Correspondence

EDITED BY KIRIAKOS XENITIDIS and COLIN CAMPBELL

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Going to war always hurts

I was disappointed and saddened by the carelessness of the title ‘Going to war does not have to hurt’ in the June issue of the Journal (Hacker Hughes et al., 2005). One does not even have to mention the considerable number of British casualties in Iraq to realise that this headline is completely ill thought out and a particularly misplaced euphemism that fails to appreciate that war in modern times always kills civilians rather than military personnel. As the historian Norman Davies points out, almost 100% of casualties in modern warfare are civilians and this is no different in Iraq today. To minimise the considerable and well-documented consequences of going to war for Army personnel and to ignore the plight of civilians is, in my opinion, shameful. Health professionals should be very careful not to collude with politicians in minimising the impact of war and armed conflict, because they can easily become a vehicle of such policies.

Hacker Hughes, J., Cameron, F., Eldridge, R., et al. (2005) Going to war does not have to hurt: preliminary findings from the British deployment in Iraq, British Journal of Psychiatry, 186, 536–537.

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Authors’ reply: Dr Lepping has expressed strong views on the plight of the Iraqi civilians who have suffered tragic and devastating casualties in the conflict that has followed the war in Iraq. Our paper was not attempting to belittle their suffering or to make excuses for the political ideologies behind the conflict; rather we examined the mental health of UK military personnel who had been deployed to Iraq in the line of duty.

With the exception of Professor Simon Wessely (who is an unpaid Honorary Civilian Consultant Advisor in Psychiatry to the Director General of the Army Medical Services), all of the authors are either civilian or uniformed members of the Defence Medical Services. As such, it is our duty and privilege (along with our many colleagues) to look after the mental health needs of the servicewomen and men of the UK’s Armed Forces to the best of our ability. It is these professional sailors, soldiers and aviators (both full-time and reservist) who are mobilised by our government to go to war on behalf of the country for whatever purpose. Their going to war is distinct from those civilian inhabitants of war zones who of course do not choose to ‘go to’ war but who inevitably suffer the consequences of warfare and armed conflict.

The effects of war on civilian populations have been extensively investigated and published elsewhere (Horton, 2004; Roberts et al., 2004) and, although continued investigation of the health needs of civilians caught up in war is indeed pressing, our paper concerned itself solely with the mental health of those professional soldiers who are called upon to fight.

The conclusion of the study was that, for a highly prepared elite group of soldiers involved in war fighting in Iraq, there was a positive effect on soldiers’ mental health, at least in the short term. In that context, the title of the short report was, in our opinion, highly appropriate.

Declaration of interest

J.H.H., F.C., R.E. and M.D. are employed by the UK Defence Medical Services. S.W. is an unpaid Honorary Civilian Consultant Advisor in Psychiatry to the Director General of the Army Medical Services.

Traumatic events v. life events: does it really matter?

We read with great interest the paper by Mol et al. (2005). However, we would like to point out some weaknesses. First, ‘serious illness (self)’ was considered a life event rather than a traumatic event. There is a vast literature concerning post-traumatic stress disorder (PTSD) in people with AIDS and cancer. Serious illness definitely meets the DSM-IV criterion A1 for life-threatening situations (Barak et al., 1998).

Second, there is a big problem with Mol et al.’s definition of ‘sudden death’ of loved ones, which ranges from watching a loved one die violently to hearing about the death of a loved one or a close relative. The same is also true for accidents and serious illness. The magnitude of a traumatic event is linked directly to PTSD symptomatology (Sungur & Kaya, 2001). If we were to exclude sudden death and accidents from the traumatic events group we would see a significant difference between the life events group and the traumatic events group, with more symptoms in the latter. This is a crucial point since most people in the traumatic events group reported sudden death or accident as their worst event; they also had a low level of PTSD symptomatology. If Mol et al. had subdivided the sudden death and accident groups according to the magnitude of the event, this would have helped to determine whether the event could be considered a life event or a traumatic event. This is important when dealing with the issue of traumatic grief (Stroebe et al., 2001), which is a combination of PTSD and bereavement. If the participants had undergone normal grieving the sudden death should be considered a life event rather than a traumatic event.

Third, the magnitude of the traumatic event was clearly associated with PTSD
symptomatology, with extremely traumatic situations such as physical and sexual abuse being related to very high PTSD symptomatology scores. However, the number of participants with exposure to such traumatic events was very small ($n=9$ for physical abuse, $n=4$ for sexual abuse and $n=13$ for physical and sexual abuse as a child).

Notwithstanding, the basic message of the paper is important: the line between life events and traumatic events is at best thin, and sometimes nonexistent. The best support for this can be found in the case of the Dutch farmers (Olf et al, 2005) whose cattle were exposed to foot and mouth disease leading to the killing of the herds. This was not a life-threatening event for the farmers, but was a major life event that can easily be considered traumatic.


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**Post-partum depression**

We would like to raise some concerns about the paper by Evans et al (2005), which oversimplifies the aetiology of post-partum depression. Depression in pregnancy and post-partum has been globally linked to psychosocial issues (marital problems, social support, childhood adversity) and pregnancy-related factors, all of which interact with personality (Patel et al, 2002; Dennis & Boyle, 2004). For the findings of the paper to be clinically relevant, it would have been useful to study the relative roles of at least some of these mediating variables, rather than focusing on personality alone.

We also feel that using six items from an interpersonal sensitivity scale for assessing the main explanatory variable is not fully justified. The items chosen measure only some aspects of the self; more-robust measures such as the Dysfunctional Attitude Scale (Weissman, 1979), or the Crandell Cognitions Inventory (Crandell & Chambless, 1986) could have been used to assess self-schemas.

We would also like Evans et al to speculate on why some women developed depression earlier and some later (after 3 years) despite having high negative self-schemas at baseline. Is it possible that self-schema change with experiences such as motherhood, or that support might have mediated the later onset of depression? Also, did women in the higher tertiles for negative self-schema score develop depression earlier?

In the absence of information about important psychosocial variables and factors related to the development of schemas, it is difficult to presume that negative self-schemas are alone sufficient to predict the onset of depression. The inclusion of women who had negative self-schemas but did not develop depression, and repeat assessment of those with negative self-schemas would have also better delineated state versus trait concerns. Finally, it would have been useful to have a control group of non-pregnant women to determine whether personality as a vulnerability factor is unique to pregnancy and the post-partum period.


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Authors’ reply: Drs Chandra and Sudhir appear to have misunderstood the aim of our paper. The paper is not primarily about post-partum depression. We did not aim to study the overall aetiology of post-partum depression nor did we aim to accurately predict post-partum depression from the negative self-schema measure. We did aim to test an important aspect of the cognitive theory of depression, namely whether a measure of negative self-schema is associated with the onset of depression. We found there was an association and that it was equally strong whether the onset was during pregnancy, in the post-partum period or 3 years later. In the main analysis we adjusted for the psychological and socio-economic variables outlined in Table 3.

We agree, as stated in our discussion, that a more detailed questionnaire such as the Dysfunctional Attitude Scale would have provided a more comprehensive measure of self-schema. Furthermore, repeated measures would have allowed comparison with other studies and a test of the stability of these ‘schemas’. It is possible that schemas change with an experience such as motherhood, although theoretically they should be relatively stable. As these were secondary data analyses of an existing data-set, we were limited to the data available to us and these did not include any more-detailed or repeated measures of schemas.

There are clearly multiple factors that influence the onset of depression. The correspondents ask why some women have earlier onset than others. This may well be related to changing support or adverse events, but it was not the aim of our paper to address this question. Rather than speculate, the ALSPAC data-set provides an opportunity to answer this question and many others by undertaking further detailed analyses of those data.

The analyses we presented in Table 4 indicate that the strength of the association between negative self-schema and onset of depression does not diminish with time, so it is unlikely that those in the highest tertiles for depression have onset which is sooner.
This work needs to be replicated in other large longitudinal studies of both non-pregnant women and men.

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Hypertension and depression in late life

The ‘vascular depression’ hypothesis has attracted considerable interest, but its basis is by no means clear. Kaimal & Nair (2005) in their recent correspondence mention vascular comorbidity in late-onset depression, citing a high prevalence rate of hypertension in particular. However, despite reasonably consistent findings of neuroradiological abnormalities associated with late-life depression, there is actually little evidence that hypertension or other ‘traditional’ vascular risk factors are responsible. What evidence there is for higher comorbidity comes from case-control studies comparing people with late-life depression who are known to clinical services with community controls; these studies carry a high risk of selection bias. Studies carried out in community populations have not generally found any associations between hypertension and depression (Kim et al., 2004), even in high-risk samples (Stewart et al., 2001).

The direction of causation between vascular disorders and depression is also unclear (Baldwin, 2005). Evidence for neuroradiological abnormalities associated with depression is derived almost entirely from cross-sectional studies, and there is currently much stronger prospective evidence that depression is a risk factor for vascular disorders than vice versa. The same appears to be the case for sub-syndromal depressive symptoms that may not be recalled or reported in later life and might well have been present in people whose depression is classified as ‘late-onset’. Neuropathological studies of late-life depression do not find the microvascular abnormalities that would be expected if hypertension were responsible, but instead find increased large vessel disease (Thomas et al., 2001), which could equally be a consequence as a cause of depression.

The uncertainty surrounding specific vascular processes in late-life depression is not just of academic interest, since there are important public health implications. Depression is strongly predicted by poor physical health and associated disability and there is little evidence at present to justify distinguishing specific disorders as responsible. The relationship between vascular processes and affective disorder is interesting, but there is a danger that more obvious risk factors for late-life depression (disability, poverty, loneliness) are ignored because they do not fall within the exciting world of organic psychiatry.


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Detection of depression in elderly care home residents

Dr Eisses et al (2005) showed how the training of care home staff can help in detecting depression among elderly care home residents. In view of the high prevalence of depression in this group, these results are to be welcomed. However, I would like to raise a few points about the relevance of this report to care homes in England.

First, 8 out of 23 homes (35%) declined to participate, citing lack of interest or feeling that it was too much work. This matches my own experience as a consultant old age psychiatrist dedicated to providing support to care homes with nursing. I would welcome any suggestions on how to engage homes in training to improve the detection of depression in this vulnerable population.

Second, I was impressed by the stability of the workforce, who had spent on average 9.5 years in the participating homes (the shortest period being 10 months). This is considerably longer than is found within some care homes in England, particularly in the large cities. It may be that training would be less effective when staff tend to have a higher rate of turnover.

Finally, I note that the study excluded residents with dementia who scored less than 15 out of 30 on the Mini-Mental State Examination. This decision is understandable as the scale used, the Geriatric Depression Scale (GDS), is difficult to administer to this group. However, I wonder about the effect on the staff. Training staff to exclude these residents from an assessment of depression could send a false signal that these individuals are somehow immune to depression – which most certainly is not the case. Also this approach probably could not apply in care homes with nursing in England. Whereas in the homes studied the prevalence of dementia was only some 9% of the population, dementia in care homes with nursing is high, with ‘non-EMI’ (elderly mental illness) care homes with nursing having a prevalence of dementia as high as 75% (Macdonald & Carpenter, 2003). We use the Cornell Scale for Depression in Dementia (Alexopoulos et al., 1988) for those people with dementia who can not respond to the questions of the GDS.


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Suicide in India

We read with interest the editorial on suicide prevention from a life course perspective (Gunnell & Lewis, 2005). It offers a broad conceptual overview of the issues related to suicide.

Recent reports from Vellore suggest that suicide rates in India are grossly underreported (Joseph et al, 2003; Aaron et al, 2004; Abraham et al, 2005; Prasad et al, 2005). The average annual suicide rate was 95 per 100 000 for the years 1994–99. The rates in adolescent males and females and those over 55 years were 148, 58 and 189 per 100 000 respectively. Data from India on the contribution of mental illness to suicide rates are limited. A study from Chennai reported a higher risk of mental disorder among people who die by suicide compared with controls (Vijaykumar & Rajkumar, 1999). However, other evidence suggests that chronic stress and precipitating life events rather than severe mental disorders are the major risk factors for suicide. Recent adverse life events, interpersonal stress and relationship difficulties, severe financial distress, the use of alcohol and issues related to gender have all been associated with suicide (Prasad et al, 2005). The depiction of suicide in the mass media is also contributory. Last but not least is the fact that many people seem to accept suicide as an option when faced with extreme mental distress.

Although psychiatric disorders are often associated with suicide in the West and medical models are employed, in developing countries social, economic and cultural factors must be considered when attempting to explain the persistently high rates, the impulsive and stress-related deaths and the apparent widespread ‘acceptability’ of such an option in society. Considering suicide as a single phenomenon or even as a single final pathway might be simplistic. Many diverse approaches, tailored to regional factors, will have to be implemented simultaneously to produce any global reduction in suicide rates.


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Metabolic syndrome and schizophrenia

In his recent editorial Dr Thakore (2005) rightly highlights the importance of the association of the metabolic syndrome and one of its consequences, type 2 diabetes, with schizophrenia. Despite acknowledging that antipsychotic drugs can induce substantial weight gain, he avoids ascribing the metabolic disturbances in schizophrenia to drug-induced obesity. His suggestion that untreated schizophrenia is itself associated with metabolic disturbance is based on a study of 19 people who had substantially greater deposits of intra-abdominal fat than a control group (Ryan et al, 2004). This contrasts with other studies showing that 40 antipsychotic-naïve patients with schizophrenia had no elevation in intra-abdominal fat compared with controls (Zhang et al, 2004) and that 50 did not differ from a control group in terms of body mass index, fasting plasma glucose or insulin (Arranz et al, 2004). In attempting to explain discrepancies in terms of methodological differences, Dr Thakore is wrong to state that the control group of Zhang et al consisted of ‘elderly men’; controls were well matched for age and gender with the patient group.

These larger studies also show that antipsychotic drug treatment is associated with increased intra-abdominal fat (Zhang et al, 2004) and insulin resistance (Arranz et al, 2004), despite negative findings from Ryan et al (2004). The risk of diabetes in schizophrenia is higher in patients receiving olanzapine rather than conventional antipsychotics (Koro et al, 2002); olanzapine is particularly liable to induce weight gain. These and other studies indicate that antipsychotic drug treatment can result in metabolic morbidity. It would thus be misleading, if not dangerous, to imply that obesity resulting from treatment with some antipsychotic drugs is not associated with the development of the metabolic syndrome and type 2 diabetes.

Dr Thakore listed criteria for the metabolic syndrome; these have now been superseded by a more clinically accessible and less stringent definition. The core criterion is central (abdominal) obesity, defined by waist circumference, plus two of four risk factors from elevated triglycerides, reduced high-density lipoprotein cholesterol, raised blood pressure and raised fasting plasma glucose (International Diabetes Federation, 2005).

Declaration of interest

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Author’s reply: Critical methodological differences between the studies of Zhang et al (2004) and Ryan et al (2004) might explain why the two fail to agree. Zhang et al reported a standard deviation of 50 for the age of the controls, indicating that some were elderly; moreover the groups were not matched for gender. This is important as elderly males have higher amounts of intra-abdominal fat (IAF). Lifestyle parameters such as diet, exercise,
smoking and alcohol intake were not measured or indeed compared between the two groups. Furthermore, we are not given any indication as to how an individual was selected for scanning, as not all of the controls and patients recruited had a magnetic resonance imaging (MRI) scan. The authors did not use the same scanning techniques as Seidell et al. (1990), who were among the first to describe the single-slice technique for estimating IAF area. There were large differences in terms of inversion and repetition times. Moreover, the most critical aspect of using a single scan to estimate IAF is that the scan is taken at the level of L4/L5 vertebra, which is best located by a radiological lateral scout and not palpation as performed by Zhang et al. Furthermore, MRI is not a ‘precise and reliable means of determining the two fat measures with better resolution than computed tomography’, as it can erroneously estimate the amount of IAF by 20%.

From a statistical perspective, a one-way ANOVA should have been used to compare any differences between the three groups, as the use of multiple t-tests might have led to a type 1 error. A ‘non-fasting glucose’ level is not a standardised measure and is therefore meaningless. The actual values for fasting glucose decreased in both male and female patients, and fasting insulin levels decreased in females following treatment. Therefore, what Zhang et al. show is that treatment with these two antipsychotics improves the metabolic profile of their patients despite an alleged increase in IAF.

Koro et al. (2002) claim that olanzapine is associated with a higher risk of developing type 2 diabetes than risperidone, but this is difficult to interpret because Table 1 in their paper clearly indicates that the number of new cases of diabetes is greater in patients on risperidone (5.1%) than olanzapine (2.0%). There is little doubt that antipsychotics contribute to the development of type 2 diabetes in patients with schizophrenia. What is questionable is the magnitude of this effect. To date, the attributable risk for such an effect ranges between 2.03% for clozapine, 0.8% for quetiapine, 0.63% for olanzapine and 0.05% for risperidone (Leslie & Rosenheck, 2004).

Despite the evidence presented the debate still centres on the diabetogenic effects of certain atypical antipsychotics. The purpose of the editorial was to put these issues into perspective to ensure that patients with schizophrenia, irrespective of their prescribed medication, would be offered screening for both diabetes and the metabolic syndrome.


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CBT for treatment-resistant schizophrenia

We read with great interest the report on the randomised controlled trial (RCT) comparing cognitive–behavioural therapy (CBT) with supportive counselling for refractory psychotic symptoms of treatment-resistant schizophrenia (Valmaggia et al., 2005). It has a very convincing design but a few points need further discussion.

The sample size was calculated a priori, but an adequate number of patients could not be recruited. The small sample size led to a lack of statistical power, a limitation mentioned by the authors. However, this applied only to one intervention, supportive counselling, whereas there was an adequate estimated sample in the CBT group. Out of 62 participants randomised, post-treatment assessment was possible for 50 and follow-up was completed by 42. Although sample attrition is understandable in this kind of study the withdrawal rate is relatively high. More people in the CBT group refused assessment post-treatment compared with those who received supportive counselling. The reason for this needs to be explained. Loss of data by the assessor, leading to exclusion from the intention-to-treat analysis was greater for the group who received supportive counselling; this group already had fewer participants and the loss of data might have influenced the result.

The treatment groups were not comparable at the beginning of the study for one illness variable. The supportive counselling group reported significantly more emotional distress related to auditory hallucinations. This is important because there was no difference between the groups post-treatment and at follow-up assessment. In addition, the changes in negative symptoms reportedly favoured supportive counselling.

Valmaggia et al. stated that a larger percentage of participants in the cognitive–behavioural condition showed a 20% reduction in symptoms on the positive sub-scale of the PANS-S (Positive and Negative Syndrome Scale); however, comparative figures for both treatments and statistical significance would have illustrated this better.

Previous RCTs of the effect on symptoms of CBT compared with other psychological interventions showed a number needed to treat (NNT) of 5 (National Institute for Clinical Excellence, 2003). In the index study, the NNT was 3 but the confidence intervals were large in the two areas where a significant difference was measured for CBT.

Valmaggia et al. stated that CBT for refractory psychotic symptoms of schizophrenia should be available in in-patient facilities. However, the evidence from their study is not unequivocal. Although the literature suggests benefits from psychological intervention in this group of patients, more robust evidence is still required to confidently recommend one particular type of therapy over others.


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Personality subtypes and cognitive impairment in anorexia nervosa

I read with interest the article by Drs Thompson-Brenner and Westen (2005) about personality subtypes in eating disorders. Subnutrition from any cause is known to impair cognitive function and several workers have identified this in connection with anorexia nervosa (Macdonald, 1995).

The authors give no data on body mass index or weight. However, 38% of their sample had met criteria for anorexia nervosa at some point, 56% were fasting 4 days a week and half were exercising excessively.
It is likely therefore that cognitive impairment was present in many. Hence the ‘personality subtypes’ identified may vanish on refreeing, or may be more pronounced, having been masked by cognitive blunting. It seems best to seek for personality subtypes in eating disorders during periods of adequate nutrition.


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Authors’ reply: We appreciate Dr Macdonald’s point that subnutrition may cause cognitive changes and other temporary conditions that may appear to affect personality. Data regarding personality in the context of adequate weight and nutrition are important for the accurate description of personality in eating disorders. It can be difficult, however, to ascertain whether changes in personality functioning that take place through successful treatment or maturation precipitate positive nutritional changes or the reverse. Multiple studies do suggest that personality characteristics similar to those we describe in our report precede the development of eating disorders (Andervelh et al, 2003) and persist following remission of symptoms (Holtkamp et al, 2005). Although we did not report the data in detail, only 6.9% of those in our sample had a weight 15% below ideal, and the number of days of fasting was not correlated with either measure of personality pathology, suggesting this issue may not have compromised our data regarding personality to a significant extent.


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Psychedelics in psychiatry

In his editorial ‘Can psychedelics have a role in psychiatry once again?’ (Sessa, 2005), Dr Sessa offers a detailed historical and heuristic perspective of psychedelics, with particular reference to psychotherapy. Reading the article the feeling was of sensed (by the author) repulsion of the ‘neurobiological’ psychiatrist in relation to ‘research that explores alternative states of consciousness’, ‘psychedelics research’ as a ‘visible neurobiological substrate for the very human experience of religious encounter’ and generally a possible use of psychedelics in psychiatry. Perhaps we are some of those psychiatrists who ‘have been conditioned to consider such work as mysticism’ but we found such a proposition challenging. We would like to discuss recent neurobiological findings related to one of the psychedelics mentioned by Dr Sessa, which perhaps would offer an explanation as to why these substances have limited scope in psychiatry today.

3,4-Methylenedioxymethamphetamine (MDMA), also known as ecstasy, is largely consumed by young adults as a recreational drug. Common doses of this popular compound (60–120 mg, equivalent to 1–2 tablets) produce unexpectedly high blood levels, with MDMA present at high concentration at the receptor level. The drug induces dose-dependent neurotoxicity in animal models and humans; this mainly involves the central serotonergic system (Ricarte et al, 2002). Serotonin is important for brain development and maintenance of neural and glial function in the mature brain (Azmitha, 2001). Another interesting mechanism involves the ‘pruning’ of serotonergic neurons (Ricarte et al, 2000). The drug appears to reduce the number of serotonin axons and axon terminals but nerve cells will often replace terminals upstream for the damaged ones. The resulting effect is of substantial impaired connectivity. Younger brains are particularly susceptible because of increased neuroplasticity, resulting in a substantial reorganisation of brain connectivity.

Functional magnetic resonance imaging studies suggest a decreased activation in inferior temporal regions, the hippocampus, angular gyrus and striate cortex associated with working memory performance (Daumann et al, 2003, 2005), with the hippocampus and globus pallidus being possibly more sensitive (Renehan et al, 2001; Jacobsen et al, 2004; Daumann et al, 2005). More recent voxel-based morphometry studies support the hypothesis that the use of MDMA leads to reduction in cortical grey matter in multiple brain regions, including the neocortex, brain-stem, cerebellum and anterior cingulate gyrus, reflecting compromised serotonergic activity (Cowan et al, 2003). Although we have mentioned only a few studies, there is substantial evidence to suggest considerable neurotoxicity of compounds such as those mentioned by Dr Sessa. There are concerns about possible long-term adverse effects of psychedelics in both infrequent and regular users, which explain why psychiatrists are reluctant to consider such substances in their pharmacological armamentarium.


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Author's reply: I thank Drs Arnone and Schifano for their letter, which opens the debate about the undeniable dangers of recreational drug misuse, compared with the relatively safe clinical use of MDMA. This topic has received considerable media interest, with particular attention centred on the damaged brains of users of ecstasy – an illegal compound, which may or may not contain varying amounts of MDMA, together with any other substance. Ecstasy is usually taken in combination with other illegal drugs (Curran, 1998) or large amounts of alcohol, and often in circumstances involving high temperatures, which are known to exacerbate neurotoxicity (Malberg & Seiden, 1998).

All studies referred to by Drs Arnone and Schifano involve either recreational ecstasy users (mostly with no controls for other illegal drugs) or animal models with high and frequent dosage regimes that do not relate to those used for medically supervised MDMA psychotherapy research. The approach is therefore analogous to opposing the controlled, clinical research of opiate drugs after quoting studies of the morbidity of illegal heroin users.

Physiological studies involving infrequent and moderate doses of pure MDMA (as used in the psychotherapeutic setting) consistently demonstrate that the drug causes insignificant neurotoxicity, neuropsychological, mood or memory effects (Ludewig et al, 2003; Halpern et al, 2004).

Of course, there are risks when using any treatment – even in a controlled setting. All drugs, from paracetamol to cancer chemotherapy, are potentially harmful and must only be used after considering the risks and benefits – which includes considering the risk of doing nothing.

In relation to the opening comments, as a working psychiatrist, far from being 'repulsed' by neurobiological psychiatry, I am acutely aware of the importance of a holistic approach. Indeed, psychedelic psychotherapy, and the complexity of issues it raises, is a startling example of the effective interplay of concurrent psychological and organic treatments.

Given their history, MDMA and the other psychedelics are contentious treatments. However, it is possible for psychiatrists to think creatively and also consider safety and realistic risk–benefit ratios.

After all, if these compounds do have the potential to improve the speed and depth of psychotherapy, then they at least deserve clinical research in order to establish whether they can be useful tools to fight the global burden of neurotic illness.


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Biology and stigma

We wish to offer a brief response to the article by Drs Angermeyer and Matschinger (2005) entitled ‘Casual beliefs and attitudes to people with schizophrenia’.

Underlying all forms of discrimination, including psychiatric stigmatisation, is an exaggerated attribution of ‘otherness’ to certain individuals or groups, so that there is an assumption (made by the discriminator) of the existence of fundamental differences between himself and ‘the other’. Be it in the area of criminology, race, morality or mental health, this myth is further reinforced by the use of historically and culturally determined dichotomous constructs such as good/evil, citizen/alien, sane/insane and normal/pathological. The association of a biological marker with any stigmatised group acts as a signifier, further emphasising that group’s distinctiveness. Previous attempts to elucidate biological markers in criminals and in certain ethnic groups have served only to etch this mistaken notion of fundamental difference a little deeper in the mind of the discriminator and, in doing so, to reinforce prejudice. We believe that the findings of Angermeyer & Matschinger may be partly explained through a similar effect following the promulgation of a biological theory of schizophrenia.

Cognitive–behavioural therapists address this myth of difference as a therapeutic strategy when working with people with psychosis by emphasising the continuity of symptoms across the range from those designated as ‘sick’ to ‘normal’ (Kingdon & Turkington, 1994). We believe that the extension of such an approach in the wider treatment of society could have a powerful role to play in the ongoing campaign against psychiatric stigmatisation. The origins of our current unsatisfactory conceptualisation of mentally ill people as being separated from ‘normal people’ by an absolute and fundamental boundary are often attributed to the Kraepelinian model. We welcome then the predictions made by Craddock & Owen (2005) of the imminent demise of this in favour of a newer, hopefully less iatrogenic paradigm.


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