The antidepressant debate continues

In her March 2002 editorial, Dr Moncrieff raises doubts about the efficacy of antidepressant drugs, and argues that side-effects of the active drug may explain some of the differences owing to the associated increased expectancy of a positive effect. Our 24-week study (Malt et al, 1999) comparing the efficacy of empathic primary-care counselling and support combined with placebo, a selective serotonin reuptake inhibitor (sertraline) or an 5-HT2/3 antagonist (mianserin) in 372 subjects with depressed mood does not support her arguments.

In our study the general practitioners were required to systematically explore possible side-effects. This method yields a greater prevalence of side-effects than when only spontaneously reported side-effects are considered. The mean numbers of baseline-corrected UKU-elicited side-effects (Lingjaerde et al, 1987) during the study were 7.11, 6.51 and 6.45 after 8 weeks of treatment with sertraline, mianserin and placebo, respectively, and 3.16, 3.09 and 3.02 after 24 weeks of treatment (NS). This shows that prevalence of side-effects is unlikely to explain the difference in response.

Another observation arguing against the hypothesis that non-specific side-effects may explain differences between active drug and placebo is the fact that we obtained differences in response over time among the three treatment arms. As would be expected by the pharmacodynamic profiles of the drugs, mianserin induced a faster initial response, while sertraline demonstrated an advantage in the long run explained by better efficacy among subjects with high neuroticism. At the end of the study, the physicians were not able to identify reliably the treatment given to each of their patients.

Furthermore, differences in effect size between the treatments (see Table 1) clearly demonstrated the advantage of antidepressant drugs on core symptoms of depression. These differences are well beyond the estimated mean effect size of 0.27 reported for active placebo.

Table 1 Differences in effect size on item levels measured on the Montgomery–Åsberg Depression Rating Scale between three forms of treatment in a 24-week randomised treatment trial of patients with depression (n = 372) in primary care. Intention-to-treat data obtained from Malt et al. (1999)

<table>
<thead>
<tr>
<th></th>
<th>Sertraline v. placebo</th>
<th>Mianserin v. placebo</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed sadness</td>
<td>0.04</td>
<td>0.19</td>
</tr>
<tr>
<td>Reported sadness</td>
<td>0.47</td>
<td>0.39</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0.34</td>
<td>0.22</td>
</tr>
<tr>
<td>Insomnia</td>
<td>0.59</td>
<td>0.36</td>
</tr>
<tr>
<td>Appetite</td>
<td>−0.09</td>
<td>0.05</td>
</tr>
<tr>
<td>Concentration problems</td>
<td>0.08</td>
<td>−0.15</td>
</tr>
<tr>
<td>Lassitude</td>
<td>0.36</td>
<td>0.24</td>
</tr>
<tr>
<td>Inability to feel</td>
<td>0.10</td>
<td>0.22</td>
</tr>
<tr>
<td>Pessimistic thoughts</td>
<td>0.54</td>
<td>0.45</td>
</tr>
<tr>
<td>Suicidal ideation</td>
<td>0.13</td>
<td>0.04</td>
</tr>
</tbody>
</table>

1. Patients with psychotic ideation or active suicide plans were excluded from the study.

Beyond this trial found statistically significant differences between active drugs and placebo, these differences were very small and of doubtful clinical relevance. The difference in the reduction of scores on the Montgomery–Åsberg Depression Rating Scale (MADRS) between the active drugs and placebo consisted of a maximum of 3 points. The MADRS scale has a total of 60 points and mean baseline values in this study were 27. In subjects in whom depression was characterised as severe or major depression, the differences were smaller still, and were not statistically significant.

It is also worth pointing out that although this trial found statistically significant differences between active drugs and placebo, these differences were very small and of doubtful clinical relevance. The difference in the reduction of scores on the Montgomery–Åsberg Depression Rating Scale (MADRS) between the active drugs and placebo consisted of a maximum of 3 points. The MADRS scale has a total of 60 points and mean baseline values in this study were 27. In subjects in whom depression was characterised as severe or major depression, the differences were smaller still, and were not statistically significant.

It is argued that treatment of mild depression in primary care with antidepressants is the worst case of the inappropriate medicalisation of misery and social problems. This may be harmful to the individuals concerned by encouraging reliance on physical treatments, and to society by masking the social conditions that are the sources of modern discontent.

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Effects of exercise on depression in old age

The study by Mather et al (2002) is a laudable work but has some important shortcomings. The control group had received health education, and the authors have justified this approach. Considering the fact that well-designed studies addressing the usefulness of exercise in depression in old age are lacking, we believe inclusion of a control group that did not receive another intervention other than continuing antidepressants could have made Mather et al’s findings more meaningful. Another issue is attendance rate: despite the fact that exercise facilitated recovery from depression and that no one dropped out, the reasons for a low mean attendance rate in the exercise group remain unclear. Also, whether this low attendance rate contributed to the lack of significant group differences in outcome measures at the 34th week (final assessment) needs clarification.

With regard to the statistical analysis, besides the analysis of outcome at certain points, the authors could have used the Wilcoxon test for paired samples or another comparable statistical test to detect differences in outcome from baseline scores. The authors state that both groups had scores of secondary outcome measures at the 10th and 34th weeks that were significantly different from baseline, but this statement is not supported by an appropriate statistical analysis.

Given that the authors had great difficulties while recruiting the study sample, rectification of the above limits could have made their conclusions more robust.


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Authors’ reply: We are grateful to Dr Jagadheesan and colleagues for allowing us to reiterate the methodological strength of our trial design. If our control group had been as Dr Jagadheesan proposes (continued antidepressant treatment only), then we would wrongly have concluded that exercise is highly effective as an adjunct to drug treatment in old age depression. In fact when compared with the effects of a structured social intervention, group exercise offered only a modest additional benefit. Our non-exercise control crucially allowed us to disentangle the psychosocial effects of coming together as a group from the effects of exercise itself. This novel use of a non-exercise control intervention which matched the exercise intervention in duration, frequency and social contact represents an important methodological advance which future researchers will wish to consider (Lawlor & Hopker, 2001).

Perhaps Salmon may shed some light on the low mean attendance rate by his comment that advocacy of exercise as a treatment for depression ‘must puzzle clinicians, who in treating depressed people, often have to contend with an absence of motivation to tackle much less strenuous features of life’s routine’ (Salmon, 1990).

The Results section of our paper is succinct, in part because of editorial constraints on article length. A typical finding (such as a group comparison of reduction in Hamilton Rating Scale for Depression score at 10 weeks) gives only a comparison of proportions and the associated P value, with the statement ‘we used Fisher’s exact test in our comparison on the two groups’ implied, but unwritten. In fact, we prided ourselves on the explicit statement of numerator and denominator here and elsewhere (23/42 v. 14/43) when many authors might simply have said ‘55% v. 33%, P=0.05’.

We are surprised that Dr Jagadheesan et al advocate use of the Wilcoxon test for paired comparisons: this would have resulted in a highly undesirable plethora of comparisons (10 weeks v. baseline; 34 weeks v. baseline; 34 weeks v. 10 weeks). Statistical propriety necessitates the use of an approach which recognises the temporal ordering of the trio of results for each subject in each group; hence our appropriate use of repeated measures.

The analysis which resulted in the stated findings for secondary outcome measures was a one-sample t-test on the logarithms of the ratios of outcome:baseline scores.

There is a desperate need for better-quality research in the area of depression, and we believe that our trial design offers an important methodological advance.

Declaration of interest

M.E.T.M. is codirector of DD Developments, a University of Dundee company providing exercise classes for older people and whose profits support research into ageing.


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Unethical use of placebo controls

Klysner et al (2002) evaluated the prophylactic efficacy of citalopram in comparison with placebo in elderly patients, and stated ‘the highly recurrent nature of major depression in the young and the elderly warrants long-term antidepressant treatment’. In view of this, is it ethical to use a placebo arm? The answer to this question depends upon whether or not there is an already available treatment of proven or accepted value. In this context, Cochrane (1989) stated that ‘placebo controlled trials are appropriate when there is no existing treatment for a disorder, otherwise comparison trials are indicated. No new treatments should be introduced into medicine unless they have been shown, in randomised controlled trials, to be superior to existing treatments, or equivalent to existing treatment but cheaper or safer’. Similarly, section 12.4 of the National Health and Medical Research Council (1999) statement on ethical conduct in research involving humans states: ‘the use of a placebo alone or the incorporation of a non-treatment control group is ethically unacceptable in a controlled trial where: (a) other available treatment has already been clearly shown to be effective; and (b) there is a risk of significant harm in the absence of treatment. If there is genuine uncertainty about the net clinical benefit of a treatment, a placebo controlled trial or a trial with a no-treatment arm may be considered’.

The use of placebo in this clinical drug trial raises questions of deception, of patient information and of informed consent. The patients in the placebo group were left without any active treatment for 48 weeks – this raises doubt as to whether patients were fully informed, before giving their consent, that they might receive a placebo by random allocation. We are keen to
know why the authors did not try to compare the efficacy of citalopram with existing antidepressants.


National Health and Medical Research Council (1999) National Statement on Ethical Conduct in Research Involving Humans. Canberra: NHMRC.

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Authors' reply: Drs Jainer and Soni have addressed an important issue in clinical trials in depression when commenting on our article. Our study was the first specifically designed and conducted to evaluate the therapeutic value of prevention of recurrence of a depressive episode in an elderly population. The study was designed using the concept of the three phases of antidepressant treatment: acute, continuation and maintenance treatment (Montgomery et al, 1988). The study is unique in that the majority of the population had suffered only one documented depressive episode upon admission into the study.

At the time the study was initiated, there was sparse evidence for the value of prophylactic treatment after a first episode of depression in elderly patients. Thus, the requirement that there be no ‘other available treatment [that has already been clearly shown to be effective’ was fulfilled.

Prior to initiating the study, the local ethics committee approved the protocol as well as the patient information and the informed consent form. The patient information explicitly mentioned the use of placebo in the double-blind period. All patients gave written informed consent before being included in the study.

Existing guidelines clearly stipulate that treatment of at least 6 months’ duration is necessary to reduce the risk of relapse. The study complied with this by providing active treatment with citalopram for 24 weeks. Only patients in remission, after a total of 24 weeks of treatment with citalopram, were randomised to double-blind treatment with citalopram or placebo. The patients were closely monitored during the double-blind period until discontinuation or completion. Patients with recurrence of depression in the double-blind treatment period were withdrawn and treated at the investigators’ discretion.

In addition, an active-comparator trial can only provide information regarding relative effect, but not whether prophylactic treatment is clinically warranted. The absolute value of prophylactic treatment can only be concluded from a placebo-controlled trial. Thus, the study had a placebo-controlled design for the double-blind period, in accordance with the National Health and Medical Research Council guidelines as cited by Drs Jainer and Soni (‘If there is a genuine uncertainty about the net clinical benefit of a treatment, a placebo controlled trial or a trial with a no-treatment arm may be considered’). The study established that long-term treatment with citalopram is effective in preventing recurrence of depression in the elderly and is well tolerated. With this knowledge, along with other currently available information, we certainly agree with the authors that the appropriateness of conducting similar studies in the future should be considered. However, our opinion notwithstanding, there is no consensus regarding the need for prophylactic treatment in the elderly. Until clinical practice and guidelines are changed, studies of a similar nature will have to be undertaken to convince the scientific community of the value of long-term treatment.

Declaration of interest

The study in question was funded by H. Lundbeck A/S. M.A. is an employee of H. Lundbeck A/S.


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Costs of dementia

In their recent paper, Wosthenholme et al (2002) demonstrated that changes in cognitive and functional status have independent and significant effects on the costs of care in dementia. We agree with the authors that models of costs based solely on measures of cognitive changes are inappropriate to describe variables influencing the costs of dementia. From 1994 to 1999 we conducted in Italy a longitudinal study on costs of Alzheimer’s disease (the CoDem Study), based on information obtained every 6 months from a sample of 148 patients with Alzheimer’s disease living at home (73.6% female, mean (s.d.) age 78 (7.8) years, mean (s.d.) Mini-Mental State Examination (MMSE) score at baseline 8.9 (8.3)), estimating direct and indirect costs of dementia (Trabucchi et al, 1996). In a preliminary analysis after the first year of observation, using a logistic regression analysis, we found that greater annual costs for Alzheimer’s disease are significantly associated with more disability than with cognitive decline (Bianchetti et al, 1998). Following this line of investigation, we evaluated the modification of costs with the progression of the disease at the end of the 6-year longitudinal study with a Markov state transition model based on the comparison of costs for different stages of cognitive and functional decline (measured using the MMSE and the Basic Activities of Daily Living (BADL) scale) (Jonsson et al, 1999). In our study total costs (per year) for dementia care varied from €15 450 (£9972) for independent patients (BADL lost=0), to €21 463 (£13 853) for partially independent subjects (1–3 BADL lost) and €23 762 (£15 336) for totally dependent patients (4–6 BADL lost). Using the MMSE, the costs varied from €18 024 (£11 633) for patients with mild Alzheimer’s disease (MMSE >20), to €19 665 (£12 692) for patients with moderate decline (MMSE 15–20) and €25 351 (£17 077) for patients with severe cognitive decline (MMSE 8–14) (Trabucchi, 1999).

Our data, obtained in a sample of subjects with Alzheimer’s disease living in a different social and cultural context, strengthen those obtained by Wosthenholme and colleagues, emphasising in particular the need to demonstrate an effect on functional status in the cost-effectiveness analysis of interventions in dementia.

Declaration of interest

The CoDem Study was funded by a grant from Bayer Pharmaceuticals, Milan, Italy. A.B. and M.T. have received financial support from various pharmaceutical...
CBT for psychosis

I am writing to reply to Turkington et al (2002: p. 525), who claim in their interesting and recently published paper on cognitive–behavioural therapy (CBT) for psychosis, that ‘The NNT [numbers needed to treat] of 13 for improvement in overall symptoms was compatible with the results achieved when CBT was delivered by expert therapists (Kuipers et al, 1997)’. We do not think this claim is justified.

First, in our study 64% of the CBT group achieved clinical improvement compared with 47% of the controls (Kuipers et al, 1997). We did not present the NNT but they are 6 at the end of treatment and 3 at the end of follow-up (Kuipers et al, 1998).

Second, the two studies address different questions in different samples. Our study tested whether CBT for psychosis could improve outcome compared with treatment as usual, in a sample comprising subjects deliberately chosen to have at least one distressing, positive, medication-resistant symptom of psychosis (not from ‘lists of patients with schizophrenia receiving treatment’; Turkington et al, 2002: p. 523). We were aiming at a treatment-resistant group, a rather different sample from that recruited by Turkington and colleagues. Neither study compared 9 months of CBT with a briefer intervention. Nor did they test the efficacy of two different kinds of CBT.

We believe that it is misleading to claim comparability of trials between ‘expert’ and ‘non-expert’ therapists, and between results from 6 sessions and 20 sessions. Evidence for the efficacy of CBT for psychosis is at an early and promising stage; we think it is unhelpful to make unsubstantiated comparisons across trials, and hope that these comments provide some clarification.


Author’s reply: Our study was designed specifically to answer the question raised by Jones et al (1999) of whether the benefits achieved by expert therapists in research settings could be replicated by non-expert therapists working in community mental health teams. An end-of-therapy comparison was therefore necessary with one of the methodologically robust studies quoted in the above review. Kuipers et al (1997) was chosen because a similar, good clinical outcome analysis on overall symptoms had been reported at end of therapy. The appropriate end-of-therapy comparison is 14/28 (50%) for cognitive–behavioural therapy (CBT) as measured at the level of 20% improvement in overall symptoms in the original Kuipers et al (1997) paper compared with 112/257 (44%) as measured at the level of a 25% improvement in our study. These results show a comparable effect size for CBT in the two studies, considering that our study had to satisfy a more stringent criterion for a good clinical outcome. The difference in the numbers needed to treat is solely due to an improved performance in our treatment as usual group compared with standard care.

It is certainly correct to state that the two study populations were different by definition. However, consideration of the demographics as reported in the two papers shows that there was little difference in those who actually ended up being enrolled in the two studies. The mean number of admissions in Kuipers et al (1997) was 5.2 for the CBT group and 4.3 for standard care and in our study 4.71 for CBT and 5.18 for treatment as usual. We ended up enrolling a more treatment-resistant group because of the fact that patients with schizophrenia whose symptoms were well controlled with medication often did not see the need to enter the study when it was offered to them.

It is certainly true that the CBT delivered by Kuipers and colleagues was of 20 sessions’ duration with a more sophisticated treatment manual. This makes the result of our brief CBT intervention as delivered by psychiatric nurses all the more impressive. We await the analysis of our short-term follow-up results to see whether the impressive durability results reported above can be equalled. If CBT is to make a real impact in terms of the management of schizophrenia, it will need to be delivered by non-expert therapists in community mental health teams. The real issues for expert cognitive therapists are to organise training courses, provide supervision and to deliver more complex CBT for those patients with schizophrenia who are more psychologically difficult or who have comorbidity such as post-traumatic stress disorder, alcohol dependence and social phobia. There is therefore a potential role for both expert and non-expert therapists in the management of every patient with schizophrenia.


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Screening for PTSD

We read with great interest the article by Brewin et al (2002). The authors examined the efficiency of the 10-item version of the Trauma Screening Questionnaire (TSQ) in detecting post-traumatic stress disorder (PTSD). In our opinion, the scale design has some limitations which may have a negative influence on its practical application.

First, the TSQ contains five re-experiencing items and five arousal items, but not the avoidance and numbing symptoms. According to DSM-IV diagnostic criteria for PTSD (American Psychiatric Association, 1994), the patient requires the presence of at least one re-experiencing symptom (criterion B), three avoidance symptoms (criterion C), and two arousal symptoms (criterion D). The criterion C is the least frequently met criterion but critically significant to the diagnosis of PTSD (Maes et al, 1998). Some trauma survivors, who express most PTSD symptoms, do not fulfill the avoidance criterion and are diagnosed as having 'partial' PTSD. Other brief screening instruments, such as the four-item SPAN (Melzter-Brodby et al, 1999) or the seven-item scale by Breslau et al (1999), place much weight on the avoidance and numbing symptoms. Therefore, this specific item composition may influence the efficiency of the TSQ.

Second, the TSQ uses the frequency threshold allied to a 'yes/no' response format. Although comparison of scores derived by frequency and by severity indicated a degree of similarity, the severity dimension might provide better discrimination than the frequency dimension (Melzter-Brodby et al, 1999). In our clinical experience, subjects can score the severity variable more accurately than the frequency ones (Chen et al, 2001). The item selection and scoring method have greater influence on the efficacy of the rating scale.


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Author's reply: Drs Lu and Shen claim that our Trauma Screening Questionnaire (TSQ; Brewin et al, 2002) is flawed because it omits avoidance and numbing symptoms and asks about symptom frequency using a simple 'yes/no' response format. It is puzzling then that the performance of the TSQ is superior to that of all comparable screening measures, including ones that follow Lu and Shen's recommendations. Their views are clearly contradicted by the data from the two studies we reported. Our reasons for designing the TSQ in the way we did were based on empirical and practical rather than theoretical considerations. In our original article we discussed some general principles for designing successful screening instruments, whereas Lu and Shen's comments seem more relevant to a diagnostic instrument. The two types of measure tend to be administered by different professionals, under different circumstances, and with different aims in mind. It seems to us that, as a screening instrument, what the TSQ gains in simplicity and clarity more than compensates for the absence of symptoms that may be difficult to understand and judgements that may be difficult to make.


Transcultural psychiatry

Drs Bhui & Bhugra (2002) address the interesting area of explanatory models for mental distress. They do not, however, justify why we should elicit patients' explanatory models. The notion that members of a specific cultural group hold similar ideas about illness and that culture can be distilled into a set of specific 'beliefs' is considered outdated and oversimplified by medical anthropologists. Kleinman (1980) points out that explanatory models are idiosyncratic and are justifications for actions rather than causes. Bhui & Bhugra themselves cite Williams & Healy (2001), who point out that it is difficult to distil a single set of causal explanations that might relate to behaviour, diagnosis or adherence to medication treatment.

The assertion by Bhui & Bhugra that shared understanding of illness between patient and healer distinguishes traditional healing systems from Western biomedicine is simply not borne out by the anthropological literature. In many systems of traditional healing, patients have little understanding of how the treatment 'works' and it is the healer who holds highly esoteric knowledge. There is little empirical evidence that eliciting explanatory models improves satisfaction. The one study cited (Callan & Littlewood, 1998) in fact found that 79% of patients with divergent explanatory models (a comparison of the explanatory models of doctors and patients) were satisfied with psychiatric services.

Of course, patients do have cultural understanding of their illness but this may not be very sophisticated and may not directly relate to decisions about treatments. There is a large amount of data from medical anthropological research which suggests that treatment choice is determined primarily by social and political factors rather than by underlying explanatory models (Pelto & Pelto, 1997). Even a study using the Explanatory Model Interview Catalogue (Weiss et al, 1992) among leprosy patients suggests that those who held theories of humoral imbalance rather than biomedical theories of infection, sanitation and hygiene had the best biomedical clinical attendance records for leprosy treatment. In terms of treatment outcomes patients may not be interested in how a treatment works (Last, 1981) as long as it does work. The weight of empirical evidence suggests that people are keen to utilise biomedical treatments regardless of their cultural beliefs without giving up traditional explanations of illness. In fact, as my own data (Dein, 2001) suggest, among Asian psychiatric patients, biomedical and traditional models of illness are held concurrently and informants agree that biomedical.
Pathways to care in ADHD

I was interested to read Sayal et al’s (2002) article on pathways to care for children at risk of attention-deficit hyperactivity disorder (ADHD). By using Goldberg & Huxley’s (1980) pathway to care model I felt that the study oversimplified the complexity of professional input to this group of children, a point raised by the authors in their discussion. I think it is important, when considering improvements to services for children with ADHD, that the role of education is highlighted.

Teachers, as a profession, are well placed to observe children and are familiar with age-appropriate behaviour. Indeed, Goodman et al (2000) found that teachers were more sensitive at identifying children with hyperactivity than were their parents. Although teachers’ involvement in the assessment and monitoring of children with ADHD is well established (Dulcan et al, 1997), their role in identification is less clear. This is highlighted by the fact that only some child and adolescent mental health services (CAMHS) accept referrals directly from schools. By involving teachers in the identification of children with ADHD, access to children would improve from 74% seen in primary care to nearly 100%. This would significantly improve the sensitivity of any screening measure.

It is essential that CAMHS do not develop services for children in isolation, but instead utilise the skills of other professionals to improve care. If children with ADHD are to have their needs met, it is essential that we start to think outside of the medical model.


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Authors’ reply: Dr Cribb’s comments are welcomed. Our discussion (Sayal et al, 2002) also highlights the potential role of both parents and teachers in making referrals to child and adolescent mental health services (CAMHS). The paper is developing new methodology and we deliberately posed it on a simplified system, selecting an area where most referrals come from general practitioners (GPs). GPs are also the main referrers to CAMHS nationally and their role in primary care trusts will be of great importance in shaping specialist services. Nevertheless, this is only one component of tier 1 services and 48% of CAMHS referrals come from other sources (Audit Commission, 1999). We plan to widen our programme to examine the role of other sources of referral.

Restricting referrals to particular agencies imposes barriers to access, and the resulting delay in referrals might exacerbate severity or chronicity of problems. Kurtz et al (1996) described a service that only accepted GP referrals. It failed to reduce the number of referrals and generated resentment from other agencies. Comparisons of CAMHS with different referral systems will improve knowledge in quantifying the barriers to access to services. This could contribute to assisting the successful implementation of the National Service Framework for Children.

The role of teachers in the pathway to care merits particular comment. Relationship difficulties with teachers are a predictor of referral of hyperactive children to CAMHS (Woodward et al, 1997). Our study has demonstrated that selective targeting can lead to particularly high rates (98%) of teacher participation in research. This is likely to reflect their concern about behaviour and emotional difficulties in children. Teachers are a rich potential source of child mental health information for parents. However, in considering referrals from schools, it is imperative that teachers fully discuss their concerns with parents. Parents need to agree to any referral. For hyperactivity, in particular, it needs to be ascertained that the problems are pervasive. Unless this happens, there is a risk that learning difficulties are wrongly identified as hyperactivity. This also highlights the importance of adequately resourced educational psychology services to support schools, and health service input in the training of teachers.


Consciousness still a mystery
Baroness Greenfield’s (2002) editorial is shaped by the metaphors of empiricism. The brain is a network. The mind is distinct patterns of neural connectivity. Currently, the evidence for such a scenario is limited. Brain connections may, as she says, ‘actually reflect experience’, but no pattern of connectivity has ever been related to any particular mental state. The alternative hypothesis of functional specialisation merits more than the scant consideration granted in the editorial, given the recent interest in the notion of modularity (Fodor, 1983; Pinker, 1999). Perversely, Greenfield chooses to support her ‘network’ hypothesis by reference to a study showing regionally localised brain changes in taxi drivers (Maguire et al, 2000).

Consciousness is introduced as a dimensional variable quantifying the current extent of this connectivity. Seemingly, the more connected our brains are the more conscious we are. But is this anything more than metaphorical footing around? She presents no evidence for what a conscious brain state might look like. Where consciousness occurs is surely rather an unimportant issue. The hard question, which Greenfield ignores, is ‘How can pain (which hurts so) possibly be the same thing as insensate molecules rushing around in nerve fibres?’ (Papineau, 2002). On this our ignorance remains as complete as it ever was.


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One hundred years ago

Ladislav Haskovec and akathisia: 100th anniversary

Akathisia is a syndrome of objective and subjective motor restlessness manifested by an inability to sit or stand still. The patients are distressed and they pace constantly. Today, it is mostly known as a side-effect of antipsychotic medications. However, the phenomenon was observed before the introduction of antipsychotics, and the term ‘akathisia’ (derived from the Greek ‘inability to sit’) was coined in 1901 by Ladislav Haskovec, MD. A Czech neuropathologist, Haskovec was born in 1866 and died in 1944.

After graduation from the Charles University School of Medicine in Prague, he spent a year in Paris working with Professor Charcot, the leading neurologist at that time. His original primary interest was neuropathology, but he soon branched out into many other areas. He published on thyroid function, tuberculosis, alcoholism, neuroses, obsessions, mechanisms of consciousness, seizure disorders and heredity. His publications and presentations earned him international recognition and numerous honours in Austria, Czechoslovakia and France. He was appointed full professor at the Charles University in 1919, and served as Dean of the Charles University School of Medicine in 1925–1926.

Throughout his long career, Haskovec was an astute clinician. He coined the term ‘akathisia’ to describe symptoms he observed in two of his patients. These two case reports were presented at the meeting of the Societe de Neurologie in Paris on 7 November 1901 (Haskovec, 1901). (English translations of Haskovec’s papers with a commentary were published elsewhere (Berrios, 1995).) The patients were adult males who had a multitude of symptoms including insomnia, vertigo, various aches and pains, and paraesthesias. Both men complained of generalised tremor; apparently this was not observed during examination. The prominent symptom in both patients was that they were unable to remain sitting down for any length of time. When sitting, at least one of the patients had a sensation in his legs as if he were jumping (today, a clinician would perhaps describe this sensation as a feeling of restlessness). The movements were described as involuntary by the patients who actually wanted to stop them; one of them tried to hang on to a table to prevent himself from getting up. After jumping up from the sitting position, the patient kept walking around, and conversation with him was only possible when he was moving. Gait was normal in both patients; neurological examination revealed no clear abnormalities, and there were no signs of psychosis.

Haskovec tentatively diagnosed one man with ‘hysteria’ and the other one with ‘neurosenhia’. He speculated about underlying mechanisms along the lines of ‘hyperexcitability’ or ‘fatigue’ of various brain structures, using theoretical concepts of his era.

The report elicited discussions with French neurologists who accepted the new term but wanted to apply it differently from its originator (Haskovec, 1903). Nevertheless, today’s phenomenology of akathisia remains essentially the same as described by Haskovec 100 years ago. Neurology textbooks of the pre-antipsychotic era described akathisia in Parkinsonian patients, and the importance of the term has further increased after the introduction of antipsychotics in the 1950s. Neurologists and psychiatrists are indebted to Haskovec for his astute observations.


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The antidepressant debate continues
U. F. Malt
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