *Psychiatry and Sexual Health: An Integrative Approach*, edited by J.E. Mezzich and R. Hernandez on behalf of the WPA Educational Program on Sexual Health. A volume on diagnosis and cultural formulation is in production. The terms negotiated for these books include agreement to publish them on the WPA website for free access 18 months after publication date.

The world of publishing is undergoing radical change. As publications move from the print world to a digital world, new possibilities are likely to arise to improve access to and dissemination of scientific knowledge (7). M. Rondon, member of the WPA Operational Committee for Publications, passed on to me a web news announcement that CrossRef (www.crossref.org), a nonprofit publishers' membership association aiming to improve access to published scholarship through collaborative technologies, has recently agreed with the International Network for the Availability of Scientific Publications, INASP (www.inasp.info) to include journals from developing countries within its linking network. This partnership will contribute to the INASP mission to enable worldwide access to information, and to its work with publishers in developing and transitional countries to improve the quality and visibility of their publications. Wiley is a founder member of CrossRef. WPA will aim to ensure, through its partnerships with Wiley, WHO and other organizations, that psychiatrists and their patients in all countries obtain the benefits of these advances. We need to trust at the same time that progress is as rapid in reducing the digital divide as it is in the advance of digital publishing.

References

1. Short R. Peer review will become "the job of the many, not the select few". Br Med J 2007;334:30.
2. Patel V, Kim R-I. Contribution of low- and middle-income countries to research published in leading general psychiatry journals, 2002-2004. Br J Psychiatry 2007;190: 77-8.
3. Patel V, Sumathipala A. International representation in psychiatric literature: survey of six leading journals. Br J Psychiatry 2001; 178:406-9.
4. White J, Patel V., Herrman H. Australian and New Zealand contribution to international mental health: a survey of published research. World Psychiatry 2007;6:49-53.
5. Saxena S, Paraje G., Sharan P et al. The 10/90 divide in mental health research: trends over a 10-year period. Br J Psychiatry 2006;188:81-2.
6. Saxena S, Sharan P, Saraceno B. Research for Change: the role of scientific journals publishing mental health research. World Psychiatry 2004;3:66-72.
7. Watson R. EC to promote open access publishing. Br Med J 2007;334:389.

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