Correspondence

EDITED BY MATTHEW HOTOPF

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Culture-specific psychiatric illness?

It is depressing that an editorial in a major psychiatric journal can still maintain that “there is no solid evidence for a real difference in the prevalence of common psychiatric disorders across cultures” (Cheng, 2001). Cheng collapses the socioculturally determined understandings that patients bring to bear on their active appraisal of their predicament and on their expressions of distress and help-seeking to the term “illness behaviour”. The (Western) psychiatrist is to see through this mere packaging to the psychopathology within, which he knows to be universal and the ‘real’ problem. Cheng goes on to assert that disturbed people in “less-developed” societies present somatically because of their “limited knowledge of mental disorders”. There is a distinct echo here of the imperial era, when it was pressed upon indigenous people that there were different types of knowledge and that theirs was second-rate. Sociocultural and sociopolitical phenomena were framed in European terms and the responsible pursuit of traditional values was regarded as evidence of backwardness (Summerfield, 1999).

All of psychiatry is culture-bound, not just a few syndromes in the DSM or ICD: even presentations by patients with organic disorders are embedded in particular ‘lifeworlds’ and local forms of knowledge. Western psychiatry is but one among many ethno-psychiatries. Cheng commits what Kleinman (1987) called a category fallacy: the assumption that because phenomena can be identified in different social settings, they mean the same thing in those settings.

The World Health Organization is falling into the same trap in its claims that ‘depression’ is a worldwide epidemic that within 20 years will be second only to cardiovascular disease as the world’s most debilitating illness. The implication of such medicalisation is to deflect attention away from what millions of people might cite as the basis of their suffering, for example, poverty. In whose interests, apart from the pharmaceutical industry’s, can this be?

We need a psychiatry that recognises the limitations of a technical approach and sees acknowledgement of sociocultural and political contexts as an ethical obligation (Bracken & Thomas, 2001). If Cheng were to see this as a challenge to the whole project – to (Western) psychiatry as a global enterprise propagating supposedly universal and morally neutral facts – then so be it.


D. Summerfield CASCAID, South London and Maudsley NHS Trust, 307 Borough High Street, London SE1 1JF, UK

Andrew Cheng’s contribution (2001) to the debate on the universality of cultural particularity of psychopathology follows the conventional distinction between the pathogenic form of the illness, presumed to be biological, and its pathoplastic content of psychological or social origin (Littlewood, 1996). In his rephrasing, content is merely the “subjective complaint” or “illness behaviour”, form the “objective symptoms”. He then dissect such culture-specific patterns as koro into the ‘real’ illness (panic attacks) and the “false belief” apparently found in people of “low intelligence” with “limited knowledge of mental disorders”, thus proving his case.

His procedure is an act of faith in the possibility (and usefulness) of this Kantian distinction, which has been an article of psychiatric belief since Kraelelin and Birnbaum (Littlewood, 1990). While possibly of some utility for the major psychoses where we may trace some biological aetiology, it seems bizarre to assume that we will find universality in all patterns of psychiatric interest. Eating disorders, multiple personality disorder, overdosing, shoplifting, agoraphobia, school refusal, to mention some Western patterns alone: each is constructed by context and meaning as it is constructed by biological difference. Could we consider school refusal as a universal pattern in the absence of elementary schools in certain societies? What would be left here without social context? What then our analogues of school refusal?

To assert that the business of psychiatry is only the biological (and why should that presume the universal?) is to restrict our discipline to veterinary science. To ignore meanings as potentially causal is to offer an etiolated psychopathology, one presumed to be ‘scientific’ in advance (Kleinman, 1988). To offer a general model of all psychopathology with fixed relations between the social and the biological is certainly non-empirical, and only potentially redeemed if we then exclude the social a priori from any potential patterns. To search for universality is double-sided laudable: to presume it is not.


R. Littlewood Royal Free and University College London Medical School, Department of Psychiatry and Behavioural Sciences, Wolfson Building, 48 Riding House Street, London W1N 8AA, UK

Author’s reply: Littlewood states, “In his rephrasing, content is merely the ‘subjective complaint’ or ‘illness behaviour’, form the ‘objective symptoms’”. This is a misunderstanding of what I have tried to emphasise in my editorial. One of the major points in my work is that the patient’s subjective
complaint belongs to ‘illness behaviour’, which is different from ‘objective symptoms’ assessed by psychiatrists, preferably using a standardised procedure.

Littlewood mentions the Western patterns of eating disorders, multiple personality disorder, overdose, shoplifting, agoraphobia and school refusal. Many of these, if not all, are also found in non-Western societies (e.g., see Kleinman & Lin, 1981). Furthermore, school refusal is not a formal diagnosis in either the ICD–10 or the DSM–IV; rather, it is a behavioural problem possibly with underlying ‘etic’ psychopathology (depression, separation anxiety, phobia, learning disorders and so forth) and socio-environmental factors. In any society, primitive or modern, there are certain forms of teaching activity not run by modern school institutions. Presumably, the same refusal to attend these various forms of ‘school’ exists, with similar underlying psychiatric and socio-environmental factors. The ways of this refusal and the context of the socio-environmental factors are likely to be ‘emic’. For effective management of school refusal, both the underlying potential etic psychopathology and the emic illness behaviour and socio-environmental factors must be carefully examined. This is an alternative example of what I intended to elaborate using the example of koro.

The long-standing debate over etic/emic and semantic issues in cross-cultural psychiatry is unlikely to be satisfactorily resolved in the near future. However, it is believed that the development of standardised clinical interviews with emphasis on cross-cultural equivalence at the level of symptoms (e.g., Cheng et al, 2001) helps to avoid the so-called “category fallacy” (Kleinman, 1987).

It should be stressed that the under-reporting of psychological symptoms by interviewees from developing nations that I mentioned in my editorial does not mean that these people do not have, or cannot differentiate, emotions. People are people, and the very low rate of reporting of psychological symptoms to doctors by people in developing countries may be due to greater social stigma towards mental illness, their lack of knowledge about mental illness and a much less psychologically oriented medical practice. More studies into this area are needed, and I believe that anthropologically oriented researchers can make a great contribution to this endeavour.

The etic/emic approach to psychopathology does not imply that psychiatry is confined only to biology. The emic patho-

plastic shaping and illness behaviour closely associated with different sociocultural settings are equally important in psychiatry and require culture-specific approaches in combination with biological treatment. After all, mental disorders are believed to be the product of gene/environment interaction (Cheng & Cooper, 2001).


A.T.A. Cheng Institute of Biomedical Sciences, Academia Sinica, Taipei 11529, Taiwan

Cross-cultural psychiatric interviews and research instruments

We read Andrew Cheng’s (2001) editorial with much interest. We strongly agree that the development of cross-culturally comparable diagnostic interviews is a pressing need.

In a recent survey in our unit in Sri Lanka of 43 patients presenting with depressive disorder, one-third of these on presentation made a subjective complaint of a “burning sensation of the body” (literal translation) and related secondary distress and denied having most of the core depressive symptoms although the symptom manifestation was of a depressive disorder. Thus, finding semantic or psycholinguistic equivalence for psychiatric symptoms across cultures will be a challenging, albeit necessary, exercise.

We believe that the lack of valid diagnostic tools is an important factor in the limited capacity for psychiatric research in developing countries, which in turn contributes to the underrepresentation of such research in high-impact journals noted by Patel & Sumathipala (2001).

A case in point is that in Sri Lanka the only validated psychiatric rating scales in the native languages are the Mini-Mental State Examination (MMSE) and the General Health Questionnaire (GHQ–30). Efforts at validating the Hospital Anxiety and Depression (HAD) scale (D. de Silva, personal communication, 2001) in Sinhala (the language of the majority) show that the sensitivity and specificity of such an instrument is low. This is noteworthy considering the fact that locally developed diagnostic instruments may not find ready acceptance in high-impact journals.


K. A. L. A. Kuruppuarachchi, S. S. Williams Faculty of Medicine, University of Kelaniya, PO Box 6, Thalagolla Road, Ragama, Sri Lanka

Mental and physical illness

The editorial by Kendall (2001) independently reflects the view of Baker & Menken (2001) that it is time to abandon the term ‘mental illness’. All three authors emphasise that an important reason for so doing is that the term is stigmatising and underlining of the care and treatment of millions of psychiatric (Kendell) and neurological patients (Baker & Menken). Interestingly, Kendall suggests that the term ‘psychiatric illness’ is more acceptable, whereas Baker & Menken propose instead ‘brain illness’. The former seems to replace the mind by the psyche and the latter by the brain.

Like Kendall, I have reviewed the historical processes that have led to the evolution and divergence of psychiatry and neurology as separate disciplines with all the ensuing confusing theoretical and practical uncertainties and complications for professionals and patients alike, including stigma (Reynolds, 1990). Modern neuroscience, which has demonstrated how brain function is profoundly influenced by psychological and social as well as biological factors, has opened the way for resolving some of these uncertainties and divisions. I share the view that one way forward is to build practical bridges between neurology and psychiatry (Reynolds & Trimble, 1989). For example, it does not make sense for neurologists and psychiatrists quite separately to tackle the problem of stigma towards brain and mental illnesses without
some dialogue or coordination, especially as neurologists sometimes blame the stigma of brain disease on misunderstandings created by inappropriate application of the term 'mental'.

I do not, however, share the possibly unrealistic and impractical view that the term 'mental illness' should be abandoned. Brain/mind issues have been debated by professionals, philosophers, patients and the public for centuries, and this will continue for some time to come. It is reminiscent of proposals to abolish the word 'epilepsy' because this neurological condition is so stigmatised. Similar suggestions have been made in the past for the words 'cancer' and 'leprosy', which together with 'epilepsy' were three great unmentionables for much of the 20th century (Reynolds, 2000).

Stigma results from ignorance, misunderstanding, fear and prejudice, and the way to combat it is by education and raising public awareness. Rather than abandon the word 'epilepsy' the International League Against Epilepsy (professional), the International Bureau for Epilepsy (patients/public) and the World Health Organization (political) have jointly initiated a global campaign to bring epilepsy 'out of the shadows' (Reynolds, 2000).


E. H. Reynolds Institute of Epileptology, Weston Education Centre, King's College, Denmark Hill Campus, Cutchcombe Road, London SE5 9PJ, UK

Kendall (2001) begins his editorial on the distinction between mental and physical illness by quoting with approval Lady Mary Wortley Montagu's comment that "madness is as much a corporeal distemper as the gout or asthma". This suggests that he might be a physicalist, that is an advocate of the view that all facts about mind and mentality are physical facts, but at no point does he say this explicitly. He is critical of Cartesian dualism – without saying exactly why.

Kendall then makes a proposal of his own: "In reality, neither minds nor bodies develop illnesses. Only people (or, in a wider context, organisms) do so, and when they do both mind and body, psyche and soma, are usually involved". But he does not explain how the individual person, the mind and the body are supposed to be related to one another and how this would heal the Cartesian split, nor does he offer any arguments in favour of this suggestion. If illnesses can be attributed only to people and not to minds or bodies, then we might expect Kendall to want to talk only of illnesses in general, and not of two different types of illness, as he continues to do in this editorial. Astonishingly, in the very next sentence he appears to be endorsing Cartesian dualism, the view he has already rejected: "Pain, the most characteristic feature of so-called bodily illness, is a purely psychological phenomenon". If pain is a "purely psychological phenomenon", then it can have no physical component. So there is at least one purely psychological, non-physical phenomenon in the world – a fact that is incompatible with physicalism. But, apparently oblivious of this, Kendall again dismisses Cartesian dualism when he observes that "the differences between mental and physical illnesses... are quantitative rather than qualitative", a remark that suggests physicalism again. Just how could differences between mental and physical illnesses be quantified? How can phenomenal consciousness or 'raw feelings' (i.e. what it is like to have certain mental experiences, such as pain or pleasure, visual hallucinations or paranoid delusions) differ only quantitatively and not qualitatively from physical phenomena?

Kendall seems to teeter between Cartesian dualism and physicalism and he presents no arguments for an alternative to dualism that might lend support to his proposed changes in terminology.


P. Crichton Department of Psychological Medicine, Royal Marsden Hospital, Fulham Road, London SW3 6JJ, UK

Kendall's (2001) editorial made two mistakes in its reasoning, which led to an unhelpful conclusion. One cannot say that mental and physical illness should be conflated because, irrespective of the balance, mental and physical symptoms are expressed in both. This is insisting that differences in degree are not differences at all. There are indeed many disorders that have both mental and physical expressions. However, to claim that anxiety-related chest pain and myocardial infarction are both physical disorders is to confute precisely what we wish to distinguish, even if anxiety can cause both. We contrast the terms 'mental' and 'physical' because the contrast says what we mean, and we have good reason for meaning it. As Kendall himself points out, no alternative has been found.

Proposing that disturbances in bodily function are necessary for psychiatric disorder does not imply that psychiatric disorders are physical disorders. Consider a computer virus. It may exist as a series of electrical states in a computer, a set of statements in a computer language, even a series of thoughts in someone's head, so its existence is not dependent on any physical object. None the less, it may disrupt a computer's function despite there being no physical fault in the machine. It is generally accepted that such arguments show that mental states might themselves be functions (Heil, 1998), and so purely functional psychiatric disorders are quite possible.

These mistakes lead Kendall to suggest that stigma might be reduced if all psychiatric disorders were to be regarded as physical. This makes mental illness literally unspeakable. But not speaking of something truly implies an attitude towards it of denial, shame and horror, not acceptance. The concept of mental health and its promotion is currently competing successfully with 'madness' in popular culture. By falsely declaring 'mental' to be meaningless, the editorial threatens this progress. It may also consign those of our patients who are not sufficiently biological in their pathology to that therapeutic underclass, the 'worried well'.


D. M. Foreman Department of Psychiatry, Keele University, Thornborough Drive, Hartshill, Stoke-on-Trent ST4 7QB, UK

Author's reply: I agree with much of what Dr Reynolds says and with Baker &
Menken's view, to which he refers. I also, like him, look forward to the time when psychiatrists and neurologists speak the same language. Both will need to change a good deal for this to be possible, but increasing understanding of the cerebral substrate of emotions and cognition will eventually provide a powerful stimulus to both specialties. I was not, though, suggesting that we should talk of psychiatric illnesses instead of mental illnesses because I prefer Greek to Latin derivations. The term 'mental illness' implies a disorder of the mind. By substituting 'psychiatric illness' I wished to imply simply that these are disorders which, if they come to specialist attention, are normally treated by psychiatrists. I should emphasise, too, that my objections to the term 'physical illness' are almost as great as to 'mental illness'. Both encourage doctors and patients alike to make inappropriate and damaging assumptions and to ignore the role of psychological and social influences across the whole spectrum of illness. That is why I do not think it is appropriate simply to combine mental and neurological disorders as 'brain disorders'.

In reply to Dr Crichton, I did not quote Lady Mary Wortley Montagu with either approval or disapproval, but simply to illustrate the fact that in the mid-18th century it was still the accepted view that madness was no different from other diseases. More importantly, Dr Crichton is confusing the difference between mental and physical events and what are misleadingly called mental and physical illnesses. There are indeed still many mysteries about the relationship between mental and physical (cerebral) events and no unanimity among either philosophers or neuroscientists about the nature of that relationship (although Descartes' original 'substance dualism' has passed into history). But this, although important, is irrelevant to my argument that there is no fundamental or qualitative difference between the heterogeneous collections of illnesses we currently distinguish as physical and mental. Both physical and mental phenomena are conspicuous in both – as aetiological factors, as features of the illness itself and as influences on outcome. And pain is indeed a purely subjective phenomenon, even though there are good reasons for assuming that it usually, perhaps always, has physical (cerebral) concomitants.

In reply to Dr Foreman I can only say that he should have read my editorial rather more carefully. I did not argue that psychiatric disorders are physical disorders. Rather, I drew attention to the extensive evidence of somatic abnormalities in almost all common mental disorders and to the lack of any characteristic features of either the symptomatology or the aetiology of so-called mental illnesses that reliably distinguished them from physical illnesses (and vice versa). Nor did I declare "mental to be meaningless", or argue that there are no important differences between mental and physical illnesses. My argument was that "the differences between mental and physical illnesses, striking though some of them are, are quantitative rather than qualitative, differences of emphasis rather than fundamental differences, and no more profound than the differences between diseases of the circulatory system and those of the digestive system, or between kidney diseases and skin diseases". And far from wanting mental illnesses to be regarded as physical illnesses, I argued that both terms are misleading. Finally, I did not say that "no alternative has been found" for the term mental illness. On the contrary, I suggested that "we should talk of psychiatric illnesses or disorders" instead. Nor was this merely a suggestion. The most recent edition of the Companion to Psychiatric Studies, which I co-edited (Kendell & Zealley, 1993), deliberately refers to psychiatric illnesses or disorders rather than to mental disorders throughout its 950 pages, and explains the reasons for doing so.


R. E. Kendell
Department of Psychiatry
University of Edinburgh, 3 West Castle Road, Edinburgh EH10 5AT, UK

General psychiatry and suicide prevention

I am grateful to Eagles et al (2001) for their recent editorial on the role of psychiatrists in the prediction and prevention of suicide. I am a member of the Royal Australian and New Zealand College of Psychiatrists' working group on suicide, and we are currently deliberating how to vote on a proposal to disband our group and hand responsibilities back to the College – after all, suicide is part of mental health.

Eagles et al start with how traumatic it is for psychiatrists when their patients commit suicide. Is this not a bit self-indulgent? Our surgical colleagues dealing in trauma frequently contend with the death of ordinary people in the operating theatre. More importantly, the authors do not even mention the suffering of family members affected by suicide.

In their conclusions Eagles et al focus on four points: first, they advocate less epidemiology and more multi-centre treatment trials with suicidal people; second, they advocate more support for traumatised psychiatrists; third, they make a plea to politicians and health service planners to realise what a difficult task suicide prevention is for us; fourth, they note that prediction is a very limited art (I entirely agree), but claim that "all of our patients are at increased risk of suicide". Taking their first and last points together, perhaps if they were more aware of epidemiological data they would realise Blair-West et al's (1999) calculations have refuted the suggestion that 15% of people with depression eventually kill themselves: for this to be true, the annual number of suicides would have to be several times greater than it currently is. They recalculated the lifetime risk of suicide in people with depression as 3.4% with a lifetime risk of 7% for males and 1% for females.

As regards traumatised psychiatrists, I would simply say that all traumatised workers deserve support and that support should be in proportion to their trauma. I suspect that psychiatrists would rank well down the list, below fire, ambulance and police officers and many other medical workers – not to mention contemporary farmers in the UK!

The point relating to re-educating politicians and health planners about our limitations in influencing suicide rates has some validity. However, prevention is much more than that which might result from prediction. Nowhere in the editorial did I find any mention of basic public health concepts such as primary, secondary and tertiary prevention (Silverman & Maris, 1995). If general psychiatrists have not woken up to the fact that this is the basis of national suicide prevention strategies, I think I will have to vote in favour of retaining our local specialist-interest suicide prevention group.


C. Cantor PO Box 1216, Noosa Heads, Queensland 4567, Australia

Authors’ reply: Dr Cantor seems to have misconstrued the intended scope and content of our editorial. We did not set out to comment upon national suicide prevention strategies but, as the title suggested, we sought to discuss the role of psychiatrists specifically in attempting to prevent suicide among the patients we treat. We agree wholeheartedly that any strategy that focused exclusively on psychiatrists as the agents of suicide prevention would be absurd. Indeed, this was one of the main points we were trying to make.

Dr Cantor thinks that our ignorance of the epidemiological data makes us state that “all of our patients are at increased risk of suicide”. This is in fact an epidemiological statement, which he interprets concretely. The fact that the lifetime risk of suicide among people with recurrent depression has been adjusted downwards actually renders statistical prediction of a rare event even more difficult. Largely for this reason we cannot predict which of our patients will commit suicide or when they might do so, and thus we must regard the entire cohort of patients we see as collectively at increased risk of dying by suicide and view their clinical management accordingly.

We take issue that it is “self-indulgent” to suggest that psychiatrists find the suicide of their patients to be traumatic. We know this to be the case from our survey in Scotland (Alexander et al., 2000) and from other, more qualitative accounts (Hendin et al., 2000). While valid comparisons among professional groups are difficult to make accurately, we in Aberdeen are more than a little interested in the impact of ‘critical incidents’ on colleagues in the caring and emergency services (e.g. Alexander, 1993; Alexander & Klein, 2001). One crucial difference between psychiatrists on the one hand and other doctors and other professionals on the other is the issue of blame. While, as we try to point out, it is often illogical for psychiatrists to take responsibility for the suicide of our patients, we frequently do, and this distinguishes it from the deaths that other professionals encounter. Finally, presumably we would wish our patients (and their families) to feel cared for and understood. Surely, as professionals in psychiatric services, we should accord the same opportunities to each other.


J. M. Eagles, D. A. Alexander Royal Cornhill Hospital, Aberdeen AB25 2ZH, UK

Psychiatric training in developing countries

Jacob (2001) successfully highlights the problems of community care of people with mental disorders in developing countries. Both he and the *Journal* are to be commended for addressing the mental health issues of the vast populations of such countries, a topic generally overlooked in the literature. The author is right to point out that most programmes have failed to deliver and that the success of local model projects has not been repeated at a national level. From personal experience as both a trainee and a trainer and from discussion with colleagues in a similar situation, I believe the most important reason for this is the inappropriate training of psychiatrists in developing countries.

The suitability of the training in developed countries for psychiatrists who will ultimately work in developing countries is increasingly being questioned (Mubbashar & Humayun, 1999), but questions have rarely been asked about the training in their own countries. Unfortunately, the training in most developing countries is still based on models of psychiatric services and theories derived from developed nations. An obvious example is the concept of community psychiatry. This concept and its enactment, derived from the history of modern Western psychiatry, cannot be applied in developing countries (Farooq & Minhas, 2001). Young psychiatrists from developing nations who trained in this model of community psychiatry will find the realities of psychiatric services in their own countries totally different from what they have learnt in training.

Moreover, the training in many developing countries remains narrowly focused on acquiring clinical skills. This is despite the fact that a World Health Organization expert committee recommended long ago that trained mental health professionals should devote “only part of their working hours” to the clinical care of patients (World Health Organization, 1975). As Jacob points out, the realities of mental health care in the community in developing countries demand that training is broad-based and equips the psychiatrist to work effectively with other disciplines, particularly primary care. This, however, is rarely the case in many developing countries.

The training of psychiatrists in developing countries needs a total paradigm shift to address the problems raised by Jacob. Both the mental health professionals and the policy makers need to address this as a priority. If they do not, most of the mental health initiatives in these countries will fail.


S. Farooq Department of Psychiatry, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan

Vascular risk factors for stroke and depression

Stewart *et al. (2001)* present an important study of the association between the vascular risk factors for stroke and depression. Although the non-participation rates and levels of physical morbidity were high in the sample, they did not find any association between risk factors for vascular disease and level of depression in the older
adult population (aged 55–75 years) studied. We have prospectively studied 45 stroke patients (aged 26 to 65 years) for psychiatric morbidity. The most common disorder was depression (in 78% of the patients), followed by generalised disorder (in 17%). Younger age, physical disability (resulting in occupational and social dysfunction) and past history of stroke were strongly correlated with depression. Vascular disease has been found to be associated with a more prolonged duration of depression (Hickie & Scott, 1998), but in our sample 52% of the patients with depression recovered within 3–6 months of treatment. However, two patients who were unemployed when they were disabled by stroke did have depression of prolonged duration. Uncontrolled hypertension (moderate to severe) was associated with the presence of generalised anxiety disorder. The role of medication (especially beta-blockers, calcium channel blockers and sedatives) in producing depression is an important variable and could not be ruled out in six patients. Although laterality of brain lesion (i.e. left hemispheric lesion) and risk of depression have been reported (Robinson & Price, 1982), the subject remains controversial and we did not find any such association. A detailed prospective study on a larger sample of patients from all age groups and different socio-demographic backgrounds is needed to establish the association of depression with various demographic and vascular risk factors for stroke.


M. S. Bhatia, R. Chandra Department of Psychiatry, University College of Medical Sciences, Dilshad Garden, Delhi 110095, India

Somatoform disorders: a topic for education

Bass et al. (2001) believe that somatoform disorders are ignored by psychiatrists and health service planners because of the nature of diagnostic practice, a current preoccupation with only “serious mental illness”, limited experience of patients with medically unexplained symptoms in general hospital settings, and stigma. They do not mention whether they have found an increasing fear of litigation to be another contributing factor. Currently, it appears to play a part in delaying referral to psychological services while the patient is exhaustively investigated for any physical pathology. Any comment they might make regarding this practice would be of interest.

Certainly, as they mention, a lack of training of non-psychiatric practitioners in this area contributes greatly to non-referral within the general hospital setting. We would, however, dispute their comment that psychiatrists working in this area find that patients with somatoform disorders “comprise between one-third and one-half of all referrals to the liaison psychiatry service”. A review carried out several years ago of the nature of referrals to the consultation–liaison services of two general hospitals in Dublin City (Cullinan et al., 1997) suggests a much smaller number of such referrals. Over a 6-month period 491 patients were referred and patients with diagnoses falling into categories F40–F48 of ICD–10 (neurotic, stress-related and somatoform disorders) accounted for only 12% of referrals in one hospital and 15% in the other. As a significant number of the patients in these categories were suffering from adjustment disorders, the numbers diagnosed with somatoform disorders, formed an even smaller percentage of all referrals.

It is worth noting that these were the diagnostic categories provided by the psychiatrists who assessed these patients. The reason for the referrals given by the medical/surgical teams was “no organic cause for symptoms found” in just 1.7% of cases in one hospital and 10.2% in the other. Perhaps somatoform disorders are even more neglected than previously thought? Education of both psychiatric and non-psychiatric personnel regarding these disorders would appear to be in need of urgent review.


R. Cullinan Department of Psychiatry, St Camillus Unit, St Vincent’s Hospital, Donnybrook, Dublin 4, Ireland

We read with interest Bass et al.’s (2001) review on somatoform disorders. Although the authors usefully pointed out that these disorders are common and cause severe disability, we were dismayed to find that, ironically, they neglected to mention one of the more common somatoform disorders: body dysmorphic disorder (BDD; also known as dysmorphophobia). A distressing or impairing preoccupation with an imagined or slight defect in appearance, BDD has reported rates in the community of 0.7–2.3% (Phillips, 2001). People with this disorder commonly present to psychiatrists, dermatologists, cosmetic surgeons and other physicians (Phillips & Castle, 2001).

Body dysmorphic disorder causes severe distress and marked impairment in functioning (Veale et al., 1996; Phillips, 2001). A high proportion of patients require hospitalisation, become housebound and/or attempt suicide. Completed suicide has been reported in both psychiatric and dermatology settings. Mental-health-related quality of life is poorer than that reported for patients with depression, obsessive–compulsive disorder and a variety of physical illnesses, including recent myocardial infarction and type II diabetes.

Like the other somatoform disorders, BDD is often neglected by psychiatrists. The diagnosis is usually missed in mental health settings (Phillips & Castle, 2001). This is unfortunate, because a majority of these patients request and receive non-psychiatric treatments, such as dermatological treatment and surgery, which are usually ineffective. Many patients consult numerous physicians, request extensive work-ups, and pressure dermatologists and surgeons to provide unsuitable and ineffective remedies. Some patients, in desperation, even perform their own surgery. As one dermatologist stated, “The author knows of no more difficult patients to treat than those with body dysmorphic disorder” (Cotterill, 1996).

The good news is that emerging data indicate that a majority of these patients can be successfully treated with selective serotonin reuptake inhibitors or cognitive–behavioural therapy (Phillips, 2001). It is important that psychiatrists and other physicians screen patients for this disorder so that effective treatment can be provided. Body dysmorphic disorder is a severe psychiatric illness that we cannot afford to neglect.

Dermatology Clinics, 14, 457–463.

Petersfield: Wrightson Biomedical, in press.


K. A. Phillips Butler Hospital, Brown University School of Medicine, Providence, Rhode Island, USA
D. J. Castle Fremantle Hospital, University of Western Australia, PO Box 480, Fremantle, WA 6959, Australia

Advice for authors is premature

In their recent article Patel & Sumathipala (2001) lament the low level of international representation in high-impact psychiatry journals and argue that such a phenomenon is curtailing the development of the psychiatric discipline in both developed and developing countries. Although I agree with the basic argument put forward, some of the advice given to prospective authors is, at best, premature. To be more specific, they explicitly advise authors from countries outside the ‘Euro-American’ group (Western Europe, North America and Australia/New Zealand) to submit their manuscripts to the three high-impact European psychiatric journals (British Journal of Psychiatry, Acta Psychiatrica Scandinavica and Psychological Medicine), rather than to the three high-impact American psychiatric journals (American Journal of Psychiatry, Archives of General Psychiatry and Schizophrenia Bulletin), because the former publish a higher proportion of articles from ‘rest-of-the-world’ (RoW) authors. While this may be so, and indeed their data suggest that it is, does not necessarily follow that such authors will improve their chances of publication by submitting to the three European journals in preference to the three American ones. Such authors should be concerned with differential acceptance rates rather than with the proportion of published papers by RoW authors. Although no acceptance rate data were provided by the three American journals, data on the three European journals indicated a much lower acceptance rate for RoW authors than for Euro-American ones (the fact that the three American journals refused to provide acceptance rate data should not be assumed to indicate that they show an even greater bias). Given these data, it would seem wrong to suggest that RoW authors should favour the three European journals when submitting manuscripts for publication. Such advice should perhaps be reserved until the data are more conclusive.


G. J. Faunce Department of Psychiatry, University of Sydney, NSW 2006, Australia

Chromosome 22q11 deletions and severe learning disability

In a previous study (Murphy et al, 1998) we identified 74 patients thought to be at risk of velo-cardio-facial syndrome (VCFS) from 265 hospitalised individuals with learning disability. We screened these 74 people and found two cases of VCFS, giving a minimum prevalence of 0.7% in the entire sample. Recently, we found a further individual with VCFS among the 191 individuals who were not selected for screening. This gives a revised minimum prevalence estimate of 1.1%.

The patient, a 50-year-old Caucasian female with severe learning disabilities, had no reported complications arising during gestation, delivery or early childhood. Developmental milestones were globally delayed. She presented to the psychiatric services as a teenager with a 12-month history of becoming withdrawn and subdued, having lost some previously learned skills and displaying outbursts of bizarre behaviour and aggression. She was subsequently admitted on several occasions and was definitively admitted as a long-term patient within 2 years of initial presentation, owing to her enduring challenging behaviour. At this time, her mental age was assessed to be between 2 and 4 years and she had not developed any effective speech or signing. She remained in hospital for over 30 years, then was discharged to a staffed house in the community. While in hospital she remained largely quiet, timid and withdrawn. However, there were episodes of prolonged challenging behaviour, during which time she became suspicious, would scream, cry and make unintelligible noises, often accompanied by physical aggression. These episodes were treated with a variety of behavioural modification strategies, augmented with various antipsychotic preparations with partial success. No formal diagnosis of psychotic illness was made and she was therefore not selected for chromosome 22q11 deletion studies as she did not meet screening criteria (Murphy et al, 1998).

In 1999 she was readmitted to the local assessment and treatment unit because of aggression, agitation, deterioration in her skills and bizarre behaviour including ambidexterity, vocalising and gesturing as if responding to abnormal perceptions. It was felt that these symptoms were suggestive of a psychotic disorder and she was successfully treated with an atypical antipsychotic. A provisional diagnosis of schizophrenia was made, although one cannot be categorical in individuals lacking effective communication skills (Royal College of Psychiatrists, 2001). Fluorescence in situ hybridisation (FISH) analysis confirmed a deletion or chromosome 22q11.

In our 1998 study, we concluded that chromosome 22q11 deletions appear to be aetologically significant in a proportion of individuals with idiopathic learning disability, especially in those with mild learning disability and psychosis. The importance of the patient described above is that she has severe rather than mild learning disability and that, despite presenting with seriously challenging behaviour at the time of the study, she was not diagnosed as having a formal psychotic illness.

Although individuals with VCFS are characterised predominantly by borderline or mild learning disability, it is important for clinicians to be aware that occasionally such individuals may present with more severe learning disabilities, as in this case. We suggest that any individual with moderate or severe learning disabilities with a history of bizarre or uncharacteristic challenging behaviour, even in the absence of a formal diagnosis of psychosis, should be referred for chromosome 22q11 deletion studies.


Earliest evidence of post-traumatic stress?

Written evidence of stress reactions to trauma is relatively new, dating back to the 17th century (Trimble, 1985). I would like to present the oldest post-traumatic reaction in recorded history. This historical evidence is based on a cuneiform tablet describing the death in battle of King Urnamma (2111–2094 BC) and its consequences (Fluckiger-Hawker, 1999). This tablet provides the earliest known description of exposure to a traumatic event leading to a post-traumatic reaction. The following paragraphs present the destruction bestowed on Sumer and the people’s reaction to the event:

“From the […] the […] evenly in/on the land. […] struck, the palace(s) was collapsed. […] spread panic rapidly among its Black-headed who dwelt there. […] established its abandoned places in Sumer. In its vast […] cities are destroyed, the people are seized with panic. Evil came upon Ur […]”

(Urnamma A: 1–6. In Fluckiger-Hawker, 1999)

“They weep bitter tears in their broad squares where merriment had reigned. With their bliss (fulness) having come to an end, the people do not sleep soundly.”


These verses describe exposure to a traumatic event, followed by psychiatric symptoms. This evidence gives us a glimpse of a traumatic reaction in antiquity and allows us to compare it to modern reactions. Furthermore, it reveals the core features of post-traumatic stress disorder, which are based on exposure to a traumatic event followed by characteristic symptoms such as sleep disturbances (American Psychiatric Association, 1994).


M. Ben Ezra Department of Psychology, Tel Aviv University, Tel Aviv 69978, Israel

One hundred years ago

Isolating the phthisical insane

The Journal of Mental Science (January, 1900) publishes a paper on the necessity of isolating the phthisical insane read at a recent meeting of the Medico-Psychological Association by Dr. Eric France of Claybury Asylum. The paper shows from the post-mortem records of the London County Asylums at Claybury, Colney Hatch, and Cane Hill that while 67 deaths were certified as due to tuberculosis, active tuberculosis was found post-mortem in 112 cases, this last figure not including 10 doubtful cases. “It will thus be seen,” he says, “that at these three asylums the number of patients dying with active tuberculosis, as compared with those certified as dying from this cause, practically stand in the proportion of two to one.” A number of valuable tables of mortality follow and the conclusion urged is that cases of phthisis in asylums should be isolated in the early stage of the disease before they begin to disseminate the bacillus to the detriment of others. The tuberculin test is now generally admitted to be the most valuable diagnostic measure for indications of early mischief. Of 55 suspected cases injected with tuberculin 45 reacted and 10 did not react. Of the latter five were alive and healthy in July, 1899, the other five died and the post-mortem examinations made disclosed no trace of tubercle. In the discussion which followed the paper Sir William Broadbent, Sir James Crichton Browne, and Professor Clifford Allbutt strongly urged the need of special provision for the isolation and open-air treatment of such cases.

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